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Central Auditory Deficits Associated with Compromise of the Primary Auditory Cortex

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Abstract
The subject of this study was a 46-year-old female who had suffered a cerebrovascular accident (CVA). Magnetic resonance imaging revealed damage in the area of the distribution of the middle cerebral artery involving most, if not all, of the primary auditory area of the left hemisphere. No auditory problems were noted prior to the CVA; however, following the CVA, the subject reported a number of auditory difficulties. Pure-tone thresholds were normal post-CVA, and performance on speech recognition testing was good in both ears if ample time was provided between a response and the presentation of the next test item. Duration pattern, intensity discrimination, and middle latency response test results were abnormal for both ears, and right ear deficits were evident on an auditory fusion test and two dichotic speech tests (digits and rhymes). This case is significant in that it demonstrates a good correlation between damage to known key auditory regions and central auditory test results.

Key Words: Auditory cortex, auditory processing disorder, cerebrovascular accident, middle latency response

Abbreviations: ABR = auditory brainstem response; CVA = cerebrovascular accident; MLR = middle latency response; MRI = magnetic resonance imaging; SISI = short increment sensitivity index

Sumario
El sujeto de este estudio fue una mujer de 46 años de edad que había sufrido un accidente vascular cerebral (CVA). La resonancia magnética reveló daño en el área correspondiente a la arteria cerebral media, involucrando la mayor parte, o casi toda el área auditiva primaria en el hemisferio izquierdo. No se habían notado problemas auditivos previos al CVA; sin embargo, luego del CVA, un número de dificultades auditivas fueron reportadas. Los umbrales para tonos puros eran normales posteriormente al CVA, y el desempeño en las pruebas de reconocimiento del lenguaje era bueno en ambos oídos, si se otorgaba tiempo suficiente entre una respuesta y la presentación de la siguiente palabra. El patrón de duración, la discriminación de la intensidad y la prueba de respuestas de latencia media resultaron anormales en ambos oídos, en tanto se hacían evidentes las deficiencias en el oído derecho durante la prueba de fusión auditiva y en dos pruebas dicóticas de lenguaje (dígitos y

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rimas). Este caso es significativo para demostrar una buena correlación entre el daño a regiones auditivas clave conocidas y los resultados de pruebas auditivas centrales.

Palabras Clave: Corteza auditiva, trastorno de procesamiento auditivo, accidente cerebro-vascular, respuestas de latencia media

Abreviaturas: ABR = respuestas auditivas del tallo cerebral; CVA = accidente cerebro-vascular; MLR = respuestas de latencia media; MRI = imágenes por resonancia magnética; SISI = Indice de Sensibilidad a Pequeños Incrementos

Kimura (1961) was among the first researchers to demonstrate that patients with lesions involving either the right or left temporal lobe tended to show impaired performance for dichotic speech stimuli presented in the ear contralateral to the involved hemisphere. Since the time of her landmark investigation, there have been a number of other investigators who have examined the performance of patients with central auditory nervous system compromise involving either one or both temporal lobes on dichotic speech tests (e.g., Lynn and Gilroy, 1972; Speaks et al, 1975; Olsen, 1983; Musiek et al, 1994; Musiek and Lee, 1998). Although contralateral ear deficits were often noted in patients with compromise of either temporal lobe in these studies, this finding was by no means universal. Moreover, the extent of the reported deficits for those patients demonstrating contralateral performance decrements varied considerably both within and across these investigations.

In his investigation of 67 patients with surgical removal of the anterior portions of the temporal lobe, Olsen (1983) found that excision of the anterior portion of either temporal lobe did not necessarily result in dichotic speech deficits. In fact, more than 40% of the patients in his study showed normal performance for both ears following excision of either the right (18 of 33) or left (10 of 34) anterior temporal lobe. Of the remaining 39 subjects in this investigation, 21 showed contralateral ear deficits, 9 had ipsilateral ear deficits, and 9 demonstrated bilateral deficits. A review of information detailing the surgical procedures used for these patients suggests that the temporal lobe excisions were likely to have compromised areas of the temporal lobe known to be distal to the primary auditory area (see Jones and Powell, 1970). Given this site of surgical compromise, the lack of abnormal auditory findings in many of these subjects is not surprising. What is more surprising, especially when one considers the reported site of temporal lobe compromise in this pool of subjects, is the finding of auditory performance deficits in a significant number of these subjects. An explanation for this unexpected finding can be offered if one considers the characteristics of the subject pool. All of the subjects had preexisting central auditory nervous system compromise that rendered them candidates for surgical intervention. Olsen noted that in many of the subjects with postsurgical deficits, abnormal performance was also evident prior to surgical excision. Therefore, the postsurgical deficits in these subjects could not be attributed definitively to the compromise of the temporal lobe that resulted from the surgical excisions. It is quite possible that the performance decrements noted in these subjects were related to central nervous system damage that may have compromised more of the temporal lobe than simply the surgically excised areas.

In other studies of patients with hemispheric lesions that presumably affected more posterior regions of the temporal lobe, contralateral deficits were more commonly noted (Lynn and Gilroy, 1972; Speaks et al, 1975; Musiek et al, 1994; Musiek and Lee, 1998). However, the presence of contralateral ear deficits was not observed for all patients with "apparent" posterior temporal lobe damage. These disparate results have led many clinicians and researchers to question the value of contralateral ear deficits as a reliable indicator of auditory dysfunction

associated with temporal lobe compromise.

Of relevance to the present discussion are the results of two investigations that were conducted on patients undergoing commissurotomies at the Dartmouth-Hitchcock Medical Center in New Hampshire. Baran et al (1986) found that patients undergoing anterior commissurotomies for control of intractable seizures showed no obvious postsurgical performance decrements on a number of central auditory tests, including dichotic speech tests and a pattern perception test, whereas Musiek et al (1984) found severe left ear deficits on dichotic speech tests and a binaural deficit on a temporal patterning test when the surgical sectioning compromised the posterior portion of the corpus callosum. Since the interhemispheric fibers that connect homologous portions of the anterior temporal lobe course through the anterior portion of the corpus callosum and those that connect the posterior portion of each temporal lobe travel through the posterior portion of the corpus callosum, these results lend support to Jones and Powell's (1970) findings and suggest that the anterior portion of the temporal lobe may not play a critical role in the processing of auditory information, while the posterior portion of this structure is intimately involved in the processing of auditory information.

In many of the studies cited earlier, the precise locations of the lesions were not well documented, and this may have served as the basis for at least some of the conflicting results. It is likely that the central auditory nervous system compromise in many of the cases without significant auditory findings may have involved areas of the temporal lobe not critical for auditory processing (i.e., the anterior portion) as the temporal lobe contains many nonauditory areas. In other cases (i.e., those with posterior temporal lobe compromise), the lesions may have spared some of the essential auditory fibers, permitting normal or only mildly reduced performance in the contralateral ears of these subjects.

Although the information provided thus far clearly implicates the posterior portion of the temporal lobe as being essential for the processing of auditory information, it is by no means the only central structure that is involved. Other investigators have documented the importance of structures outside, but in close proximity to, the posterior temporal lobe (e.g., parietal lobe, insula), which if compromised may result in significant contralateral ear deficits on dichotic speech tests (Hyman and Tranel, 1989; Fifer, 1993; Musiek et al, 1994; Habib et al, 1995) and bilateral deficits on patterning tests requiring verbal report (Musiek et al, 1994).

A careful consideration of the available evidence leads one to the following predictions: (1) that lesions confined to the anterior portion of the auditory cortex are not likely to result in significant performance decrements on central auditory tests, including dichotic speech and temporal patterning tests; (2) that the closer a lesion site is to the primary auditory cortex (i.e., posterior temporal lobe), the larger the observed auditory effects will be; and (3) that significant auditory areas are located in other areas of the brain (i.e., parietal lobe, insula) that lie in close proximity to the primary auditory cortex (i.e., the posterior temporal lobe), which, if compromised, may result in performance deficits similar to those observed in cases with lesions affecting the posterior portion of the temporal lobe.

The case to be presented in this article is unique for a number of reasons: (1) this patient has sustained a lesion that has compromised virtually the entire primary auditory cortex of the left hemisphere; (2) there does not appear to be any evidence of preexisting central nervous system compromise in this patient, which permits the unqualified attribution of post-lesion auditory deficits to the area(s) compromised by the lesion; (3) the precise locus and extent of the damage has been defined carefully through imaging techniques; (4) the audiologic findings are quite dramatic in that they demonstrate essentially complete extinction of contralateral ear performance on dichotic speech tests and a severe bilateral performance decrement on an auditory pattern test; and (5) the case demonstrates a good correlation between the site-of-lesion and the central auditory test results.

CASE REPORT

History

This patient was a 46-year-old female when she was seen at the Dartmouth-Hitchcock Medical Center for a diagnostic evaluation. At the time of her evaluation,

she presented with a history of a cerebrovascular accident (CVA) that was sustained six months prior to her visit. A review of her radiology and medical records revealed that the CVA affected the primary auditory area of the left hemisphere and resulted in mild receptive and expressive aphasia with little, if any, motor involvement. The patient had been evaluated post-CVA by a speech-language pathologist and was receiving speech and language services until her insurance coverage for these services was spent. At the time of her audiologic evaluation, she was no longer receiving services.

Prior to her CVA, the patient was an executive sales manager with considerable responsibility and job-related stress. She reported that she had been seeing a psychiatrist at the time of her CVA and that she had been on a number of medications at that time. She indicated that she believed that these medications may have caused, or in some way contributed to, her stroke. Following her CVA, the patient was no longer able to function effectively as an executive sales manager, and she had to change careers. At the time of her visit to our clinic, she was learning to work as a chef/cook.

The patient denied the existence of any auditory difficulties prior to the time of her stroke. However, following her CVA, she reported a number of auditory complaints, including (1) extreme difficulty hearing in the presence of background noise, (2) difficulty following conversations whenever more than one speaker was talking, and (3) difficulty comprehending the speech of "fast speakers." She also indicated that she no longer enjoyed listening to music, as it now sounded distorted, discordant, and often "scrambled."

In addition to these difficulties, the patient reported difficulties reading, writing, and auditorily recognizing complex words. She indicated that she now tends to express herself in simple sentences, as she can no longer formulate lengthy sentences. She also mentioned that she can read aloud but that she has difficulty reading silently. Her vision appears to be normal, and at the time of our evaluation she was still on a number of medications. She is right-handed.

Audiologic Exam

All audiologic testing on this patient was carried out in a sound-treated booth. Pure-tone and spondee thresholds were established in a conventional clinical manner, as was routine speech recognition testing using the Northwestern University Auditory Test No. 6. However, due to the patient's report of difficulty with the conventional procedure, additional speech recognition testing was conducted by extending the time interval between the patient's response and the presentation of the next test item (e.g., from an interval of approximately one second to one that was approximately five seconds in duration). Different word lists were used for each ear and each presentation level, and 25 words were presented for each individual test.

A number of additional tests were administered to assess the integrity and functioning of the patient's central auditory nervous system. These central auditory tests included dichotic digits, duration patterns, dichotic rhymes, auditory fusion, intensity discrimination, and the (auditory) middle latency evoked response (MLR). The dichotic digits and pattern perception tests used in this study have been described numerous times elsewhere and will not be detailed here (for information on these tests see Musiek and Pinheiro, 1985; Musiek, 1994; Chermak and Musiek, 1997).

The dichotic rhyme task used in this study involved the presentation of 30 pairs of monosyllabic words (consonant-vowel-consonant) to the two ears in a dichotic manner. The words in each pair were identical with the exception of the initial consonant (e.g., "ten" and "pen"), and the patient was asked to repeat all of the words that were heard. The acoustic and temporal characteristics of the words in each pair are such that for normal listeners the spectral information contained in the two individual test stimuli tends to fuse, resulting in perception of only a single test item. Therefore, when asked to repeat all of the words heard, patients with normal auditory function will tend to report only the word presented to the left or right ear, but not both. As a result, normal scores for each ear tend to hover around 50% with a slight right ear advantage (see Musiek et al, 1989). Abnormal performance is indicated if the score in either ear is too high or too low.

The intensity discrimination test that was used in this investigation is a modification of the short increment sensitivity index (SISI). After orientation and practice

with the test procedure, ten 5 dB intensity increments were superimposed on a 1000 Hz (carrier) tone presented at 55 dB HL. Following the presentation of the 5 dB increments, ten 2 dB intensity increments were presented. Each ear was tested independently, and the patient was required to press a response button every time an intensity increment was heard. The interstimulus interval between increments was varied in a random fashion, but in all cases it exceeded a minimum of five seconds, and percent identification rates were recorded for each ear at both increment levels.

The Auditory Fusion Test–Revised was administered to each ear with a slight modification of the standard test protocol. Two 500 Hz tone pulses were used, and the interval between these tones was varied from 0 to 300 msec (see McCroskey and Keith, 1996). The patient was asked to indicate if one or two tones were perceived and fusion thresholds for both ears were determined in the manner suggested by McCroskey and Keith (1996).

The MLR was carried out using a 100 μsec square wave passed through ER-3A sound insert phones. The click stimulus was presented to right and left ears independently at a rate of 9.8 clicks per second at 70 dB nHL. Neuroelectrical activity was detected by standard scalp electrodes attached at C3, Cz, and C4 electrode sites and referred to the ipsilateral ear to which the stimulus was presented. Electrode impedance was maintained at less than 5 kohms for each electrode and was balanced across the electrode array. Filter settings of 20 to 1500 Hz were employed, and 1000 (accepted) trials were run. The MLR waveforms derived for both ears were displayed on a 72 msec time window and were replicated.

Audiologic Findings

Pure-tone thresholds fell within normal limits for both ears (Figure 1), as did the spondee threshold derived for each ear (i.e., 15 dB HL in both the left and right ears). Word-recognition scores of 32% in the right ear and 42% in the left ear were noted when testing was done in a conventional manner. However, as the patient indicated that the stimuli were being presented in "too rapid" a fashion, additional testing was done with the examiner extending the time interval between a patient response and the presentation of the next stimulus item (see discussion above). When tested under these conditions, the patient's performance rose to 80% correct identification in the right ear and 82% in the left ear (Figure 2).

Results for three of the central auditory tests (dichotic digits, duration patterns, dichotic rhymes) are displayed in Figure 3. These results show normal performance for the left ear on the dichotic digits test and better than expected results for the left ear

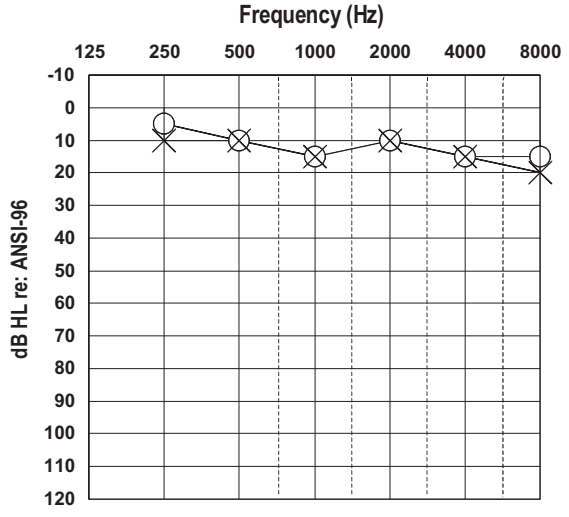

Figure 1. Audiogram for a 46-year-old female who sustained a CVA affecting the primary auditory cortex in the left hemisphere.

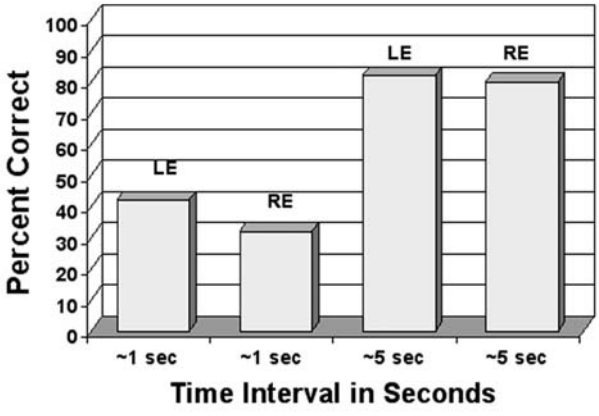

Figure 2. Speech recognition scores for a 46-year-old female with a left-sided CVA under two response conditions. The conditions varied in terms of the time interval that lapsed between the patient's response and the presentation of the next stimulus (i.e., ~1 sec versus ~5 sec).

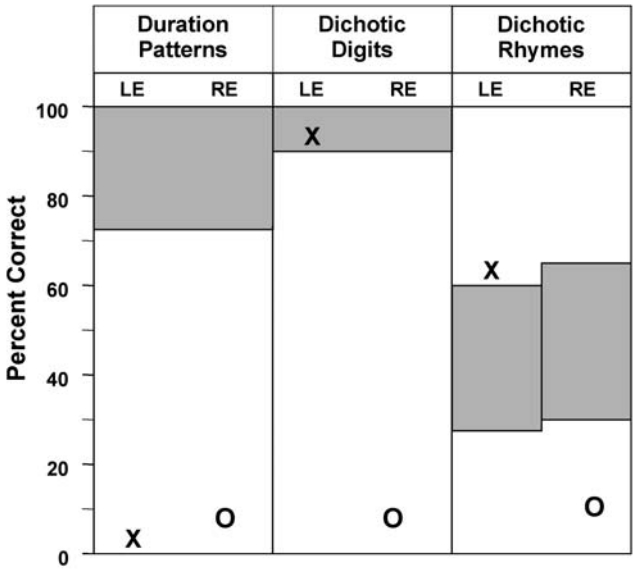

Figure 3. Central auditory test results for a 46-year-old female with a left-sided CVA. The shaded regions on the figure represent the range of normal performance for an adult patient.

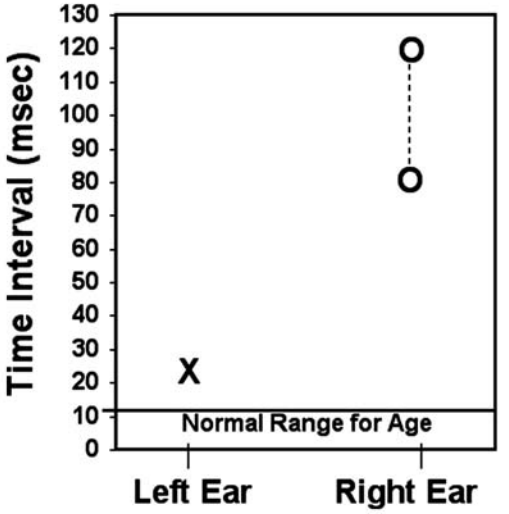

Figure 4. Auditory fusion test results for a 46-year-old female with a left-sided CVA. The line cutting across the figure represents the cutoff for normal performance for adults in the fourth decade of life.

on the dichotic rhyme test (recall that normal performance averages around 50% with an upper cutoff of 60% for the left ear). On both of these tests, right ear performance was severely compromised (8% and 10% for the digits and rhyme tests, respectively), as was the performance of both ears on the duration patterns test (7% for the right ear and 3% for the left ear when a verbal report response procedure was utilized).

Auditory fusion test results revealed abnormal performance for both ears (Figure 4). The patient was unable to detect the presence of two tones in the left ear until the tones were separated by an interstimulus interval (i.e., gap) of 25 msec. In the right ear, performance was more variable (ranging from gap requirements of 80 to 120 msec upon repeated testing) with all results falling clearly outside the range of normal performance. Moreover, it should be noted that although performance for both ears fell outside the range of normal, a comparison of the performance of the two ears revealed a significant asymmetry in performance; that is, a mild ipsilateral deficit was noted in the left ear, and a severe contralateral ear deficit was found for the right ear.

Results of the modified SISI procedure are displayed in Figure 5 with results showing

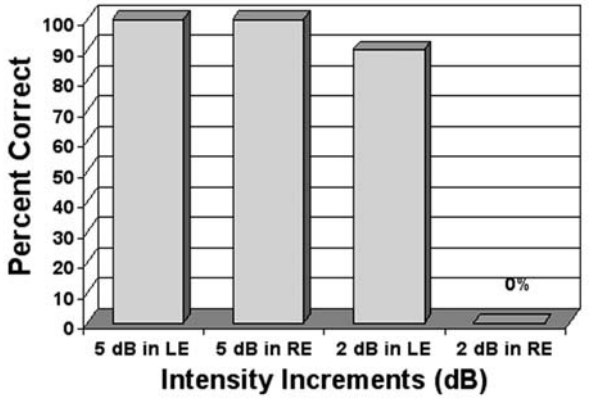

Figure 5. Intensity discrimination test results for a 46-year-old female with a left-sided CVA at two intensity increment levels (5 and 2 dB) superimposed on a 55 dB HL carrier tone.

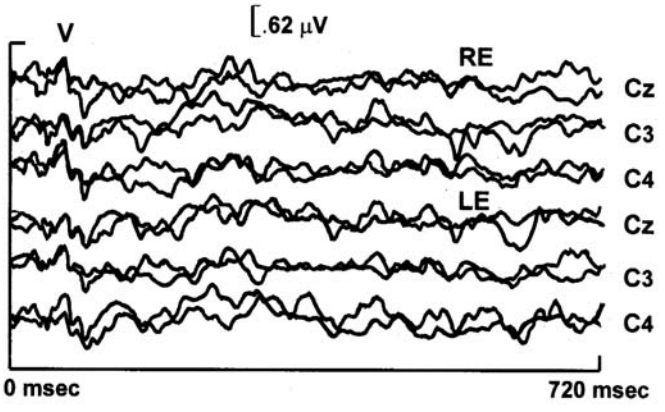

Figure 6. MLR results for a 46-year-old female with a left-sided CVA. The waveforms displayed were derived at three electrode sites (C3, Cz, and C4) for the right and left ears.

high identification rates for the 5 dB intensity increments for both ears (i.e., 100%). For the 2 dB intensity increments, left ear performance remained quite good (i.e., 90%), and right ear performance dropped to 0%.

Finally, the MLR results are shown in Figure 6. Inspection of these tracings reveals the presence of wave V of the auditory brainstem response (ABR) at normal latency measures (~5.6 msec) at each electrode site for each ear, whereas the MLR results are absent for all waveforms derived.

Radiologic Findings

Radiologic findings in this case document compromise of the left temporal lobe in the area of the primary auditory cortex extending into the posterior parietal lobe (anterior-posteriorally and laterally the lesion affects Heschl's gyrus, the planum temporale, the supramarginal gyrus, and the posterior parietal lobe of the left hemisphere, and medially the lesion involves the first temporal sulcus, the insula, and Heschl's gyrus). Two magnetic resonance images (MRIs) are provided to document the lesion site. The first shows an enhanced signal area that includes the primary auditory cortex (Heschl's gyrus and the planum temporale) and extends into the inferior posterior parietal lobe (Figure 7). The second image is a saggital view between the most lateral and most medial aspect of the temporal lobe (Figure 8) revealing a large left-sided defect involving most of Heschl's gyrus and the tissue immediately anterior to this structure. The area of compromise is additionally shown on a coronal template of the brain (Figure 9). This area of infarct is likely associated with disruptions to the vascular supply from the middle and posterior temporal branches of the middle cerebral artery.

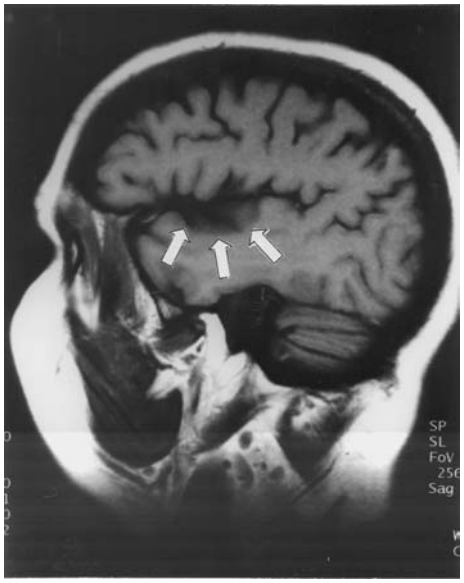

Figure 8. A lateral T1 magnetic resonance image showing involvement along the Sylvian fissure in the left cortex. This view shows the compromise of key auditory regions of the superior temporal plane.

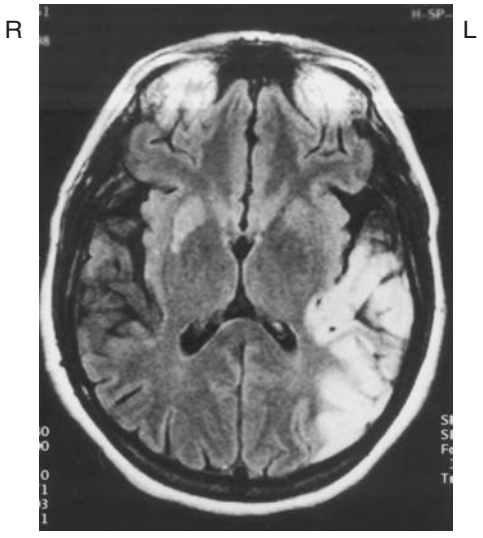

Figure 7. A transverse T2 magnetic resonance image showing an enhanced signal in the area of the left primary auditory cortex (Heschl's gyrus and planum temporale) with the lesion extending into the posterior parietal lobe. The area of infarct represents distribution of the middle cerebral artery.

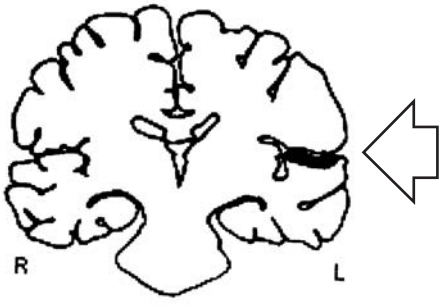

Figure 9. A coronal template of the brain showing the authors' interpretation of the site of lesion in a 46-year-old female with a left-sided CVA. The shaded area depicts damage in the region of the left superior temporal gyrus.

COMMENTS

The patient's symptomatology in this case was well correlated to the central auditory test findings. Damage was confined essentially to the primary auditory area of the left hemisphere, which had devastating effects on appropriately selected central auditory tests. In many cases in which the central auditory nervous system damage involved only portions of the primary auditory area, or portions of the temporal lobe distal to the primary auditory region, the effects on central auditory test results have been nonexistent or marginal (Olsen, 1983; Mueller, 1987; Mueller et al, 1987). In this case, the lesion site involved most of the primary auditory area of the left hemisphere, and the central auditory test results were severely compromised. This condition, where damage is isolated to the primary auditory region, is a relatively rare occurrence in nature. It does, however, provide us with a good insight as to how certain central auditory tests can be affected when the auditory areas truly are damaged.

The dichotic test procedures that were used in the present investigation showed marked performance deficits in the ear contralateral (i.e., the right ear) to the lesion site (i.e., left hemisphere). This is the classic and anticipated finding for these kinds of central auditory tests (Kimura, 1961; Speaks et al, 1975; Musiek and Baran, 1987). The left ear (i.e., ipsilateral ear) scores on the dichotic listening procedures did not show any compromise indicating that the callosal fibers were essentially unaffected by the lesion in this case. It is interesting to note that the left ear performance of this patient on the dichotic rhyme test was higher (i.e., better) than would have been predicted based upon the normative data. This "better than expected" performance is most likely due to the fact that the competition for the speech processing capacities of the left hemisphere, which would have arisen from an intact right ear to the left hemisphere pathway, was eliminated (or significantly minimized) by the extensive damage to the primary auditory area of the left hemisphere. Under such circumstances, the processing of the critical neural input in the language processing areas of the left hemisphere would have been largely confined to information arriving by way of the contralateral pathway from the left ear to the right hemisphere (i.e., primary auditory area) and connecting via the interhemispheric connections to the left hemisphere (i.e., language-processing areas outside of the primary reception areas, which one would presume were not affected or only minimally affected by the lesion in this case).

The duration pattern test results showed performance scores below the level of chance performance for both ears. The bilateral deficit noted on this test is to be expected for a patient with a unilateral hemispheric lesion since both hemispheres and the hemispheric connections are needed to decode and verbally report these patterns (Musiek et al, 1990).

The results of the intensity discrimination measure employed in this investigation were also consistent with the lesion site and the other behavioral test results. The patient was unable to discriminate a 2 dB intensity change for the right ear but performed "close to normally" for the left ear on this task. These results are consistent with the findings of Thompson and Abel (1992), which showed poor auditory discrimination in patients with lesions at, or near, the primary auditory cortex. It is interesting to note that Thompson and Abel (1992) found greater effects for left as opposed to right hemisphere lesions. Although we did not have a case of a right hemisphere lesion for direct comparison in the present investigation, the severity of the deficits noted in our patient would appear to be consistent with these observations. In the absence of a standardized and commercially available test of intensity discrimination, a modified SISI procedure was used in an effort to assess intensity discrimination in the present patient. Given the findings of Thompson and Abel (1992) and the abnormal test results noted for the patient in the present investigation, it would appear that assessment of intensity discrimination in patients at risk for central auditory nervous system dysfunction should be given serious consideration. At the present time, a clinically feasible and well-standardized test of intensity discrimination does not exist. If such a test were to be developed, it is anticipated that its use within an auditory test battery could serve to document more fully the nature and the extent of the auditory deficits in patients with central auditory lesions.

The Fusion Test–Revised results showed a marked right ear deficit with great variability. The left ear performance also

showed a deficit relative to published norms (McCroskey and Keith, 1996), but not nearly the involvement reflected by the right ear score. This finding is consistent with the click fusion test findings for patients with cortical lesions that have been published previously (Lackner and Teuber, 1973). This test result, as well as that noted for the auditory duration patterns test, indicates a deficit in the temporal processing of auditory information, a common finding in individuals with compromise of the primary auditory areas of the brain (Musiek et al, 1990, 1994).

The findings on all of the tests mentioned above were consistent with what is expected and known for central auditory test results when key auditory regions are compromised. Two other test findings, that of routine speech recognition and the middle latency evoked response, also showed marked deficits, but some specific findings are difficult to explain. As was discussed above, this patient performed markedly better on speech recognition tests when she was given extended time after her response to one item and before the presentation of the next test item. It is difficult to say whether this effect is auditory or nonauditory in nature. When the speech recognition test items were administered at a typical pace, the patient performed poorly and complained that she felt rushed during this test procedure. The standard pace of administering this test may have been too rapid for full deployment of critical auditory and cognitive processes, such as the timely allocation of attention and important trace memory and language functions. This in turn may have caused stress on the part of the patient, which in turn may have compromised her overall performance. When extended time was given between her responses and the following test items, the patient may have felt more relaxed and more confident in her responses. This in turn may have served to elevate her scores.

It has been demonstrated previously that the auditory comprehension abilities of aphasic patients can be enhanced when extra time is provided on listening tasks (Liles and Brookshire, 1975). It is interesting to note that when presented with shorter test items on the dichotic speech tests (words or digits), the patient in the present investigation did not appear to be rushed, even though there were marked deficits in one ear. Could it be that the presentation of a carrier phase along with the stimulus items in the speech recognition test played a role? Perhaps the processing of an entire sentence caused greater stress due to greater memory and attention allocation demands.

One may further speculate that when the test items were presented at a regular pace that there may have been some delayed cognitive, memory, or auditory processes that lingered in time, which in turn affected the attention devoted to, and comprehension of, subsequent test items. This is highly speculative but may offer a plausible explanation, as this type of phenomenon (i.e., trace memory) is known to exist for some time period after a task is completed in normal brains. It may stand to reason that this phenomenon may be even further extended in pathological brains.

The MLR demonstrated essentially no responses across electrode sites for either the left or right ears. In unilateral brain lesions, a greater deficit is typically noted from an electrode positioned over or near the lesion site (Musiek et al, 1994). This laterality effect was not seen in this case. Rather, there was no response on either side or at the midline for both the left and right ears. This finding has been seen before in unilateral hemispheric lesions, but it is relatively rare (Kileny et al, 1987). In this case, the lesion may have affected neural tissue deep into the posterior half of the Sylvian fissure. This may have caused a disruption of the impulses traveling via the corpus callosum from the left hemisphere (damaged) to the right hemisphere causing a dyschronization of the neural responses or neural activity responsible for generating the activity over the Cz and C4 electrodes. The lack of response over the left hemisphere (C3) is simply related to the fact that this is the electrode near the actual lesion site (Musiek et al, 1999).

The results of this study suggest that it is no longer good enough for clinicians and researchers to define the location of a lesion as being in the temporal lobe. Behavioral and electrophysiological test results associated with temporal lobe lesions can range from the absence of deficits as has been documented in patients with lesions limited to portions of the temporal lobe located distally to the primary auditory area (see review above) to severe or marked deficits as was documented in this case. Much of the existing skepticism regarding the efficiency

of central auditory tests in documenting central auditory nervous system compromise in patients with temporal lobe pathology may be dispelled if one takes into consideration the fact that not all portions of the temporal lobe have auditory functions. The finding of no obvious deficits in patients with lesions limited to the anterior sections of the temporal lobe does not argue against the utility of a central test in defining auditory dysfunction. To the contrary, negative findings in patients with lesions limited to the nonauditory areas of the temporal lobe would suggest that the test enjoys a high specificity rating. Threats to the efficiency of a test would come only when the test fails to document a deficit in auditory function in cases with compromise of the areas of the temporal lobe known to be responsive to auditory stimuli (see Turner, 1991 for a discussion of test efficiency). It is therefore important that investigators and clinicians who are attempting to correlate auditory findings with anatomical information be careful when delineating the lesion site. Advances in imaging techniques now make it possible to precisely identify both the location and extent of central auditory compromise. With this information in hand, clinicians and researchers are in an advantageous position to establish links between anatomical sites and central auditory tests.

SUMMARY

This case study elucidates the ways in which auditory function can be compromised in a patient when the primary auditory areas are affected. It also demonstrates how central auditory tests, when appropriately selected, are sensitive to lesions of key auditory areas. The patient's performance on the dichotic tests administered (digits and rhymes) is consistent with the classic finding of a contralateral ear deficit on dichotic speech tests in patients with hemispheric lesions affecting the auditory areas of the cortex. In addition, bilateral deficits were noted on the duration patterns test, as was expected given the site of lesion. What is especially significant in this case is that the contralateral ear deficits on the dichotic speech tests and the bilateral ear deficit on the pattern perception test were "marked" and reflected the location and the extent of the lesion site (i.e., the primary auditory area in the left hemisphere). A number of additional, but "unusual," findings were noted in this case. These included the observation of improved word-recognition scores with the provision of extended time between a patient response and the presentation of the next test item and the observation of bilateral deficits on the MLR test. Some possible explanations for the physiological bases of these observations have been suggested in this manuscript. However, these are curious and novel phenomena that are subject to further investigation. The identification of similar deficits in other patients would help to identify whether the observed results are common in patients with lesions limited to the primary auditory areas or whether the deficits noted are unique to the current patient.

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