J Am Acad Audiol 1:146-150 (1990)

Improvement of Central Auditory Function after Partial Temporal Lobectomy in a Patient with Seizure Disorder

Frank E. Musiek*†
Mary Ellen Bromley†
David W. Roberts‡
Lloyd Lamb§

Abstract

This case study reports the auditory findings in one patient with left temporal lobectomy. Behavioral and electrophysiologic central auditory tests were administered just before and 1 week after surgical removal of the left anterior temporal lobe, including the amygdala and portions of the hippocampus. The dichotic digits test and late auditory evoked potentials with P-300 showed marked improvement postoperatively, consistent with improved central auditory function. Postoperative behavioral results on the dichotic digits test are consistent with anatomic evidence that there are few, if any, auditory connections to the anterior temporal lobe. Although electrophysiologic recordings improved postoperatively, they are still considered abnormal. This case study illustrates the use of both behavioral and electrophysiologic procedures for monitoring central auditory function.

Key Words: Temporal lobectomy, amygdala, hippocampus, P-300, dichotic digits, auditory evoked potentials

Temporal lobectomy is considered a viable means of controlling intractable seizure disorder (Penfield and Flanigin, 1950). Since a major portion of the temporal lobe is involved in auditory function, a number of investigators have studied the effect of the seizure disorder or temporal lobectomy on a variety of central auditory test procedures (Collard, 1984; Berlin et al, 1975; Olsen, 1983; Kimura, 1961; Oxbury and Oxbury, 1969; Mazzacchi and Parma, 1978). In these studies, central auditory system test results remained essentially the same, became worse, or in some cases improved slightly after temporal lobectomy. The test procedures commonly used were dichotic listening tasks. Improvement in test scores after temporal lobectomy was usually noted when the right hemisphere was involved; decrements were usually noted when the left side was involved (Olsen, 1983; Kimura, 1961; Oxbury and Oxbury, 1969; Mazzucchi and Parma, 1978). We report on a patient with intractable seizures who demonstrated marked improvement on both behavioral and electrophysiologic tests after left temporal lobectomy.

RELEVANT HISTORY

At the time of testing, this 20-year-old female patient had suffered from a generalized seizure disorder as well as occasional clonic activity of the right arm since the age of three. There was no history of either otologic or audiologic problems; however, it was reported that the patient had difficulty following verbal directions. Early electroencephalography (EEG) data

*Section of Neurology, Department of Medicine
†Section of Audiology and Otolaryngology
‡Section of Neurosurgery, Department of Surgery; Dartmouth-Hitchcock Medical Center, Hanover, New Hampshire
§Section of Communication Disorders, University of New Mexico Hospital, Albuquerque, New Mexico
Reprint requests: Frank E. Musiek, Ph.D., Section of Otolaryngology and Audiology, Dartmouth-Hitchcock Medical Center, 2 Maynard Street, Hanover, NH 03756

indicated focal slowing from the anterior left temporal lobe region, but subsequent EEGs showed some spread of activity to the mid/posterior left temporal lobe, as well as additional involvement of the left frontal lobe and right temporal lobe. Recent EEGs confirmed that the primary site of involvement was the anterior left temporal lobe. The Wechsler Adult Intelligence Score-Revised placed this patient in the low-average range with no differences between verbal and performance scores. The patient had completed high school and training to work as a medical assistant and dental technician. It was difficult for her to pursue these vocations because of her seizures, which often occurred two or three times a day. Medications (phenytoin, carbamazepine, and divalproex sodium) that were maintained during the preoperative and postoperative test period mitigated the severity of the seizures to some extent, but did not control them adequately. Therefore, a left anterior temporal lobectomy was performed for seizure control.

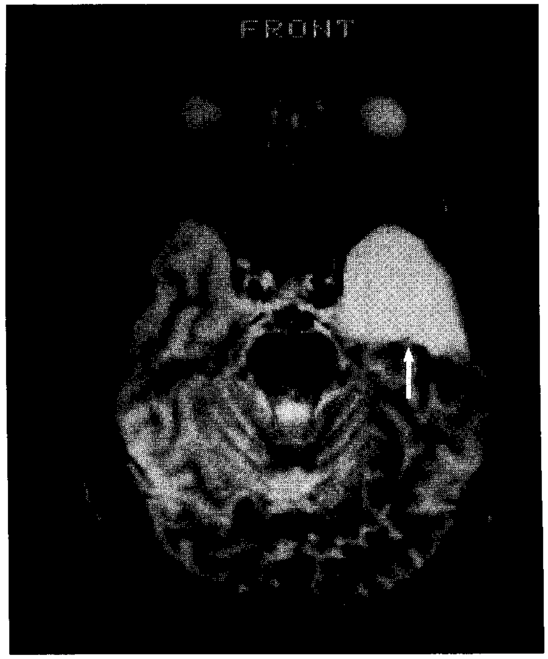

Figure 2 A horizontal view MRI scan of the brain; arrow indicates the inferior-anterior part of the left temporal lobe that has been excised.

SURGICAL PROCEDURE

At surgery a left frontotemporal craniotomy was done and electrocorticography showed epileptiform activity with phase reversal over the anterior temporal lobe. There were no abnormal gross findings in the anatomy of this region. The resection extended posteriorly from the temporal pole 3 cm in the superior temporal gyrus, 4.5 cm in the middle temporal gyrus, and 5 cm in the inferior temporal gyrus (Figs. 1 and 2). The temporal horn of the lateral ventricle was entered, and the amygdala as well as the anterior-most portion of the hippocampus were removed. There were no complications and the patient tolerated the procedure well.

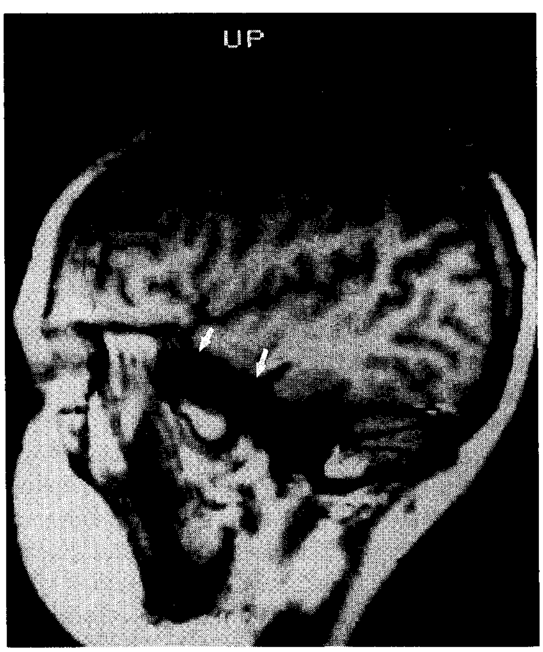

Figure 1 A lateral sagittal view magnetic resonance imaging (MRI) scan of the brain; arrows indicate the sectioned region of the anterior temporal lobe.

AUDIOLOGIC TESTS AND RESULTS

Audiologic tests were performed just before and 1 week after surgical removal of the anterior temporal lobe. All tests were conducted in a sound-treated room (IAC). The patient was alert, well oriented, conversant, and without any seizure activity. She was able to understand verbal instructions for all test sessions. Pre- and postsurgical medications were held constant.

The tests conducted preoperatively and postoperatively included pure tone thresholds, dichotic digits, and late auditory evoked potentials including the P-300. The pure tone data were acquired in the standard clinical manner. The digit data were acquired using prerecorded reel-to-reel tape played on a TEAC X-300 EE

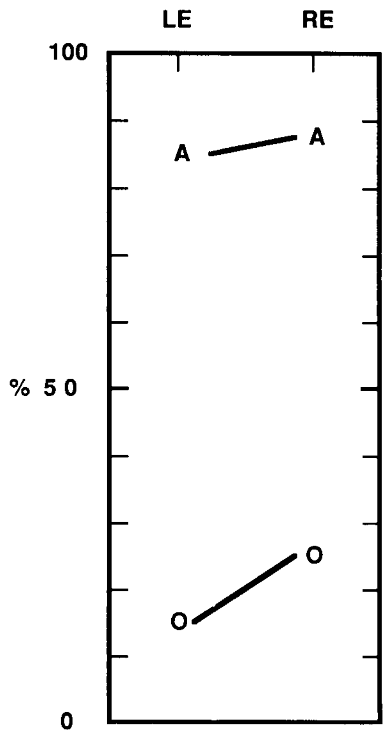

Figure 3 Preoperative (o) and postoperative (A) results on the dichotic digits test.

tape deck at 7½ i.p.s., passed through the speech circuit of a diagnostic audiometer (Grason Stadler 1701), and presented through TDH-49 earphones. Other procedural data for this test are delineated elsewhere (Musiek, 1983). The late potentials and P-300 were acquired using the standard odd-ball paradigm with 1,000 and 2,000 Hz tone bursts (10 ms rise/fall, 20 ms plateau) representing the frequent and rare stimuli. The stimuli were presented at 75 dB SPL, and C-3 and C-4 were selected as recording sites, in order to define laterality effects. Other details of the late potentials and P-300 procedure can be found elsewhere (Kibbe-Michal et al, 1986).

The pure tone thresholds for frequencies from 250 to 4,000 Hz were well within the normal range ($\leq$20 dB HL) bilaterally and showed no change after surgery. The dichotic digits test showed markedly abnormal scores bilaterally before surgery, but improved postoperatively to near-normal levels of performance bilaterally (Fig. 3).

Preoperatively, the late potentials and P-300 did not show any repeatable tracings for the N-100, P-200, N-200, or P-300. However, tracings obtained postoperatively with frequent stimuli in the left ear showed a shallow but repeatable N-100, P-200, and N-200 at both C-3 and C-4 recording sites. Tracings with rare stimuli in this ear showed these same potentials, as well as a large and slightly delayed P-300. The postoperative right ear tracings at C-3 and C-4 were not as large or as readable. However, on the frequent tracings, a definite P-200 was seen and on the rare tracings a noisy and broad peaked P-300 may be present (Figs. 4A and 4B).

DISCUSSION

After surgery, both behavioral (dichotic digits) and electrophysiologic (late potentials and P-300) evidence indicated improved central auditory function. This finding is especially significant since the patient, as of this report, has been without seizures since the surgery (5 months).

This case also demonstrates the use of both behavioral and electrophysiologic tests for monitoring function of the central auditory nervous system. The results of these tests provide information about changes in the central auditory system resulting from surgery or lesions. This information can be of value to the patient, surgeon, and audiologist.

The preoperative bilateral auditory deficits were consistent with bilateral EEG abnormalities in the auditory areas of the temporal lobes. The primary seizure focus was the anterior temporal lobe, which is not thought to be an auditory area, but which may have been responsible for the spread of abnormal electrical discharges to more prominent auditory areas via inter- and intrahemispheric connections. This is consistent with the interpretation that once the primary epileptic focus was removed, epileptic activity in secondary areas stopped.

The postoperative audiologic test results provide further insight to central auditory function and anatomic substrate. The anterior temporal lobe was removed, with a greater portion of mesial than lateral side excised. After this tissue was removed there was essentially normal performance on the dichotic digits test, which is in keeping with the anatomic data from Jones and Powell (1970). These investigators suggest that there are few if any auditory connections to the temporal pole; therefore, excision of this area should have little or no effect on central auditory function tests. In addition, the excision stopped the spread of electrical discharges to the main auditory areas, resulting in overall improved function.

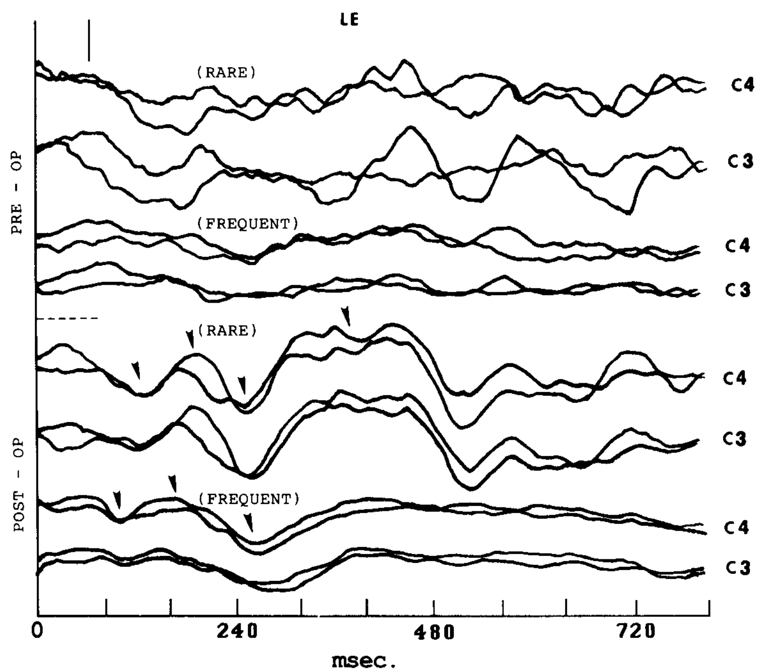

Figure 4 *A*, Preoperative and postoperative N-100, P-200, N-200, and P-300 evoked potentials for the left ear. The arrows in Figures 4*A* and 4*B* indicate responses. From left to right, the first arrow = N-100, second arrow = P-200, third arrow = N-200, and fourth arrow = P-300. (The calibration marker at the upper left = 5 microvolts.)

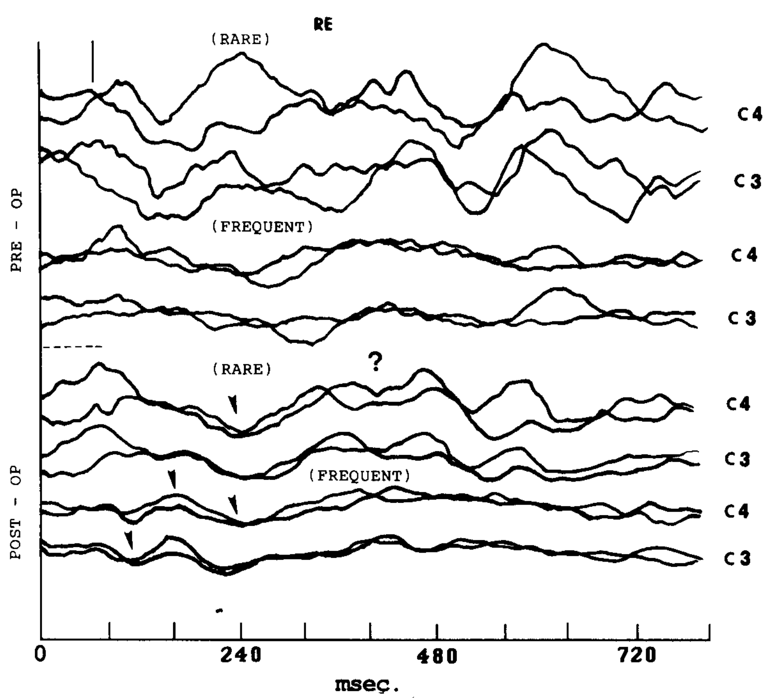

Figure 4 *B*, Preoperative and postoperative evoked potentials for the right ear.

The postoperative late potentials and P-300 results are more difficult to explain. Although these responses showed marked improvement, the C-3 and C-4 tracings from the right ear must still be considered abnormal for a 20-year-old person. The P-300 may have been affected by excision of the left amygdala and part of the hippocampus. These structures have been considered relevant to the generation of the P-300, based on depth-electrode recordings (Halgren et al, 1986). Other investigators, however, have demonstrated that unilateral removal of the amygdala and hippocampus had no marked effect on scalp-recorded P-300 in humans (Johnson and Fedio, 1987). Recordings obtained from monkeys with bilateral lesions of the amygdala and hippocampus (Paller et al, 1988) provide additional evidence that these structures may contribute little to the generation of the P-300. Once the epileptic activity was controlled in our patient, a P-300 was observed, despite removal of the amygdala and portions of the hippocampus. Hence, our findings would argue against these structures being primary generators of the P-300. Since amplitude and latency differences in electrode sites were not noted, the abnormal findings could represent a contralateral ear effect in reference to the excised area. The poor morphology of the late potentials may be related to a reduced volume conducted potential (Knight et al, 1980) along the right ear to left hemisphere tract, which could also affect the P-300. It is also possible that the poor electrophysiologic response may be related to residual neural damage caused by the seizure disorder. Since damage may have selectively affected the late auditory and cognitive potentials, and not the dichotic listening task.

Both behavioral and electrophysiologic central auditory test procedures have shown improvement in central auditory function after anterior temporal lobectomy in a patient with seizure disorder. The results indicate a marked change in central auditory function that paralleled the patient's overall improvement after surgery illustrating the use of central tests as a monitoring tool.

REFERENCES

Berlin CL, Cullen JK Jr, Hughes LF, Berlin JL, Lowe-Bell SS, Thompson CL. (1975). Dichotic processing of speech: acoustical and phonetic variables. *Proceedings of a Symposium on Central Auditory Processing Disorders.* Omaha, NE; University of Nebraska Medical Center, 36–46.

Collard ME. (1984). Central auditory tests in patients with intractable seizures. *Semin Hear 5*(3):277–295.

Halgren E, Stapleton JM, Smith M, Altafullah I. (1986). Generators of the human scalp P3(s). In: Cracco R, Bodis-Wollner I, eds. *Evoked potentials* New York: Alan R. Liss, 269–284.

Johnson R, Fedio P. (1987). Task-related changes in P-300 scalp distribution in temporal lobectomy patients. In: Johnson R, Rohrbaugh J, Parasuramen R, eds. *Current trends in event-related potential research.* Electroencephalogr Clin Neurophysiol Suppl 40:669–704.

Jones EG, Powell TP. (1970). Anatomical study of converging sensory pathways within the cerebral cortex of the monkey. *Brain* 93:793–820.

Kibbe-Michal K, Verkest SB, Gollegly KM, Musiek FE. (1986). Late auditory potentials and the P300. *Hear Instr* 37(11):22–24.

Kimura D. (1961). Some effects of temporal lobe damage on auditory perception. *Can J Psychol* 15:157–165.

Knight R, Hillyard S, Woods D, Neville H. (1980). Effects of frontal and temporal-parietal lesions on auditory evoked potential in man. *J Electroenceph Clin Neurophysiol* 50:112–124.

Mazzucchi A, Parma M. (1978). Responses to dichotic listening tasks in temporal epileptics with or without clinically evident lesions. *Cortex* 14:380–390.

Musiek FE. (1983). Assessment of central auditory dysfunction: the dichotic digits test revisited. *Ear Hear* 4:79–83.

Olsen WO. (1983). Dichotic test results for normal subjects and for temporal lobectomy patients. *Ear Hear* 4(6):324–330.

Oxbury J, Oxbury S. (1969). Effect of temporal lobectomy on the report of dichotically presented digits. *Cortex* 5:3–14.

Paller K, Zola-Morgan S, Squire L, Hillyard S. (1988). P-3 like brain waves in normal monkeys and in monkeys with medial temporal lesions. *Behav Neurosci* 102(5):714–725.

Penfield W, Flanigin H. (1950). Surgical therapy of temporal lobe seizures. *Am Med Assoc Arch Neurol Psych* 64:491–500.