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Psychoacoustic and Electrophysiologic Correlates of Central Hearing Disorders in Man

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Summary. Evaluation of central hearing disorders in neuropsychologic patients is handicapped by their insufficient ability to describe auditory deficits and by the lack of easily applicable audiological tests. A novel psychoacoustic discrimination test (PDT) was developed to determine ear asymmetries in the discrimination of changes in intensity, frequency, or temporal structure of regularly presented dichotic stimuli. In 19 of 21 patients with lesions of the auditory cortex or the acoustic radiation according to CT scan evaluation a higher error score was observed for target stimuli presented at the ear contralateral to the side of brain infarction (6 right, 15 left). In the remaining 2 and in 3 other patients with lesions sparing auditory structures no significant ear asymmetries were seen. This may indicate that auditory perception is reduced in patients with only one intact auditory cortex or one intact acoustic radiation, possibly because of a limitation in information processing capacity. Auditory evoked potential results are presented for a normal subject and two patients to illustrate electrophysiologic correlates of central hearing disorders. Using a transformation of scalp into dipole source activity (Scherg and von Cramon 1986), a unilateral loss of middle latency activity was found in case A, who had a lesion of the left acoustic radiation. The extended lesion of the right auditory cortex in case B resulted in a loss of both middle and late latency dipole source potentials of the right temporal lobe. In both cases a corresponding increase in the PDT error score on the contralateral ear was found.

Key words: Auditory cortex – Psychoacoustics – Auditory evoked potentials – Dipole source potentials – Central hearing disorders

Introduction

The ability to speak and to understand is fundamental for human communication. In neuropsychologic patients brain infarctions often involve areas subserving these functions, e.g., the auditory cortex. Little knowledge exists on the auditory impairments resulting from such lesions and on their importance in every day life, because patients have difficulties or are unable to describe their auditory disturbances adequately. Most presently available audiological tests are either designed to monitor peripheral hearing acuity or they are too complex for many patients because of a difficult response mode or high requirements of attention and memory. For this reason and because of high interindividual variability, the binaural and dichotic tests described by Milner (1962), Schulhoff and Goodglass (1969) and Efron and Crandall (1983) were not considered applicable, although using these tests laterality effects in groups of patients with temporal lobe lesions had been shown. Other dichotic tests using verbal material (Katz 1962; Feldmann 1967; Studdert-Kennedy and Shankweiler 1970) cannot distinguish an auditory perceptual from a speech perceptual deficit. In dichotic speech tests laterality effects up to a complete extinction of the information presented at the ear contralateral to an auditory cortical lesion were observed (Sparks et al. 1970). This deficit was named "hemianacousia" by Michel and Peronnet (1982), if concurrently the late auditory evoked potentials (LAEP) were reduced over the lesioned hemisphere.

Auditory evoked potentials (AEP) can help to detect affections of the auditory pathways not only in the lower brainstem, but also in the acoustic radiation and in the auditory cortex, since a method is available to separate the scalp activity into dipole source potentials of both temporal lobes (Scherg and von Cramon 1985a, b, 1986). Also, the origin of the middle latency AEP components in the auditory cortex has been confirmed by the unilateral loss of the corresponding dipole source potentials in the case of lesions to the auditory cortex or to the acoustic radiation (Scherg and von Cramon 1986). It has been questioned whether unilateral alterations of the AEP are an indication of a central hearing disorder and whether AEPs are associated with auditory perception at all (Woods et al. 1984).

This study was to explore the potential value of a novel psychoacoustic discrimination test (PDT) which was devised to monitor central auditory deficits in patients with unilateral temporal lobe lesions. Preliminary results of this test in a patient group are presented. Also, PDT and AEP findings of two typical cases are reported.

Methods and Results

Psychoacoustic Discrimination Test

The PDT was designed to detect ear asymmetries in a dichotic test paradigm (Broadbent 1954) by using an easy response mode, i.e., detection of a change occurring in an on-going regular series of stimuli. The duration of the stimuli was 200 ms or less with an interstimulus interval (ISI) of 600 ms. Subjects were asked to respond to any deviant stimulus by a

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button press, regardless of the side of presentation. The deviant stimuli (targets) replaced the non target stimuli at quasi random intervals (3–6 s) by presenting a target after 5–9 non-targets. In each run 10 targets were presented, 5 to each ear. This basic paradigm allowed us to test the discrimination of a specific stimulus property by separately changing intensity, frequency, or temporal structure of the regular stimulus. Here results are presented only for discrimination of intensity (DI) and temporal structure (DT), tested at different levels of difficulty (1 = easiest, 4 = hardest).

The DI target stimulus consisted of 2 independent 200 ms noise bursts generated by evenly disturbed random number sequences which were routed to 2 digital-to-analog converters (DAC) at a rate of 10 kHz. These signals were simultaneously presented to the right (DAC1) and left (DAC2) ears at 70 dB HL, or higher to adjust for a peripheral hearing loss. DI targets were obtained by amplifying the noise burst on one side by 10 dB (level 1), 5 dB, 4 dB or 3 dB (level 4). The dichotic DT non targets consisted of a train of 6 clicks (0.1 ms, ISI 36 ms) presented to one ear and 5 clicks interlaced on the other ear (18 ms post contralateral clicks). After each presentation the leading side (6 click train) was alternated. In targets the number of clicks in the non leading train was reduced from 5 to 1, 2, 3, or 4 clicks (levels 1–4).

A button press within 200–1800 ms after the start of the target stimulus was considered as correct detection. Performance was assessed by subtracting the number of targets missed on the left from those missed on the right ear and by computing a combined error score (ES) over all DI and DT runs at levels 2 and 3 by:

ES = 1.2* (left − right misses) / SQRT (No of trials/ear)

If ES was greater than +1 (less than −1) this was considered as a right (left) ear advantage. An ES value between −1 and +1 was considered normal. Practically this meant that a difference of 4 out of 20 trials/ear indicated an asymmetry in the PDT. In none of 6 normal subjects (age range 32–61 years, 3 males, 3 females) was a significant ear advantage observed at difficulty level 3 and overall correct performance was better than 80%.

Testing started with intensity discrimination at level 1 (10 dB), which also allowed the more severely handicapped patients to comprehend the test paradigm quickly. The test sequence depended on the performance of the patient, but in most cases at least 2 runs at level 3 (or 2) in both DI and DT categories (= 20 trials per ear at least) could be obtained with a correct performance of at least 50% on the better ear. Only 2 patients had a poorer overall performance and were therefore excluded from this study. Two patients with bilateral temporal lobe lesions and two with brainstem lesions, in whom the side of possible affection of auditory structures could not be ascertained, were also excluded.

Table 1. Relation between side of lesion and PDT ear advantage

No. of cases	PDT: ear advantage			
CT: side of lesioned auditory cortex or acoustic radiation	Right	Left	None	Total
Right	6	0	0	6
Left	0	13	2	15
None	0	0	3	3
Total	6	13	5	24

According to CT scan evaluation the remaining 24 patients (age range 23–66 years, 16 males, 8 females) were classified as most probably having a lesion of the right (6 cases) or left (15 cases) auditory cortex and/or the acoustic radiation or as having a lesion most likely not involving auditory structures (3 cases). Using the ES criterion all patients with a right hemispheric lesion had a right ear advantage and most left auditory lesions (13 of 15 cases) coincided with a better performance of the left ear in the PDT (Tabelle 1). The 2 non conforming cases appeared to have only partial lesions of the auditory cortex according to CT scans. The 3 cases with lesions sparing the auditory structures had normal PDT results (ES < 1). Statistical evaluation by the SPSS program CROSSTABS revealed a highly significant correlation ($\chi^2 = 36.5$, $P < 0.0001$, $df = 4$; uncertainty coeff. (sym.) = 0.80; Pearson's R = 0.74; $P < 0.0001$) between side of lesion and better performance of the ipsilateral ear, a higher incidence of missed targets on the ear contralateral to the lesion respectively.

Auditory Evoked Dipole Source Potentials

Middle (MAEP) and LAEP were recorded from a coronal chain of 11 electrodes spaced at equal distances from the right

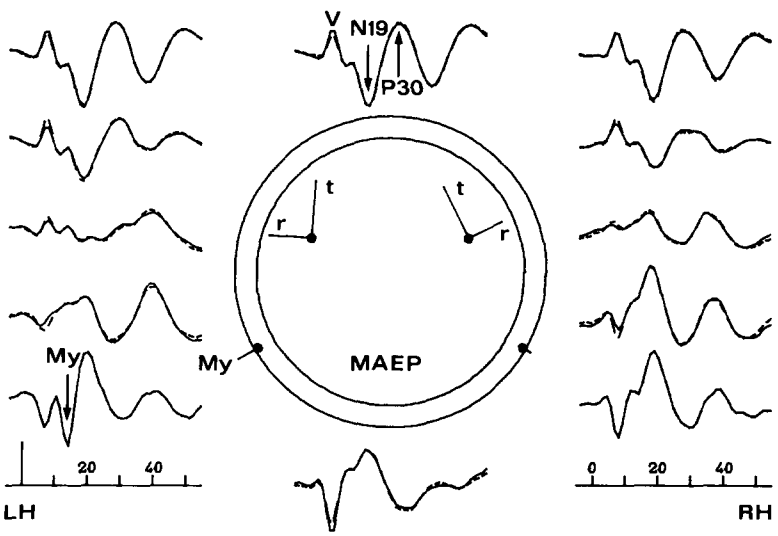

Fig. 1. Coronal scalp distribution of MAEP. Normal subject, left ear clicks at 70 dB HL, average of 10 000 sweeps. Signals were recorded over right *(RH)* and left *(LH)* hemispheres, from vertex *(top)* and neck *(bottom)*. Time scale in ms, vertical mark on left at time of click delivery (0 ms) equals 500 nV. Superimposed dashed lines show the superposition of activity from equivalent model dipoles, illustrated in the head model scheme (frontal section). The potentials due to these sources, i.e., a dipole source in each temporal lobe having a tangential *(t)* and a radial *(r)* component and 2 superficial myogenic *(My)* sources near both mastoids almost identically modelled the 12 measured signals (full lines). The largest deviation of this direct linear solution is found in the interval of brain stem activity (wave V), where a single central dipole fits more accurately

to the left mastoid and from a neck electrode. In separate runs at least 4000 (MAEP) or 192 (LAEP) responses were averaged to 0.1 ms clicks (40 ms, 1000 Hz tone bursts), delivered monaurally at 70 dB HL every 95–135 ms (1.5–2.5 s). Details of the recording and analysis procedure are given elsewhere (Scherg and von Cramon 1986). From the digitally filtered averages (MAEP: 20–300 Hz, LAEP: 3–50 Hz) dipole source potentials were computed by the direct linear approach, assuming 2 bilaterally symmetric temporal lobe generators (Scherg and von Cramon 1986). This method allowed for a transformation of scalp potentials (Fig. 1) in tangential and radial dipole source potentials of both temporal lobes. Hemispheric comparisons could then be based on amplitudes and latencies of the major peaks in these middle and late dipole source potentials (Fig. 2; N19t–P30t, N27r–P39r, N100t, N150r). The most stable parameter for hemispheric comparison was the effective dipole moment, defined as the mean dipole strength averaged over a limited time epoch (19–45 ms for MAEP, 45–200 ms for LAEP).

Typical AEP results of a normal subject (male, 38 years) of a control group of 15 normals (Scherg and von Cramon 1986) are depicted in Figs. 1 and 2. The coronal scalp distribution of both MAEP and LAEP was found to be fairly symmetric over both hemispheres in normal subjects. All wave forms around the coronal plane could be modelled by the superposition of dipole activity originating in both temporal lobes, and, if the MAEP contained myogenic activity, by additional superficial myogenic sources (Fig. 1). In Fig. 1 the model wave forms calculated from the 4 temporal lobe dipole source potentials (and from the 2 myogenic sources) traced the measured signals almost identically (0.5% of unexplained variance).

From Fig. 2 it can be seen that the first activity after the brainstem slow wave V began at 19 ms (N19t–P30t complex) and had an orientation similar to the afferent input into the Heschl's gyri. Radial activity started some 8 ms later (N27r–P39r) and was consistently found in normal subjects and in patients in the intact hemisphere. Similarly, the LAEP exhibited different tangential and radial dipole source potentials with the most prominent peaks being N100t and N150r. Hemispheric differences in effective dipole moment were sufficiently small to allow definition of confidence limits (based on 3 SDs) for both MAEP (a difference of more than 40% of the mean magnitude was significant) and LAEP (35%). This limit of normality was considerably exceeded in the two cases described below.

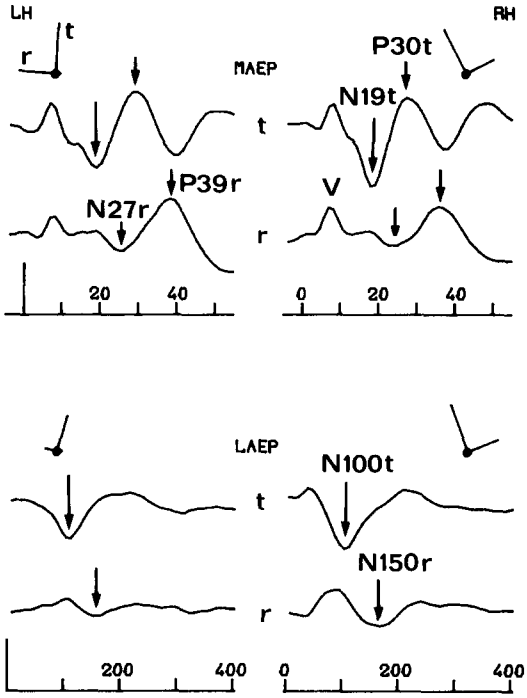

Fig. 2. Temporal lobe dipole source potentials of a normal subject, derived from the coronal MAEP (Fig. 1) and LAEP scalp distribution by linear optimization (Scherg and von Cramon 1986). Tangential *(t)* and radial *(r)* dipole source potentials are depicted for the right *RH)* and left *(LH)* hemispheres. The tangential orientation is defined by the orientation of the primary cortical activity (N19t–P30t) independently for each hemisphere. Using this definition, a secondary, 8 ms delayed, radial activity (N27r–P39r) can be consistently observed. Similarly the LAEP shows different tangential (N100t) and radial peaks (N150r). MAEP scales as in Fig. 1. Time in ms. Length of LAEP stimulus mark corresponds to 5 μV. Note that all activities, except N27r–P39r are slightly larger in the hemisphere contralateral to the side of stimulation (left ear)

Case Reports

Case A was a 52-year-old woman with an ischemic infarction of the left anterior choroidal artery, probably affecting the acoustic radiation within the temporal isthmus (Fig. 3). Testing was 2 years after the event. Pure tone hearing thresholds were normal on both ears (10 dB at all frequencies from 0.25–8 kHz). Her dipole source potentials (Fig. 4) showed a loss of the initial activity of the left auditory cortey (N19t–P30t, N27r–P39r). In contrast, the LAEP dipole activity was undiminished in the left hemisphere, but the left radial component N150r was significantly delayed. This electrophysiologic evidence for a dysfunction of left central auditory structures was confirmed by the PDT results, showing an increased error score for the right ear (ES = −1.68). On difficulty level 3, she detected correctly only 72% of right ear targets but 100% of left ear targets (in a total of 5 DI and DT runs).

Case B was a 36-year-old man having an ischemic infarction in the territory of the right middle cerebral artery (Fig. 5) probably covering most of the right auditory cortex (AI/II, AAI). Testing was 5 months after the brain event. Pure tone audiometric thresholds were normal except for a mild high frequency hearing loss above 4 kHz on the left ear. The dipole source potentials showed an almost complete reduction of right hemispheric middle and late latency components (Fig. 6). The PDT revealed a substantially poorer discrimination of left ear targets. In a total of 6 runs this patient detected 87% of right ear but only 53% of left ear targets correctly (ES = 2.2).

Discussion

These preliminary results of the PDT demonstrate a strong relation between lesions of the auditory cortex or the acoustic radiation and an increased difficulty in auditory discrimination on the contralateral ear. These findings are consistent with Kimura's (1961) hypothesis of a dominance of the contralateral afferent pathways observed in verbal dichotic tests. In the PDT there was no complete extinction of the acoustic in-

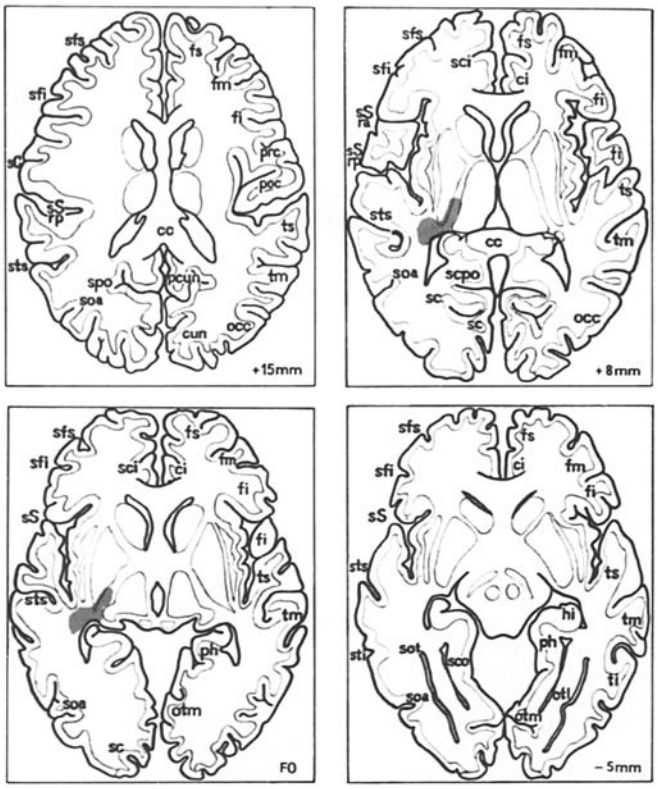

Fig. 3. Redrawings of horizontal CT scan sections at the level of the fronto-orbital *(FO)* line and at +15, +8 and −5 mm from the FO level. This CT scan (case A) showed an infarction of the left anterior choroidal artery, affecting the temporal isthmus but not the auditory cortex

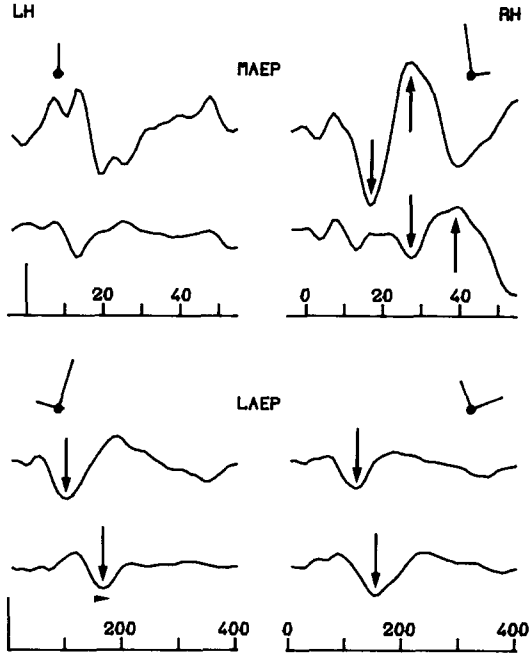

Fig. 4. Temporal lobe dipole source potentials of case A. Scales as in Fig. 2, right ear stimulation. Note the loss of MAEP source activity in the left hemisphere *(LH)* and the normal appearance of tangential (N19t–P30t) and radial (N27r–P39r) activities in the right hemisphere *(arrows)*. In contrast LAEP activity is of normal amplitude in the right and left hemispheres *(arrows)* and only the left radial activity (N150r; *horizontal arrow*) is delayed. This probably indicates intactness of the generating cortical structures while functional changes seem due to altered afferent inputs

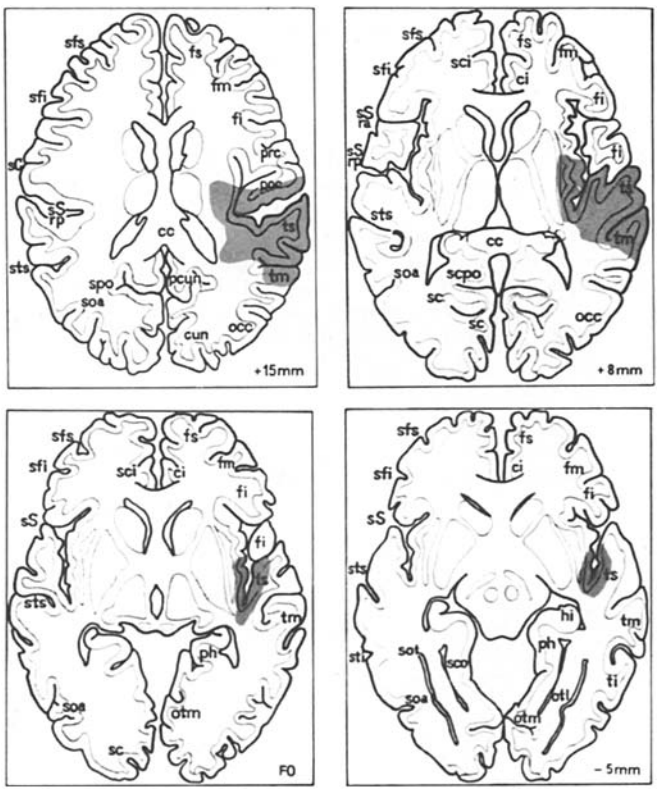

Fig. 5. Redrawings of CT scan sections of case B, showing an infarction in the territory of the right middle cerebral artery. The lesion appears to cover most of the right auditory cortex (AI/II and AAI)

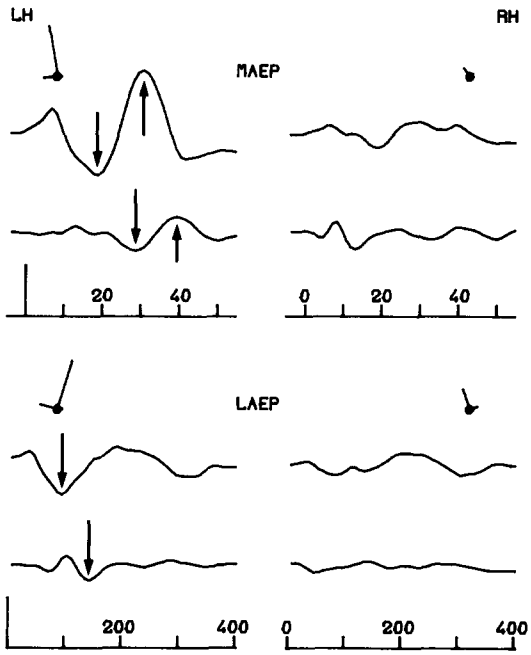

Fig. 6. Temporal lobe dipole source potentials of case B. Scales as in Fig. 2, right ear stimulation. Both MAEP and LAEP source activities appear to be greatly reduced in the right hemisphere. Typical tangential and radial wave forms, present in the left hemisphere *(arrows)* as in normal subjects, cannot be unambiguously identified in the right hemisphere

formation presented at the ear contralateral to the lesion, probably because the ipsilateral pathway connecting this ear to the intact hemisphere still provided sufficient input to allow discrimination of a certain number of targets. The relative decrease in detection probability indicated a restricted capacity for processing the ipsilateral information when simultaneously dominant contralateral input must be processed by the intact hemisphere. A certain level of difficulty in the PDT was required to approach the limits of this information processing capacity (level 3 in most patients, level 2 in some).

Our data are not yet sufficient to establish a relationship between the precise anatomical site of a lesion and the category (intensity or temporal pattern) of the auditory discrimination deficit. Also, there was no apparent hemispheric specialization of either category, as DI and DT asymmetries were observed in cases with lesions of either temporal lobe. In almost all cases having a lesion of auditory structures a deficit in at least one of the auditory discrimination categories could be observed. On the basis of these findings, one may question the relevance of dichotic tests using verbal or consonant-vowel material for detecting deficits on a speech perceptual level. It may be hypothesized that in many patients an elementary auditory perceptual disturbance underlies the observed poorer performance and the ear asymmetries in verbal dichotic tests.

A further advantage of the PDT was that it could be performed by practically all patients, including aphasics. Only in two cases were attentional and/or memory problems too severe to permit collection of a reliable number of correct PDT trials. The clear ear asymmetries found in our patient group indicate that psychoacoustic correlates of central hearing disorders may be consistently found. We are therefore hopeful that discrimination tasks of this type will further elucidate the role of the auditory cortex in auditory perception.

The two cases presented have illustrated that not only psychoacoustic but also electrophysiologic correlates of central auditory deficits can be obtained, despite the existence of opinions to the contrary (Woods et al. 1984). So far, two types of AEP alterations could be distinguished which showed a specific relation between anatomical site of lesion and the AEP changes. First, an "acoustic radiation" type (case A) with a reduction or loss of only the initial temporal lobe activity (MAEP components) and preserved later activity (LAEP) due to selective lesions of the acoustic radiation. Secondly, an "auditory cortex" type (case B) with a unilateral reduction of both MAEP and LAEP. Other cases consistent with this interpretation have been presented (Scherg and von Cramon 1986). Although the derived dipole source potentials allow clear differentiation of tangential and radial activity, which must necessarily originate in temporal lobe cortical layers of different spatial orientations (Scherg and von Cramon 1986), lesions affecting these generators separately appear to be rare.

The derivation of auditory evoked dipole source potentials presents an independent method for clarifying whether a patient has a unilateral affection of the auditory cortex or of the acoustic radiation. A combined application of these electrophysiologic methods with psychoacoustic tests promises to increase our understanding of auditory cortical dysfunction.

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