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
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...
eager to cooperate and gave her consent to the testing procedures.

Her general intellectual status was good. She accurately reproduced 3 part motor commands after a 5 min interval, but showed mild deficits in long term memory for visually presented nouns which improved following semantic cuing. Her writing contained occasional repetitions (e.g. '...I am so trying trying'), and provided some evidence of reduced content and fluent aphasia (e.g. '...I have heard say what he was going to what I hear...'). Her speech was loud and fluent, with normal grammar, content and syntax, and occasional paraphasic errors. She distinguished left from right and performed calculations with one and two digit numbers.

Over the 3 months following the insult her deafness resolved somewhat and she became able to detect extremely loud sounds, although other fundamental auditory processing abilities remained compromised. For example, she showed no startle response when an experimenter positioned behind her clapped his hands or yelled her name. On informal testing she made gross errors (25–40°) in sound localization, although she could usually identify the hemifield of stimulation.

The patient remained unable to discriminate among the sounds that she could hear. She was unable to understand spoken sentences, either with or without lip reading. She could neither repeat nor transcribe individual words or letters of the alphabet and identified monosyllables at chance levels whether responding orally or by pointing to written syllables. She was also unable to discriminate male from female voices, nor could she discriminate different pitches of the same voice.

Her comprehension problems were evident for non-verbal as well as verbal material. For example, she was unable to identify common melodies or environmental noises. She failed to match environmental noises to pictures of noise sources. She was unable to discriminate ('same-different') among environmental sounds with one exception — on several occasions she discriminated the noise of keys jangling from the noise of hand claps. She was also incapable of discriminating rhythmic patterns. For example, she could not accurately determine how many claps occurred during a short interval, although she could frequently distinguish one clap from two.

Fig. 2 shows the audiometric thresholds which were obtained immediately prior to electrophysiological testing (3 months after the second incident). Thresholds were determined by averaging values obtained with ascending and descending stimulus intensities. Test (solid line) and re-test (dashed line) audiograms are shown for each ear.

**Fig. 2.** Pure tone audiograms from patient A.B. obtained 3 months after hospital admission. Thresholds were obtained by averaging values obtained using ascending and descending stimulus intensities. Test (solid line) and re-test (dashed line) audiograms are shown for each ear.

Electrophysiological testing

**Brain-stem auditory evoked potentials (BAEPs).** Monaural rarefaction clicks were presented at rates of 10/sec (70 dB) and 50/sec (80 dB). The EEG was recorded from electrodes affixed to the vertex and mastoid process ipsilateral to the stimulated ear, amplified (bandpass 10–3000 Hz) and stored on analog magnetic tape for off-line analysis.
BAEPs were averaged over a 10 msec time-base (sampling rate = 25 kHz) using a commercially available signal averager, plotted out on an X-Y recorder and measured by hand.

The BAEPs indicated unimpaired peripheral hearing. For example, following stimulation with 70 dB nHL clicks (referenced to the threshold of a listener with normal hearing), wave V latencies fell within the short normal range (5.50 and 5.75 msec following left and right ear stimulation, respectively). The results showed little change when compared with those which had been obtained immediately after hospital admission. At that time, wave V latencies following 70 dB nHL stimulation were 5.60 and 5.70 msec for the left and right ears respectively.

**Vertex potential recording.** The EEG was amplified from 10 scalp sites (Cz, C3, C4, Fz, F3, F4, T3, T4, and electrodes over Wernicke’s area and the corresponding regions of the right hemisphere, located 12.5% posterior to vertex and 35% of the interaural distance laterally) each referenced to linked mastoids dropped across 10 kΩ resistors. An electrode below the left eye monitored eye movements. Because of the controversy over the activity of different reference sites, additional recordings were obtained from the tip of the nose and from a balanced non-cephalic reference (Lehtonen and Koivikko 1971; Wolpaw and Wood 1982). Following amplification (bandpass 0.01–100 Hz) the EEG was stored on FM tape, and digitized off-line on a general purpose computer. The digital EEG was scanned for artifacts, including EMG and EOG activity, and amplifier clipping. Artifact-free trials were averaged, plotted and quantified automatically.

The first experiment was designed to determine how reliably vertex potentials would be evoked by different supra- and subthreshold stimuli. Six different stimuli were presented in random sequence. Each was 600 msec in duration including 10 msec rise and fall times. The stimuli (which had been digitized or synthesized by computer and recorded on audio tape) included 2 pure tones (500 and 1000 Hz), 2 complex tones (respectively 110 and 500 Hz fundamentals with 5 harmonics of equal energy), and 2 one-syllable words (‘a’ and ‘dog’). They had been matched in loudness by a normal listener. Inter-stimulus intervals (ISIs) were fixed at 1.5 sec.

Two stimulus intensities were used. In the **suprathreshold condition** the stimuli were presented binaurally at 85 dB nHL. At this intensity the patient was able to indicate when a stimulus occurred by raising her finger, but was unable to discriminate them.

In the **subthreshold condition**, stimuli were presented monaurally to the patient’s right ear at 55 dB nHL. Judging from her audiogram, these ranged from 10 dB (for the 1000 Hz tone) to 26 dB (for the 500 Hz tone) below the patient’s perceptual threshold, and she was unable to detect any of them. The results were compared to those obtained in responses to the same stimulus sequence from a 28-year-old control subject. The subject was preselected and known to have normal audiometric functioning and representative AEPs.

The second experiment was designed to characterize the refractory properties of the vertex potential. Stimuli consisted of trains of tones separated by 2.0 or 6.0 sec inter-train intervals (ITIs). Within each train inter-stimulus intervals (ISIs) were fixed at either 1.0 or 0.5 sec. ITIs between trains and ISIs within trains varied in a random manner. Eight stimuli were presented in each train. The first six were identical tones (either 500 or 1000 Hz in different conditions), each 300 msec in duration with 10 msec rise and fall times. The 7th or 8th tone could be replaced by any of 4 deviant stimuli, including the frequent tone used in the other condition (e.g. 1000 Hz in the 500 Hz condition), a 2000 Hz tone, a digitized human vocalization (‘but’), and the digitized sound of a human scream. The deviant stimuli were edited by computer so that they were 300 msec in duration with rise and fall times and intensities comparable to those of the frequent tones. Deviant stimuli and infrequent tones occurred randomly and independently on 50% of the trials in the 7th and 8th stimulus positions. Stimuli were presented binaurally at intensities of 80 dB nHL. Recording procedures and electrode placements were identical to those of experiment I.
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 I

All of the stimuli in experiment I — 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 (Wl and Wr) are shown posterior to T3 and T4. Responses at the eye and balanced non-cephalic sites are shown in insets.
offset were also evident, mainly in the suprathreshold condition. Although N1 and P2 amplitudes were comparable to those recorded from the young control subject (Fig. 3, right), the wave forms differed in morphology, with A.B. showing a relative P1 enhancement and SP reduction. These changes have been reported in the AEPs of healthy elderly subjects with normal hearing (Pfefferbaum et al. 1979).

Suprathreshold stimuli produced AEPs with slightly larger amplitudes and shorter latencies than subthreshold stimuli. However, there was no apparent relationship between AEP amplitudes and perceptual thresholds for either suprathreshold or subthreshold stimuli. For example, in subthreshold conditions 500 Hz tones (26 dB below estimated threshold) produced AEPs as large as 1000 Hz tones (10 dB below threshold).

Fig. 3 also demonstrates that the subthreshold stimuli elicited N1-P2s with amplitudes and latencies within the range of those elicited in the control subject by the suprathreshold stimuli (80 dB) stimuli (e.g. 500 Hz and 110 Hz complex tones). The amplitudes fell within the high-normal range of data gathered from young controls with normal hearing using stimuli of comparable intensities and durations (Picton et al. 1978a).

The scalp distributions of the patient’s suprathreshold (solid lines) and subthreshold (dashed lines) AEPs are shown in Fig. 4. Both supra- and subthreshold stimuli elicited AEPs with similar scalp topographies. The P1, N1 and P2 were fronto-centrally distributed and comparable to the distributions of normal subjects (e.g. Picton et al. 1978b). A.B. showed slight inter-hemispheric asymmetries of the sort found occasionally in normals. P1, N1 and P2 components tended to be larger over the right temporal lobe, with differences most evident at electrodes over temporal (T3, T4) and temporal-parietal (W1 and Wr) cortex.

The coronal distribution of AEPs referenced to an electrode on the nose is shown in Fig. 5, along with reconstructed lateral views of the lesions. Using this montage the long-latency potentials appear to be inverted at the linked mastoid sites and over the right temporal lobe (Wr and T4). An equivocal inversion is evident over the left temporal lobe. These asymmetries were opposite to those obtained with a linked mastoid reference. The relative amplitudes at the nose and other sites

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**Fig. 5.** Coronal topography of AEPs in patient A.B. when responses were referenced to an indifferent electrode on the nose. AEPs 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 from the vertex (top), from C3, WI and T3 and from comparable right hemisphere placements, and from linked mastoids (bottom). Shading at 135° shows the reconstructed lesions in patient A.B. Shading at 45° shows the reconstructed unilateral lesions shown in a previous study to reduce N1 amplitudes (Knight et al. 1980).
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

![Graph showing recovery cycle of N1-P2](image)

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).
large (13.75 μV), but they declined rapidly with stimulus repetition as in normal subjects (Ritter et al. 1968; Roth and Kopell 1969; Woods and Elmasian 1983). Response amplitudes reached lower levels in trains with 0.5 sec ISIs (about 25% of maximal response amplitudes) than 1.0 sec ISIs (about 50% of maximal responses).

As in normal subjects, the refractory process was found to be partially specific for the stimulus repeated (Butler 1973). For example, following a train of 500 Hz tones, another 500 Hz tone in the 7th or 8th position produced a smaller N1-P2 than a 1000 Hz tone substituted in that position (Fig. 6, upper right). Although A.B. was unable to detect the change in the frequency of the stimulus, the degree of specificity of refractoriness of the N1-P2 was comparable to that seen in normal populations in a similar paradigm (Woods and Elmasian 1983).

Fig. 6 also shows event-related potentials (ERPs) produced by the deviant vocalizations (bottom right). These produced enhanced N1 and P2 components, followed by a small, positive shift onsetting at about 400 msec post stimulus. This slow positivity was fronto-centrally distributed and appeared to reflect the resolution of a negative potential which lasted the duration of the stimulus train. A similar, fronto-centrally distributed positivity of comparable amplitude was observed after non-deviant stimuli which terminated the train.

Discussion

Our patient's audiological symptoms closely parallel those which have been reported in other cases of cortical deafness. The most characteristic deficit is difficulty in discriminating verbal and non-verbal stimuli in the presence of comparatively intact reading, writing and speech production (Clark and Russell 1938; Lhermitte et al. 1971; Chocholle et al. 1975; Oppenheimer and Newcombe 1978; Barraquer-Bordas et al. 1980). Other fundamental auditory abilities are often impaired in cortically deaf patients, including the perception of temporal order and sound localization (Jerger et al. 1969), and these were also impaired in our patient. Less frequently, as in our case, elevated thresholds, absent auditory startle responses, and an impaired ability to discriminate stimulus frequencies are observed (Chocholle et al. 1975). The sudden onset of problems associated with well defined brain lesions, the absence of auditory startle responses and persistent auditory deficits, despite evident motivation for improvement, argue against hysterical deafness in our case. The absence of a P3 wave to deviant auditory stimuli is also consistent with an organic impairment.

In many cases of cortical deafness, significant improvement occurs in auditory functioning (particularly thresholds) over time, with symptoms resolving from 'total deafness' to a slight elevation in pure tone thresholds, but with continued impairments in the ability to discriminate sounds (Jerger et al. 1969; Miceli 1982). Although our patient's pure tone thresholds improved somewhat, they remained greatly elevated 3 months after her second stroke. It is possible that her slow recovery may be related to her advanced age.

Despite the profound auditory symptoms, P1, N1 and P2 components could be elicited by a variety of stimuli and showed normal amplitudes, scalp distributions and refractory properties. Thus, the lesions dissociated perceptual reports from the vertex potential in two ways. First, subthreshold stimuli elicited unequivocal evoked responses. Normally, N1-P2 amplitudes decrease and latencies increase as stimulus intensities are reduced; at 15–30 dB above perceptual threshold the evoked potential disappears altogether. In our patient clear N1-P2s were produced by stimuli 10–26 dB below threshold, and they were neither small nor delayed. In fact, they had amplitudes and latencies comparable to those produced in normal subjects by stimuli 70–90 dB above threshold. This would be consistent with the patient's intact peripheral hearing, as judged from BAEPs, but is at odds with her audiomeric test results. Goldstein et al. (1975) reported similar findings in a case in which audiomeric thresholds were elevated by 50–60 dB but AEPs could be recorded at 5–10 dB. Second, as in normal subjects, the patient's N1-P2 increased in amplitude when stimuli shifted in pitch during an ongoing train, even though stimulus shifts were not perceived. Similar dissociations be-
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 scanner 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 have been 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).
Summary

We studied auditory evoked potentials (AEPs) in an 82-year-old female patient who became suddenly deaf following the second of two strokes. The patient showed markedly elevated pure tone thresholds, was unable to discriminate sounds and could not understand speech. Brain-stem auditory evoked potentials (BAEPs) were normal. CT scans revealed bilateral lesions of the superior temporal plane which included auditory cortex.

Two experiments were performed. In the first, tones, complex sounds and speech stimuli were presented at intensities above and below the patient's perceptual threshold. P1, N1 and P2 components were elicited by each of the stimuli — whether or not they were perceived. In particular, stimuli presented below threshold evoked large amplitude, short latency responses comparable to those produced in a control subject. In a second experiment, the refractory properties of the N1-P2 were examined using trains of tones. They were also found to be similar to those of normal subjects. Shifts in the pitch of the tones near the end of the train (when refractory effects were maximal) evoked N1-P2s with enhanced amplitudes, although the change in pitch was not perceived by the patient. In both experiments AEP scalp topographies were normal.

The results suggest that bitemporal lesions of auditory cortex can dissociate auditory perception and long-latency auditory evoked potentials. A review of evoked potential studies of cortical deafness suggests that the neural circuits responsible for N1-P2 generation lie in close proximity to those necessary for auditory perception.

Résumé

Dissociation des potentiels évoqués auditifs et de la perception après lésion bitemporale

Nous avons étudié les potentiels évoqués auditifs à longue latence (PEA) chez une malade de 82 ans qui a manifesté une surdité subite à la suite d’un deuxième accident cérébrovasculaire. La malade ne pouvait pas comprendre une conversation et manifestait une importante augmentation des seuils de perceptions des sons purs, une incapacité à discriminer les bruits et une capacité réduite à localiser les bruits dans l’espace. La tomographie a révélé des lésions bilatérales du lobe temporal supérieur, incluant le cortex auditif. Les potentiels évoqués auditifs du tronc cérébral étaient normaux.


Ces résultats suggèrent que des lésions bitemporales du cortex auditif peuvent dissocier la perception auditive et les potentiels évoqués auditifs de longue latence. Une revue des études des PE dans les cas de surdité corticale suggère que les circuits nerveux responsables de la genèse de N1 et P2 se situent au voisinage immédiat des régions nécessaires à la perception auditive.

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