BITEMPORAL LESIONS DISSOCIATE AUDITORY EVOKED POTENTIALS AND PERCEPTION

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Bitemporal damage of auditory cortex in man can produce the striking auditory-perceptual deficits of cortical deafness in which the processing of auditory stimuli is disrupted despite intact peripheral auditory function and intact processing of stimuli in other modalities (Auerbach et al. 1982). Studies of long-latency auditory evoked potentials (AEPs) in such cases have provided conflicting evidence about the relationship of evoked potentials to perception. In some studies, AEPs have vanished in parallel with perceptual impairments (Jerger et al. 1969; Michel et al. 1980), while in others evoked potential abnormalities have been more subtle — despite equally striking audiological deficits. For example, Goldstein et al. (1975) recorded click-evoked AEPs in a cortically deaf patient with a variety of audiological symptoms. Although audiometric thresholds were greatly elevated in this patient, EPs were recorded ‘…at click intensities similar to those just effective in evoking these electrical responses in normal subjects.’ However, the responses were delayed and of subnormal amplitude. Earnest et al. (1977) also reported that auditory evoked potential thresholds were only slightly elevated in a patient with greatly elevated perceptual thresholds following bitemporal infarcts. Parving et al. (1980) recorded late responses with normal latencies, and only ‘minor deviations in waveform morphology’ in a case with bitemporal lesions and marked auditory agnosia. In a similar case, Rosati et al. (1982) recorded N70-P110 components whose amplitudes were comparable to N80-P130 amplitudes recorded in control subjects.

Because brain-stem and diencephalic processing of auditory signals persist in cortically deaf patients, they may retain rudimentary auditory-perceptual capacity, despite an inability to understand speech or to discriminate complex auditory inputs (Leicester 1980). It has been suggested that in some of these cases AEPs may be generated in subcortical auditory centers (Chocholle et al. 1975). However, a variety of other evidence suggests that the auditory cortical regions play an active, and perhaps necessary role in the generation of long-latency AEPs (Vaughan and Ritter 1970; Wood and Wolpaw 1982). However, the mechanisms of AEP generation and particularly the role of primary auditory cortex remain in dispute. Although unilateral lesions of temporal and parietal cortex reliably reduce AEP amplitudes, in different studies the reductions have been ipsilateral (Michel et al. 1973), predominantly bilateral with a slight ipsilateral preponderance (Szirtes et al. 1980), or bilateral and symmetrical (Knight et al. 1980). Two theories have been advanced based on these conflicting results: (1) primary auditory cortex is itself the generator of long-latency AEPs (Michel et al. 1976); and (2) primary auditory cortex plays little or no direct role in AEP generation, but is close to parietal-temporal areas which modulate AEP generators (Knight et al. 1980).

We recently had the opportunity to study a cortically deaf patient who had been referred to us after initial screening suggested the presence of long-latency AEPs. We were interested in determining the extent to which the AEP generators had been compromised, and the extent to which they would show unusual morphological and functional properties which might be related to the unmasking of AEPs from subcortical centers. We also investigated the extent to which the AEPs
correlated with the remaining perceptual capacities of the patient.

Case Report

The patient (A.B.) was an 82-year-old woman who had suffered successive strokes of the right and left temporal lobes. The first (in 1976) left her with increased stretch reflexes, weakness of the intrinsic hand muscles and extensor plantar responses on the left side. Auditory and linguistic functions were unaffected. In 1979 a second stroke occurred while the patient was vacuuming. It left her initially dazed, confused and totally deaf. When her son returned from work she was unable to understand what he said, but understood gestures and written messages. She was unaware of sounds, and failed to notice that she had left the vacuum cleaner running.

She was immediately taken to the hospital where upon admission she was confused and unresponsive to spoken commands, but could respond to written commands. She showed no motor symptoms other than a slight weakness and hyperreflexia on the left, dating from her previous infarction. Auditory thresholds could not be measured. However, brain-stem auditory evoked potentials showed normal amplitudes and latencies. In addition, binaural click stimuli presented at 55 dB above the threshold of the attending resident produced long-latency AEPs with prominent P1 and N1 components.

Computerized tomography obtained 1 week after the second admission (Fig. 1) revealed an extensive lesion of the right temporal lobe (from the 1976 episode) extending to the temporal-parietal junction and a new, low density, contrast enhancing lesion of the left superior and middle temporal gyri. Damage extended to the sylvian fissure on both sides and included primary auditory cortex (Heschl’s gyrus) bilaterally. The lesions were consistent with bilateral occlusion of the posterior temporal branch of the middle cerebral artery.

Behavioral testing

We examined A.B. as an outpatient on 4 separate occasions during the next 3 months. We communicated to her through writing. She was

Fig. 1. CT scans from patient A.B. Sections shown begin at the inferior temporal lobe (top left) with higher sections displayed from left to right. Dark regions reveal the extensive bilateral damage of temporal lobe structures, particularly evident in collicular level cuts.
PATIENT A.B.

--- SUBTHRESHOLD
SUPRATHRESHOLD

1000 Hz TONE

--- SUBTHRESHOLD
SUPRATHRESHOLD

500 Hz TONE

--- SUBTHRESHOLD
SUPRATHRESHOLD

110 Hz COMPLEX TONE

--- SUBTHRESHOLD
SUPRATHRESHOLD

500 Hz COMPLEX TONE

--- SUBTHRESHOLD
SUPRATHRESHOLD

'a'

--- SUBTHRESHOLD
SUPRATHRESHOLD

'DOG'

--- SUBTHRESHOLD
SUPRATHRESHOLD

SUPER AVERAGE

--- SUBTHRESHOLD
SUPRATHRESHOLD

CONTROL

--- SUBTHRESHOLD
SUPRATHRESHOLD

Fig. 3. Left: auditory evoked potentials at the vertex elicited by different stimuli presented at suprathreshold (80 dB nHL. solid line) and subthreshold (55 dB nHL. dashed line) intensities to patient A.B. Right: AEPs elicited in a control subject by the stimuli presented in the suprathreshold condition (80 dB nHL).
Results

Experiment 1

All of the stimuli in experiment 1 — including those below the patient’s perceptual threshold — produced well-defined long-latency AEPs (Fig. 3, left). These consisted of prominent P1 (suprathreshold latency 57 msec), N1 (98 msec) and P2 (185 msec) components. Small and variable sustained potentials (SP) and potentials at stimulus

Fig. 4. ERPs elicited from patient A.B. at different scalp sites, referenced to linked mastoids each dropped across a 10 kΩ resistor. Responses are averaged over the 6 stimuli shown in Fig. 3. Solid lines are responses to suprathreshold stimuli, dashed lines to subthreshold stimuli. AEPs are shown in proximity to the recording electrode site. AEPs from Wernicke’s area and its right hemisphere homolog (W1 and W2) are shown posterior to T3 and T4. Responses at the eye and balanced non-cephalic sites are shown in insets.
provide an explanation (Fig. 4). Compared to the balanced non-cephalic response (Fig. 4, bottom left), the nose and T3 have N1s of comparable amplitude, whereas amplitudes are smaller at T4. Thus, subtracting the AEPs recorded at the nose cancels the AEPs over the left temporal area and inverts them over the right. Voltage gradients at the N1 peak were comparable over the two hemispheres.

**Experiment II**

The results of experiment II, showing the recovery properties of the N1-P2, are presented in Fig. 6. At the longest ITI N1-P2 amplitudes were

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**Fig. 6.** Recovery cycle of the N1-P2 for patient A.B. averaged over responses to trains containing 1 and 2 kHz tone bursts. Mean N1-P2 amplitudes are shown normalized with respect to amplitudes elicited by the first stimulus following a 6.0 sec ITI (top left). The solid line shows the recovery cycle for trains which contained stimuli separated by 1.0 sec ISIs; the dashed line shows comparable data for trains with 0.5 sec ISIs. Representative wave forms are shown as insets. Note that the averaging epoch (1.2 sec with 0.2 sec prestimulus baseline) contains the responses to two successive tones at the shorter ISIs (bottom center). On the top right is the ERP evoked at the end of a 1.0 sec ISI train by tones of different frequencies (either 500 Hz tones in the 1000 Hz trains or 1000 Hz tones in 500 Hz trains). Shown on the lower right are ERPs to deviant auditory stimuli (digitized vocalizations).
between perception and evoked potential indices of perception may exist in other modalities. For example, long-latency visual evoked potentials have been recorded in 'cortically blind' patients (Kooi and Sharbrough 1966; Bodis-Wollner et al. 1977; Spehlmann et al. 1977; Celesia et al. 1980).

The dramatic dissociation between perceptual reports and auditory evoked potentials could occur in several ways. First, the N1 and P2 could reflect the functioning of a system which might be necessary — but not sufficient — for auditory discrimination and report. For example, an island of auditory cortex might have remained intact (thus producing the electrophysiological response) but have been isolated from surrounding brain regions necessary for stimulus evaluation and response. Although primary auditory cortex and much of auditory association cortex appeared to have been destroyed in our patient, this possibility cannot be ruled out because the CT scan averages electron density across each slice (10 mm). In this regard, the examination of brain metabolic activity in a cortically blind patient who showed intact visual evoked potentials recently revealed preserved and metabolically active striate cortex which had not been evident on CT scan (Celesia et al. 1982). If an island of auditory cortex were preserved in our patient, it might either have participated directly in the generation of the long-latency AEPs which we observed, or maintained projections to surrounding secondary and association cortex which in turn were involved in AEP generation. However, in either case the intact portion of auditory cortex would have to be capable of producing AEPs with normal amplitudes and normal, symmetrical scalp distributions, while being unable to sustain perceptual functions. This would suggest greater redundancy in the long-latency AEP generating systems than in those responsible for perception. It is also possible that parieto-temporal regions distinct from Heschl's gyrus which receive direct connections from the medial geniculate body (Galaburda and Sanides 1980) may have contributed to N1-P2 generation. Indeed, long-latency auditory evoked potentials have been recorded from exposed perisylvian cortex in cats and humans, which persist following removal of primary auditory cortex (Buser et al. 1959; Celesia et al. 1968; Buchwald et al. 1981).

It is also possible that the bitemporal lesions unmasked abnormal responses of subcortical auditory centers. However, the normal properties of the AEP — including amplitude, latency, scalp topography and recovery functions — argue strongly against this possibility.

A third explanation is that the normal vertex potential is not dependent upon the integrity of primary auditory cortex, but depends upon the integrity of nearby polysensory cortical regions. This would explain why certain lesions can reduce AEP amplitudes without damaging primary auditory cortex or altering fundamental auditory perceptual processing (Knight et al. 1980), while others, as in the present case, alter auditory perception without affecting AEPs. Of course, some lesions may damage both systems (e.g. Michel et al. 1980). In fact, in cases of cortical deafness where anatomical data are present and N1-P2s are small or absent, damage appears to extend into areas posterior and superior to auditory cortex (Jerger et al. 1969; Adams et al. 1977; Michel et al. 1980; Miceli 1982). Variable results in studies of cortical deafness derive from the fact that damage is rarely restricted to primary auditory cortex because branches of the middle cerebral artery, whose occlusion is implicated in most cases of auditory agnosia, also supply portions of the frontal, parietal and inferior temporal lobes.

What cortical regions near to primary auditory cortex might be involved in N1-P2 generation? Fig. 5 shows the superposition of lesion reconstructions in patient A.B. and in a previous group of patients (from Knight et al. 1980) who, unlike A.B., showed bilateral reductions in N1 amplitude. The region damaged in those patients but spared in A.B. appears to be area 39, the angular gyrus. Participation of the angular gyrus in N1 generation is plausible on other grounds. For example, tone-evoked N1s are reduced in amplitude if auditory stimuli are preceded by visual, electric, or vibrotactile stimuli (e.g. Hay and Davis 1971), and the angular gyrus appears to be fully capable of mediating such multi-modal integration (Geschwind 1965).
References


