The effects of lesions of superior temporal gyrus and inferior parietal lobe on temporal and vertex components of the human AEP

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Summary We recorded auditory evoked potentials (AEPs) to 1 kHz tone bursts in controls and patients with unilateral lesions centered in posterior superior temporal gyrus and adjacent caudal inferior parietal lobule (STG) or in rostral inferior parietal lobule (IPL). Controls generated a vertex maximal N94 (N1b) and P200 (P2) and additional P45, N78 and N127 temporal AEP components (P45; N1a, N1c). Similar to prior reports, in controls the N1a was most prominent over the left temporal lobe and the P45 was largest over the right temporal lobe consistent with behavioral and anatomical data indicating differential organization of left and right human temporal lobe. The N1c was recorded equally from both T1 and T2 electrodes and was enhanced in the temporal site contralateral to the ear of stimulation. The patient groups had differential effects on AEPs. Unilateral STG lesions resulted in bilateral reductions of the N1b and P45 and marked unilateral reductions of the N1a and N1c over lesioned hemisphere. IPL lesions resulted in bilateral but non-significant reductions of the N1b and N1c.

The scalp topography results in normal subjects combined with the effects of unilateral STG lesions provide supportive evidence that the temporal maximal components of the human AEP (P45, N1a, N1c) are generated by radially oriented neuronal dipole sources located in STG. The bilateral reduction of the N1b vertex response by unilateral STG lesions is compatible with a unilateral disruption of a vertically oriented dipole situated in the posterior superior temporal plane. The results emphasize the critical role of the superior temporal plane and lateral superior temporal gyrus in generation of human long latency AEPs.

Key words: AEPs; T-complex; Temporal lobe; Parietal lobe; Vertex potential

Transient auditory stimulation generates prominent negative potentials peaking at about 100 msec post stimulation with maximal amplitudes recorded at midline and temporal scalp sites. Data from scalp topography (Wolpaw and Penry 1975; McCallum and Curry 1979, 1980; Wood and Wolpaw 1982; Perrault and Picton 1984), intracranial recording (Celesia 1976) and dipole modeling (Scherg and Von Cramon 1985, 1986) have provided evidence in support of multiple generators of the long latency AEP. A positive-negative potential (P100-N150) differing in latency from the vertex maximal N100 was recorded from temporal sites in normal subjects and these potentials were referred to as the T-components. This positive-negative complex was initially proposed to arise from a radially oriented dipole in the superior temporal plane (Wolpaw and Penry 1975). Later workers confirmed the existence of a vertex maximal negativity referring to this component as the N1b and also recorded temporal maximal components which were labeled P45, N1a and N1c (McCallum and Curry 1979, 1980; Perrault and Picton 1984; Cacace and Wolpaw 1987). The N1a and N1c occurred at approximately 75 and 130 msec post stimulus and the N1a was reported to be maximal over the left temporal sites (McCallum and Curry 1979, 1980), whereas the P45 was reported to be maximal over the right temporal

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lobe (Cacace and Wolpaw 1987). In order to further investigate the origins of the temporal components (P45, N1a, N1c) and the vertex N100 (N1b), we recorded long latency AEPs in patients with unilateral lesions centered either in posterior superior temporal gyrus and adjacent caudal inferior parietal lobule (STG) or in rostral inferior parietal lobule (IPL).

Methods

Stimulation and recording

The subjects were comfortably positioned in an easy chair in a sound attenuated, air conditioned, dimly lit chamber. Stimuli consisted of 50 msec, 1 kHz tone bursts with 5 msec rise and fall times delivered at a 1 sec ISI. These stimuli were the standard non-target stimuli occurring on 80% of the trials in a tone detection paradigm the patients were engaged in. All subjects were awake and alert during the experiment and had comparable tone detection capacity and reaction times to the targets (Knight et al. 1987). The stimuli were presented monaurally at 60 dB HL over 35 dB of continuous broad-band noise (100 Hz–20 kHz) presented binaurally. Evoked responses were recorded with Ag/AgCl electrodes placed at FP1, FP2, F3, F4, C3, C4, P3, P4, T3, T4, FP1, FP2, Fz, Cz, Pz, Oz and below the left eye all referenced to a balanced non-cephalic steinal-cervical electrode. A ground electrode was attached to the forehead. Electrode impedances were kept below 5 kΩ and bandpass was set at 0.1–100 Hz. An 800 msec epoch was digitized on-line to disk at a sampling rate of 256 points/sec. The epoch consisted of 200 msec of pre-stimulus activity and 600 msec of post-stimulus response. Trials with excess muscle or eye movement were automatically excluded from the average. Sums of approximately 350 evoked responses, with replications of an additional 350 responses, were recorded for each ear.

Data analysis

Amplitudes were measured relative to the prestimulus baseline. Measurement windows were determined from initial inspection of individual subjects data and group superaverage tracings. Both peak and mean amplitudes were analyzed for all components. The mean amplitude was a measure of the mean voltage over the specified latency range and was included in the analysis since it is a less noise-biased measure. In this study the peak and mean amplitude measures produced equivalent levels of significance and only the P values for peak amplitude are reported. The latency windows chosen were N1b: 80–110 msec, N1a: 54–92 msec, N1c: 116–136 msec, P45: 40–60 msec, and P200: 190–230 msec. Latency peaks were also tabulated in these windows. Based on prior data in the literature on the T-components and on our group superaveraged tracings, we elected to treat the T3 and T4 temporal electrodes as independent measures for analysis of the N1a, N1c and P45. Following similar logic, the vertex maximal N1b and P2 were analyzed without inclusion of the temporal sites. Since the data were recorded as a subset of a protocol designed to study the endogenous N200 and P300, only T3 and T4 electrodes were available for analysis. A more detailed analysis of the scalp topography at temporal sites of the effects of lesions on the T-components would be desirable in future studies as would use of an averaged reference or laplacian derivatives. The data were organized as a function of electrode site over lesioned hemisphere and as a function of ear ipsilateral or contralateral to lesion and then subjected to univariate repeated measures analysis of variance and specific comparisons when appropriate.

Subjects and lesion reconstruction

Three groups of subjects were tested. Controls consisted of 17 right-handed subjects (57 ± 14 years) age-matched to the patients. Patients or controls with a history of substance abuse, major psychiatric or medical disorders, dementia, or multiple neurological events were excluded. No controls or patients had a history of audiological dysfunction. All controls and patients with STG or IPL lesions had pure tone audiograms performed prior to inclusion in the study. Subjects with greater than 15 dB interaural threshold differences were excluded from entry into the study. There was no difference in mean audiometric threshold between controls and patients (1 kHz
mean threshold; controls = 15 ± 8 dB, STG = 21 ± 13 dB, IPL = 14 ± 11 dB). Similarly there was no threshold elevation in the ear contralateral to lesioned hemisphere for either patient group (for STG, ipsilateral ear = 21 ± 15 dB, contralateral ear = 22 ± 12 dB; for IPL, ipsilateral ear = 13 ± 14 dB, contralateral ear = 15 ± 9 dB). After preliminary auditory screening, the initial patient group consisted of 22 subjects with single unilateral lesions centered in posterior STG or IPL.

Of this group, 7 subjects were found to have unsuitable EEG recordings for a variety of reasons. These included uncontrollable muscle activity (2), excess eye movements (2), claustrophobic

**TABLE I**

Summary of patient information for STG and IPL groups including sex, age, clinical deficits, pathology, duration and individual lateral CT reconstruction of lesion.

<table>
<thead>
<tr>
<th>Age/Sex/Right</th>
<th>CT Scan</th>
<th>Clinical Deficit As Time of Testing</th>
<th>Pathology/Duration</th>
<th>Age/Sex/Left</th>
<th>CT Scan</th>
<th>Clinical Deficit As Time of Testing</th>
<th>Pathology/Duration</th>
</tr>
</thead>
<tbody>
<tr>
<td>57/N/L</td>
<td></td>
<td>Normal</td>
<td></td>
<td>58/N/R</td>
<td></td>
<td>Visual, somatosensory extinction L side, mild L neglect on line cancellation</td>
<td>CVA/1 yr</td>
</tr>
<tr>
<td>60/N/L</td>
<td></td>
<td>Conduction aphasia</td>
<td></td>
<td>58/N/L</td>
<td></td>
<td>Mild aphasia, somatosensory L side</td>
<td>CVA/12 yrs</td>
</tr>
<tr>
<td>65/N/L</td>
<td></td>
<td>Broca, agrammatia, sensory difficulty, B superior quadrantanopsia</td>
<td>CVA/21 yrs</td>
<td>57/N/L</td>
<td></td>
<td>Conduction aphasia, unsteady, partial right inferior quadrantanopsia</td>
<td>CVA/4 yrs</td>
</tr>
<tr>
<td>56/N/R</td>
<td></td>
<td>Agrammatic, L hemianopsia, agnosias, anosognosia, pseudobulbar aphasia</td>
<td>CVA/7 yrs</td>
<td>53/N/L</td>
<td></td>
<td>Mild receptive aphasia, anosognosia, partial right inferior quadrantanopsia</td>
<td>CVA/9 yrs</td>
</tr>
<tr>
<td>54/N/L</td>
<td></td>
<td>Severe hemi aphasia, residual hemianopsia, agnosias, anosognosia, right inferior quadrantanopsia</td>
<td>CVA/2 yrs</td>
<td>51/N/R</td>
<td></td>
<td>L decreased proprioception, 3 point discrimination, aphasia, agrammatia</td>
<td>CVA/9 yrs</td>
</tr>
<tr>
<td>60/N/L</td>
<td></td>
<td>Normal</td>
<td></td>
<td>60/N/L</td>
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<tr>
<td>55/N/L</td>
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<td>Normal</td>
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Fig. 1. a: averaged lesion location across patients with lesions centered in posterior superior temporal gyrus and caudal inferior parietal lobule (STG, n = 9). b: averaged lesion extent across patients with lesions centered in inferior parietal lobule (IPL, n = 6). The 7 lines through the lateral reconstruction indicate the 7 axial sections shown for each group. The scale indicates the percentage of patients in each group having involvement of that brain region.
reaction in the recording chamber (1), marked amplitude asymmetry over plastic craniotomy plate (1) and uncertainty as to lesion localization (1). The remaining patients consisted of 2 groups. The first group consisted of 6 patients with unilateral lesions centered in rostral IPL and inferior portions of superior parietal lobe (52 ± 11 years). The second group was composed of 9 patients with unilateral lesions centered in the STG but extending into adjacent caudal IPL in most subjects (57 ± 12 years). Clinical details of both patient groups are reported in Table I.

Lesions were reconstructed from CT scans in all patients. The CT scans were transcribed onto templates drawn from brain sections at the same angle (0°) as the CT scans (De Armond et al. 1976). Reconstructions were performed independently by two of the authors (RTK and DLW) and were found to be in agreement. The axial templates were then projected onto a lateral view of the brain by computer software developed in our laboratory (see Fig. 1a and b).

Other workers have attempted to relate the CT lesions to the monkey auditory structures reported in the superior temporal plane and lateral superior temporal gyrus (Pandya and Seltzer 1982). However, since the locations of AI, AAI and AAII are uncertain in humans, we prefer to report the CT data in Brodmann terminology. It should also be noted that the CT scan is inaccurate in gauging the amount of damage to temporal-parietal junction structures and MRI imaging will be the preferred imaging method in future patient research.

The STG group had lesions which involved the posterior one-half of the superior temporal gyrus and had substantial extension into the medial superior temporal plane. The group averaged lesion centered in areas 41, 42, posterior area 22 and included significant portions of inferior supramarginal (area 40) and angular gyrus (area 39). Some subjects had involvement of areas 21, anterior 22, 37, 19, 7, 5, and postcentral gyrus (3, 1, 2). The IPL group centered in rostral supramarginal (40) and angular (39) gyri and inferior area 7. Some subjects had extension into areas 5, 3, 1, 2, 19 and caudal areas 40 and 39. No IPL subjects had involvement of auditory koniocortex and only 1 subject had involvement of posterior area 22 (Eidelberg and Galaburda 1984). We chose predominantly vascular lesions restricted to the posterior branches of the middle cerebral artery (MCA) since larger MCA lesions would likely involve insular, basal ganglia, prefrontal and anterior temporal lobe structures. As can be seen in Fig. 1a and b, these structures and the hippocampus are spared in our patients.

Results

Control N1a

An early negative potential peaking at 78 msec was most prominent over the T3 electrode in control subjects (peak voltage: T3 = −1.36 µV, T4 = −0.53 µV, F(1, 16) = 23.28, P < 0.008, see Fig. 2). Although it is not revealed in the group averaged tracings, a clear N1a deflection was observed over T4 in some of the controls. N78 amplitudes recorded at T3 and T4 were unaffected by ear of stimulation.

Control N1b

After exclusion of the T1 and T4 electrodes from scalp topography analysis, a typical fronto-central maximal vertex component (N1b) peaking at 94 msec was recorded (F1 = −5.05 µV, Cz = −5.07 µV; F(1, 192) = 30.36, P < 0.001 for scalp distribution). The N1b was enhanced (by 9%) at scalp sites contralateral to the ear of stimulation (F(1, 192) = 4.68, P < 0.05).

Control N1c

A prominent negative potential peaking at 127 msec was recorded from both T3 and T4 electrodes (mean amplitude over temporal sites = −1.56 µV). The N1c could also be seen in some subjects on the trailing edge of the N1b in fronto-polar electrodes. Unlike the N1a, the N1c was markedly enhanced in the temporal electrode contralateral to the ear of stimulation (right ear T3 = −1.85 µV, T4 = −1.36 µV, left ear T3 = −1.06 µV, T4 = 1.98 µV; F(1, 16) = 18.97, P < 0.001, see Fig. 2).

Control P45

A positive potential peaking at a mean of 45 msec was recorded. This potential was maximal at
CONTROLS

Fig. 2. Grand average AEPs for controls with responses from both left and right ear stimulation presented. The temporal electrodes for both controls and patients are presented at a different gain than for the central sites. The N1a is seen clearly only over the T3 site in controls and the P45 is maximal over T4. The N1c is enhanced contralaterally to the ear of stimulation in controls. Note the different calibration scales for temporal and central sites.

fronto-central scalp sites (FPz = -0.02 μV, Fz = 0.41 μV, Cz = 0.79 μV, Pz = 0.24 μV; over scalp sites *F* (1, 224) = 5.69, *P* < 0.025). As can be seen in Fig. 2 this potential had a discrete maximum at the T4 site (T3 = 0.60 μV, T4 = 1.11 μV). A specific comparison between T3 and T4 confirmed this focal enhancement over the right temporal site; *F* (1, 16) = 6.46, *P* < 0.02. No ear of delivery effects were observed.

Control P200

A typical fronto-central maximal P2 peaking at 213 msec was recorded (FPz = 2.03 μV, Fz = 3.74 μV, Cz = 4.67 μV, Pz = 2.95 μV; for scalp site *F* (1, 224) = 16.03, *P* < 0.001). In contrast to the N1b, no enhancement of the P2 was observed over the hemisphere contralateral to the ear of stimulation.

STG N1a

This potential peaked at 68 msec in the STG group. Since the N1a was prominent over the left hemisphere of controls, a subset of 5 left STG patients was initially analyzed. Unlike controls, no significant lateralized increase of the N1a over the T3 electrode was apparent in this subset of STG patients. A direct comparison of the mean amplitude at temporal sites of the N1a in controls and all STG patients (*n* = 9) revealed that the N1a was significantly reduced in the patient group mainly due to a reduction from the electrode ipsilateral to the STG lesion (*F* (1, 24) = 4.89, *P* < 0.04, see Fig. 3). In this analysis the data were pooled as a function of the temporal electrode either ipsilateral or contralateral to the lesioned STG. For instance, in Fig. 3 the T3 electrode represents the summed activity of responses from the T3 electrode in left STG patients and the T4 electrode from right STG patients.

STG N1b

Unilateral STG lesions produced a generalized reduction of the N1b (*F* (1, 24) = 5.70, *P* < 0.024) which was more pronounced at midline sites (at Cz, controls = -5.07 μV, STG = -3.06 μV; see Fig. 3). There was a reduction of negativity in the N1b latency range in parasagittal leads over the le-
sioned hemisphere (mean of 13%) but specific comparisons did not reach significance (Cl = -2.20 μV, Cc = -2.52 μV, Fi = -2.59 μV, Fc = -3.10 μV, P = n.s.). Latencies were comparable between control and STG patients (both 94 msec).

**STG N1c**

The STG N1c peaked at 127 msec, identical to controls. The N1c was also analyzed as a function of whether the temporal electrode was ipsilateral (Ti) or contralateral (Tc) to the STG lesion. The N1c was reduced by 85% in the temporal electrode over lesioned STG (for peak amplitude: Ti = -0.21, Tc = -1.38, F(1, 8) = 7.70, P < 0.025, see Fig. 3). Direct comparison of controls and STG patients also revealed significant group differences in N127 amplitude due to a unilateral reduction of the N127 over lesioned STG (for group × location; F(1, 24) = 6.42, P < 0.02). In a further specific comparison neither the N1a nor the N1c were reduced in the fronto-polar electrode over lesioned hemisphere.

**STG P45**

Unilateral STG lesions reduced the P45 to noise levels in most subjects. Inspection of Fig. 3 reveals P45 components in the 0.2 μV range at coronal sites. Inspection of a subgroup of 4 STG subjects with extensive right-sided lesions confirmed complete unilateral abolition of the P45 at the T4 site. In comparison to controls, the P45 had a significant reduction in the 9 STG patients over all scalp sites (F(1, 24) = 4.96, P < 0.03, see Fig. 3). A specific comparison of the P45 in controls and STG patients at temporal sites only did not reach significance (F(1, 24) = 3.04, P < 0.09).

**STG P200**

The P2 peaked at 208 msec in the STG group and had a fronto-central scalp distribution (F(1, 112) = 5.60, P < 0.025), Fp2 = 1.56 μV, Fz = 2.44 μV, Cz = 2.83 μV, Pz = 1.76 μV). No lateralized reduction of the P2 was observed over lesioned hemisphere (Fi = 2.28 μV, Fc = 2.21 μV; Ci = 2.33

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Fig. 3. Grand average AEPs for IPL and STG patients with the data collapsed over replications and over left and right ear stimulation. For the patients, the data are presented as a function of scalp electrode either over lesioned hemisphere (i.e., Ti = T4 for left plus T4 for right lesioned patients) or uninvolved hemisphere (Tc). STG lesions produced focal reductions of the N1a and N1c only over lesioned hemisphere (see Ti site) and a bilateral reduction of the N1b and P45.
μV, Cc = 2.22 μV). In comparison to controls the P2 revealed a trend towards reduction (controls Cz = 4.67 μV, STG Cz = 2.83 μV) but this did not reach significance (F(1, 24) = 3.70, P < 0.063, see Fig. 3).

**IPL N1a**

No clear reduction of the N1a amplitude for either peak or mean amplitude measures was observed in patients with unilateral IPL lesions either over lesioned hemisphere or in comparison to controls (peak amplitude across temporal electrodes; IPL = −0.94 μV, controls = −0.95 μV).

**IPL N1b**

The latency of the N1b was slightly prolonged by IPL lesions (mean 100.2 msec) but this did not reach significance in relation to controls (F(1, 21) = 4.00, P < 0.056). Although amplitude reductions were observed, neither mean nor peak amplitude measures revealed significant reduction of the N1b by IPL lesions (at Cz controls = −5.07 μV, IPC = −3.72 μV; F(1, 21) = 2.10, P = n.s.). No unilateral reduction of the N1b was observed over lesioned hemisphere.

**IPL N1c**

In contrast to STG lesions, no unilateral reduction of the N1c was observed over lesioned hemisphere. Unilateral IPL lesions produced comparable reductions of the N1c over both Ti and Tc electrodes but these did not reach significance (peak amplitude controls T = −1.45 μV, T = −1.67 μV, IPL Ti = −0.90 μV, Tc = −1.04 μV, F(1, 21) = 2.19, P < 0.15, see Fig. 3).

**IPL P45**

The P45 peaked at 49 msec in the IPL group which was not significantly different from controls. The component had a fronto-central distribution (FPz = 0.42 μV, Fz = 0.58 μV, Cz = 0.77 μV, Pz = 0.36 μV), but this scalp topography difference did not reach significance. In a direct comparison between controls and IPL patients, no significant amplitude decrease of the P45 in IPL patients was observed (IPL, Cz = 0.77 μV, controls Cz = 0.79 μV, see Fig. 3).

**IPL P200**

The P2 peaked at 213 msec in the IPL group and had a fronto-central scalp distribution (FPz = 1.42 μV, Fz = 2.21 μV, Cz = 2.92 μV, Pz = 1.99 μV, F(1, 70) = 5.59, P < 0.25). Although a trend towards reduction similar to STG lesions was observed, no significant reduction of the P2 was noted in a direct comparison to controls (controls, Cz = 4.67 μV, IPL, Cz = 2.92 μV).

**Discussion**

The AEP findings in controls and patients provide further evidence of activation of multiple neural generators during the 40–130 msec following auditory stimulation and focus on the critical role of the lateral STG and superior temporal plane in generation of these potentials. Similar to numerous previous reports in controls, vertex maximal N1b (N1 or N94) and P2 (P200) potentials were recorded with the N1b revealing an increased amplitude in electrodes situated over the hemisphere contralateral to the ear stimulated. In addition to the N1b and P2, additional AEPs (N1a, N1c, P45) were recorded predominantly over temporal recording sites in accord with prior reports of focal temporal AEP activity in this latency range (Wolpaw and Penny 1975; McCallum and Curry 1979, 1980; Wood and Wolpaw 1982; Perrault and Picton 1984; Cacace and Wolpaw 1987).

The N1a temporal component peaked at 78 msec and was most prominent in the left temporal recording electrode in controls. The latency is in agreement with prior reports of 74 and 75 msec latencies for the temporal N1a (McCallum and Curry 1979, 1980; Perrault and Picton 1984). It is unlikely that this potential reflects a myogenic artifact since its latency was longer than reported stimulus evoked myogenic activity (Picton et al. 1974). The finding that the N1a was larger over the dominant hemisphere is in accord with the results of McCallum and Curry (1979, 1980). The functional significance of the lateralization of the N1a to the dominant hemisphere is uncertain although several anatomical considerations are apparent. For instance, gyral (Geschwind and Levitsky 1968), cytoarchitectonic (Galaburda et al.
and sylvian fissure asymmetries (Rubens et al. 1976) between human left and right superior temporal planes are well described. The N1a asymmetry may simply be related to dipole orientation differences in left and right auditory regions in our population. However, the N1a could also reflect a more fundamental underlying physiological difference between left and right superior temporal plane structures, such as those related to dominant hemisphere dichotic listening superiority (Sparks et al. 1970) and language comprehension (Lester et al. 1986).

In controls, the N1c potential peaked at 127 msec and had a focal distribution centered over both left and right temporal electrodes with the N1c scalp topography extending to fronto-polar and lateral central electrodes in some subjects. The N1c amplitude was enhanced in the temporal site contralateral to the ear of stimulation with peak amplitude measures revealing 88% enhancement of the N1c. The N1c latency, scalp topography and ear effects are all in agreement with prior reports (McCallum and Curry 1979, 1980; Perrault and Picton 1984). The focal distribution centered over temporal sites combined with the marked enhancement in scalp sites contralateral to the ear of stimulation is compatible with a radially oriented dipole situated in the lateral STG as suggested by dipole modeling studies (Scherg and Von Cramon 1985, 1986).

A positive potential peaked at 45 msec in controls and had a fronto-central scalp distribution. In addition to this fronto-central maximum a discrete focal enhancement for the P45 was observed at the T4 site identical to the results reported by Cacace and Wolpaw (1987). As for the N1a, no ear of delivery effects were noted for the P45. The focal maximum of the P45 at T4 could be due to a relative reduction of N1a activity at the right T4 site. However, the results of intracranial recording indicate that a distinct positivity peaking at 40 msec and negativities at 63 and 142 msec are generated in the perisylvian STG region (Celesia 1976). These intracranial potentials may correspond to the lateral scalp P45, N1a and N1c potentials. The P45 results thus provide further electrophysiological support for an asymmetric organization of human temporal lobe.

The results of unilateral STG lesions provide further support for the interpretation of the control P45, N1a and N1c data. Unilateral STG lesions resulted in reduction of both the N1a and N1c recorded from the temporal electrode situated over lesioned hemisphere indicating that both the N1a and N1c are generated by neural structures located in the lateral superior temporal gyrus. Therefore, both the scalp topography information in controls and the effects of STG lesions are in accord with the proposal that radially oriented STG dipole sources generate the surface recorded N1a and N1c potentials. Unilateral STG resulted in widespread reduction at central sites of the P45 in addition to complete abolition of this potential at the temporal site over lesioned hemisphere. The findings in STG patients coupled with the scalp topography results in controls indicate that the P45 is more widely distributed in the STG with extension into the medial superior temporal plane producing a fronto-central peak and lateral STG extension producing the discrete T4 maximum.

Since the STG lesions covered variable amounts of AI, AII and other auditory regions, we cannot be certain as to the precise STG sources of the P45, N1a or N1c although their radial orientation suggests they may arise in auditory association cortex (Galaburda and Sanides 1980). Use of MRI scans with their accompanying enhanced gyral resolution would be useful in improved delineation of damage in sub-regions of STG. However, further caution in cytoarchitectonic interpretation of STG lesion data is suggested by recent anatomical data reporting that the human AI region is distributed throughout anterior and posterior STG and may even extend into caudal inferior parietal regions (Galaburda and Sanides 1980; Eidelberg and Galaburda 1984). The relative preservation of the N1a and N1c at the fronto-polar site over lesioned hemisphere after posterior STG lesions sparing anterior STG provides some support for this notion of a more widely distributed AI in the STG of humans.

Whereas the N1a and N1c were reduced only in the temporal electrode over lesioned hemisphere, unilateral STG lesions produced a bilateral reduction of the N1b, a finding previously reported by our laboratory (Knight et al. 1980). At least two
interpretations of this bilateral effect on the N1b by unilateral STG lesions are compatible with the data. The vertex N1b may be due to the summation of vertically and slightly medially oriented dipoles in the STG as recently proposed by dipole modeling and patient studies (Scherg and Von Cramon 1986). A unilateral STG lesion would reduce the N1b by about one-half at the vertex and result in minor hemispheric asymmetries of the degree found in the current study (13% at parasagittal sites). This interpretation agrees with initial suggestions by Vaughan and Ritter (1970) of a major superior temporal plane source of the vertex N100, with the more recent dipole modeling studies (Scherg and Von Cramon 1985, 1986) which predict about a 9% reduction of N1b at parasagittal sites and with neuromagnetic data (Hari et al. 1982). However, the results could also be explained by loss of a facilitatory effect of STG structures on a midline subcortical N1b generator. Complicating this issue is the fact that multiple generators likely contribute to the N1b response with evidence suggesting sources in supplementary motor cortex, cingulate cortex, thalamus, reticular formation and hippocampus in addition to superior temporal plane (see Näätänen and Picton 1987 for an extensive review).

However, whereas the data indicate that a significant portion of the N1b arises in medial STG, the current data strongly support a lateral STG source of both the N1a and N1c and more widely distributed medial and lateral STG sources of the P45. Future research on long latency AEPs should consider treating temporal sites independently from midline sites. Indeed, prior reports of substantial asymmetries of the N1b over lesioned hemisphere may have been due in part to inclusion of temporal sites in the data analysis (Peronnet et al. 1974; Peronnet and Michel 1977). Asymmetries in the N1a and N1c after unilateral lesions encompassing STG would produce a false decrease in negativity in the N1b latency range at temporal and other far lateral sites.

In contrast to the marked effects on the P45, N1a, N1b and N1c, neither STG or IPL significantly reduced the P2 as has been previously reported (Knight et al. 1980). However, there was a definite trend towards reduction in the STG group and study of patients with more extensive lesions encompassing anterior and posterior STG will likely reveal an important role of the STG in P2 generation.

A different pattern of effects on long latency AEPs was noted in patients with unilateral IPL lesions. Neither latency nor amplitude of the P45 or N1a was altered by IPL lesions. However, there was a trend in the direction of a bilateral reduction of the N1b, N1c and P2 by IPL lesions although none of these components were unilaterally reduced over the IPL lesioned hemisphere. Even if auditory structures extend into IPL as recently suggested (Galaburda and Sanides 1980), it is difficult to model a bilateral N1b reduction by a unilateral IPL lesion since the majority of the IPL cortex has neuronal dipole sources oriented orthogonally to the vertex. It is conceivable, however, that portions of the N1b and N1c generators are situated medially in the temporal-parietal junction at the caudal portion of the IPL. If this were true, an argument similar to that for bilateral N1b reduction by unilateral STG lesions could apply for unilateral IPL lesions. A more parsimonious explanation of possible IPL lesion effects on the N1b and N1c is that the IPL exerts a facilitatory influence on these generators. There are ample anatomical data in primates documenting direct IPL to STG pathways and additional callosal interparietal connections (Selzter and Pandya 1984; Caminiti and Sbriccoli 1985). Furthermore, the IPL has extensive connections to limbic, thalamic and reticular regions (Mesalam et al. 1977; Weber and Yin 1984). Thus, neural pathways exist by which a unilateral IPL lesion could influence either an STG or a subcortical N1b generator. It should be emphasized that these IPL effects are speculative since significant effects were not obtained in the present study encompassing only 6 IPL patients. A more extensive study employing a larger patient base with MRI delineation of degree of gyral involvement in critical temporal-parietal structures engaged during generation of long latency AEPs will be needed to assess this issue.

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References


