

Clinical Report

Lateralized Auditory Symptoms in Central Neuroaudiology Disorder

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Frank Musiek*
Linda Guenette*
Krista Fitzgerald*

Abstract

Many individuals with central auditory nervous system (CANS) pathology/disorder report a variety of auditory symptoms with difficulty hearing in noise being one of the most common complaints (Chermak and Musiek, 1992). Interestingly, there seems to be a paucity of information in the literature on lateralized hearing loss symptoms in patients with central auditory disorders. Reported here is a case where the patient reported auditory symptoms, specifically hearing loss that was lateralized distinctly to the right ear. This ear was contralateral to a stroke that involved the left hemisphere with neural compromise limited primarily to the left Heschl's gyrus. Although the patient reported other hearing difficulties, the lateralized symptom of right-sided hearing loss seemed to be the most noticed. While the pure-tone audiogram was totally disassociated from the right ear auditory symptoms, central auditory tests revealed a marked and consistent right ear deficit, which was consistent with the site of lesion. It is rare that a relatively small lesion in Heschl's gyrus results in the perception of hearing loss so specifically localized to the right ear. It is also of interest that a patient with definite complaints of hearing loss would yield a normal, highly symmetrical pure-tone audiogram. The triad of patient symptoms, anatomy of the lesion, and central auditory test findings in this case are discussed, and theories as to possible underlying mechanisms for the patient's auditory deficits are provided.

Key Words: Auditory brainstem response, central auditory processing, middle latency response

Abbreviations: ABR = auditory brainstem response; MCA = middle cerebral artery; MLR = middle latency response; MRI = magnetic resonance imaging; SLP = speech-language pathology

The relationship between auditory cortex compromise and related auditory symptoms has been demonstrated in many instances (see Musiek et al, 1994; Musiek and Baran, 2004). The symptoms linked to damage of auditory cortex and/or its closely surrounding areas include difficulty hearing in noise, understanding speech, appreciating music, localizing sounds, interpreting verbal messages, and so on (Scherg and von Cramon, 1986; Baran et al, 2004; Bamiou et al, 2006). These symptoms are usually related to lesions involving one hemisphere or a unilateral auditory pathway. Bilateral involvement of Heschl's gyri can result in profound deficits in hearing sensitivity by both subjective complaint and as measured by the pure-tone audiogram

(Musiek et al, 1994, 2007). In their most severe forms, these types of bilateral lesions can result in central deafness, that is, if the lesions are large and specifically localized to key auditory areas of the brain. In cases of central deafness the subjective complaint of little or no hearing sensitivity is associated with poor pure-tone thresholds. However, it is important to note that in central deafness the pure-tone deficit is one of central origin and is not mediated at the periphery (Musiek et al, 1994, 2007). In unilateral lesions involving the central auditory nervous system (CANS), pure-tone sensitivity is generally not affected to any significant degree even though symptoms of hearing difficulties are reported (see Musiek et al, 1994). This is likely related to the well-known concept

*Department of Speech, Language, and Hearing Sciences, University of Connecticut

Frank Musiek, 850 Bolton Rd., Storrs, CT, 06269; E-mail: Frank.Musiek@uconn.edu

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that pure-tone stimuli do not place great demand on the CANS as there are bilateral inputs to the CANS and one primary auditory cortex is often sufficient to process this simple stimulus. Therefore, laterality of sensitivity deficits or hearing difficulties is seldom reported in patients with lesions of the higher auditory system. More often the symptoms reported by individuals with CANS disorders are related as "auditory difficulties" and are not characterized by patients as "hearing loss." For example, in a previous case with left-sided Heschl's gyrus involvement (Baran et al, 2004), the patient related extreme difficulty hearing in noise, following conversations when more than one person was speaking, and understanding fast talkers. However, this patient did not distinctly lateralize symptoms to one ear or the other. This patient had a normal pure-tone audiogram bilaterally and was approximately the same age as the patient in the present case. Although not as anatomically specific to Heschl's gyrus as was the case just discussed, other cases of central auditory involvement with associated auditory symptoms (but without specific, lateralized complaints of poor hearing sensitivity) and normal pure-tone audiograms have been reported. Cases of central auditory involvement involving Heschl's gyrus, the temporoparietal area, the insular cortex, and the corpus callosum have typically revealed normal pure-tone thresholds and significant auditory symptoms, but not the lateralization of the auditory symptoms to one ear (Musiek et al, 1984, 1994; Bamiou et al, 2006). In the case presented here, the patient clearly and emphatically related a loss of hearing sensitivity, which was lateralized to his right ear. Although he did experience other symptoms, they were not as noticeable to him as was the loss of hearing, specific to one ear.

CASE HISTORY

This case is of a right-handed 42-yr-old male who suffered a left temporoparietal hemorrhage involving a branch of the middle cerebral artery (MCA). Given that the lesion compromised Heschl's gyrus, it was likely that a posterior branch of the MCA (possibly a branch of the angular artery, which courses from the posterior MCA) may have been involved (Musiek and Baran, 2007, pp. 293–294). This, in turn, would have compromised the medial portion of Heschl's gyrus and may have also involved a small portion of the posterior insula (see the radiology section). Prior to his stroke, the patient had reportedly experienced no hearing difficulties and/or neurological involvement. He is a father of two children and was active in a business career.

Immediately following his stroke, the patient reported word-finding difficulties, numbness in his right limbs, mild cognitive impairment, headaches, poor stamina, difficulty with attention, numbness specific to the right side of his body, and poor ability to hear anything of significance from his right ear. More specifically, his perception

was that he had a "hearing loss" in his right ear and described it as such. Notable improvement in cognition, occurrence of headaches, and attention were reportedly noticed shortly after the stroke; however, his hearing difficulties persisted with only minor subjective improvements. He was unable to hear adequately on the telephone with his right ear, experienced difficulty hearing in noise and carrying a tune when singing with his children, and frequently asked for repetitions during conversations. At the time of the present evaluation (approximately 3 mo after the stroke), the patient was functioning at a high cognitive level. That is, he was actively involved in a high-level demanding business, which required good communications skills and sophisticated decision making. At this time, he could carry a tune but could not comprehend verbal messages when the auditory signal was only available to his right ear (e.g., when listening on the telephone); therefore, the patient primarily used his left ear to process auditory stimuli. Unfortunately, this compensatory strategy was not effective when he was in noise. In such situations, the patient often missed what was being said. As stated earlier, the patient reported definite hearing loss in his right ear, which he strongly lateralized to his right ear, similar to patients who may have had a severe peripheral deficit in one ear. The patient had no family history of central auditory processing or learning disorders and reported no tinnitus, dizziness, or aural fullness. He had a history of borderline hypertension and was taking 50 mg of Toprol daily to treat it. There was no cranial neuropathy reported except for a mild central right facial weakness.

Immediately after the stroke the patient was seen as an inpatient by a speech-language pathologist (SLP) and was diagnosed with expressive aphasia. The SLP noted that the patient had some difficulties finding the "most optimal" word when speaking and recommended home exercises that involved word retrieval, working verbal memory, and complex auditory comprehension.

At a 2 mo follow-up, the SLP noted "excellent gains" and reported that the patient was functioning nearly normally for speech, language, and auditory comprehension. At that time the patient was using compensatory strategies in difficult listening situations for auditory comprehension, such as focusing on using his left ear to listen to the speaker and relying more on visual cues. Shortly after his speech and language consult, the patient had returned to work and reported that he was doing well. He reported that his auditory comprehension in quiet was "100% and in noise was 80% to 100%" but noted a continuous problem hearing from his right ear.

The SLP recommended home exercises that involved word retrieval, working verbal memory, and complex auditory comprehension. The patient was referred for an audiological consultation with central auditory processing testing, just prior to dismissal from speech and language therapy. The patient pursued an audiological

evaluation at another center to rule out a peripheral hearing loss. Test results at that time demonstrated normal hearing sensitivity (<20 dB HL) in both ears and excellent word recognition scores (100%) bilaterally, and the patient was referred to our center for a central auditory processing evaluation.

RADIOLOGY AND NEUROANATOMY

Magnetic resonance imaging (MRI) showed the lesion site was in the posterior, medial segment of the left temporal plane likely involving the medial aspect of Heschl's gyrus and possibly the most posterior aspect of the insula. In the views examined, it was difficult to identify if a small part of insula was also involved. When viewing a lateral image of the MRI, the lesion appeared to be located in the more inferior aspect of Heschl's gyrus. However, inspection of more medial slices of the MRI revealed that the medial aspect of Heschl's gyrus was more directly involved (Figs. 1 and 2). The lesioned area also showed some edema that may have extended slightly beyond Heschl's gyrus—primarily in its more inferior aspect. The extensive MRI, which provided sagittal sections from the most lateral aspect of the left hemisphere to the lateral aspect of the right hemisphere, including cerebellum and brainstem, revealed no other lesions.

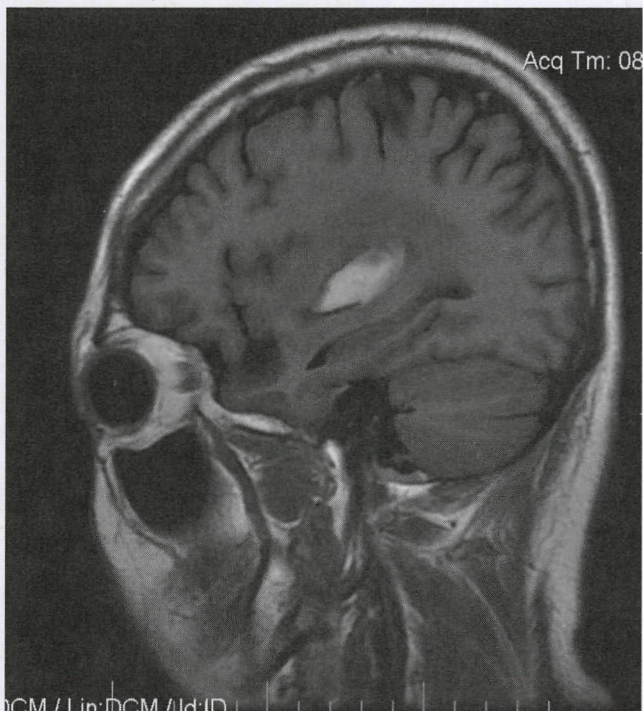


Figure 1. An MRI showing a lateral view of a sagittal section of the patient's brain revealing the lesion site.

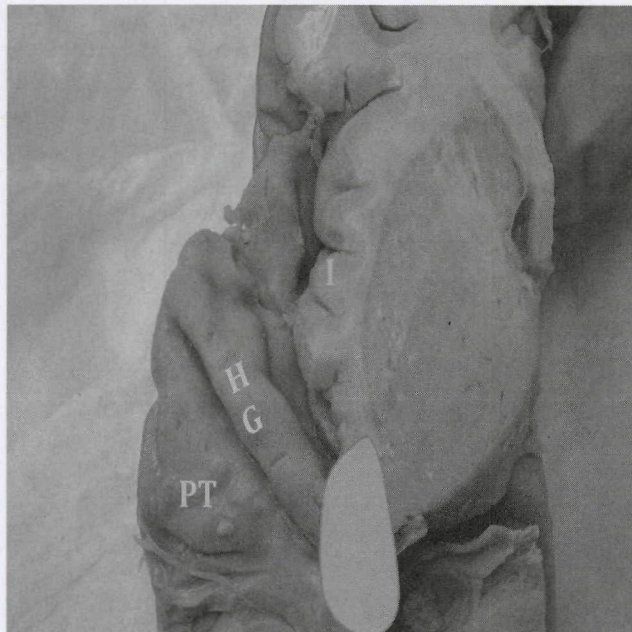


Figure 2. A view of the superior temporal plane of a laboratory brain with an approximation of the lesion site and relative size of the compromise in the current patient. I = insula; HG = Heschl's gyrus; PT = planum temporale.

METHODS

All testing was administered for both ears in a double-walled sound-treated booth. Pure-tone thresholds were established using a Grason-Stadler GSI-61 audiometer and the modified Hughson-Westlake technique. Speech recognition thresholds were assessed using a spondee word list, and speech recognition measures were established utilizing the Northwestern University (NU-6) word lists. Immittance testing included tympanometry and acoustic reflex thresholds, which were acquired using a GSI Tymptstar in a conventional manner.

Auditory brainstem responses (ABRs) were acquired using a 100 msec click presented at 80 dB nHL through ER-3A insert phones at rates of 17.3 and 77.3 clicks per second via the Nicolet Spirit 2000. Filtering was 100–3000 Hz with a 12 dB per octave roll-off. The electrodes were placed at Cz and referenced to either A1 or A2 (earlobe). Middle latency response (MLR) testing was conducted on the same equipment using the same click stimulus but presented at 70 dB nHL at 9.7 clicks per second with the filters set at 20–1500 Hz and the same filter roll-off characteristics as mentioned earlier. The electrode impedances during both ABR and MLR testing were maintained at less than 5 kohms across electrodes.

The behavioral central auditory tests administered included dichotic rhymes (Musiek, et al (1989), dichotic digits (Musiek, 1983), compressed speech (Bornstein, 1994), competing sentences (Willeford, 1977), frequency patterns (Musiek and Pinheiro, 1987), and gaps-in-noise (Musiek et al, 2005). The descriptions of these procedures have been

well documented in numerous publications and therefore will not be detailed here (see references provided above and Baran and Musiek, 1999; American Academy of Audiology, 2010). All these procedures were administered at a 50 dB SL relative to the patient's spondee thresholds except for the competing sentences test for which the target and competing messages were presented at 35 and 50 dB SL, respectively.

RESULTS

The pure-tone audiogram revealed normal hearing sensitivity bilaterally with speech recognition scores essentially within the normal range for both ears (Fig. 3). Tympanograms were within normal limits bilaterally according to the American Speech-Language-Hearing Association (ASHA) guidelines (1990), and acoustic reflex thresholds assessed at 500, 1000, and 2000 Hz were grossly within the normal to borderline normal range bilaterally (e.g., see Table 1) (Wilson and Margolis, 1991).

The ABR revealed normal absolute and interwave latencies bilaterally, and interaural latency difference (ILD) values for waves V and III were well within the normal range. There were no significant shifts of wave V for either ear at high repetition rates (Musiek and Baran, 1991) (Fig. 4).

All central auditory behavioral tests revealed poorer performance for the right ear compared to the left ear (Fig. 5), with the patient's performance on all six central auditory tests falling outside of the normal range. In addition, the patient's left ear scores for the dichotic rhyme and time compression

Table 1. Acoustic Reflexes of Right and Left Ear (thresholds in dB HL)

Hz	500	1000	2000
Probe RIGHT			
Stimulus Left	105	105	110
Stimulus Right	90	90	95
Probe LEFT			
Stimulus Right	110	100	100
Stimulus Left	90	95	90

tests fell below established norms. In comparing left and right ear scores across all the behavioral tests, the average difference was 42% yielding a decided left ear advantage.

The MLR revealed an amplitude asymmetry with greater amplitude recorded from the C4 electrode (right hemisphere) than from the C3 electrode (left hemisphere). The latencies of the MLR waves were all within the normal range for each ear and both electrode sites with no latency asymmetries noted between the two ears (Fig. 6).

Intervention

The recommendations for this patient focused on the utilization of fundamental auditory training tasks that were modified in such a way that a busy adult could manage them. The first suggestion was to work on localization and sound identification skills while simultaneously training underlying temporal (needed for sound localization) and linguistic processes. Several techniques were recommended to achieve these goals. One technique was for the patient to take a walk outside to listen for environmental sounds (such as a bird chirping). He was to then localize and identify these sounds as quickly as possible. This was to be done daily or as often as possible. Ideally this therapy was conducted with an accompanying person to provide feedback and support. A second intervention was to use a modification of the dichotic interaural intensity difference training (DIID) training program (Weihing et al, 2007) incorporating phone usage for the right ear. In brief, the patient practiced using the phone on the right ear in quiet situations and then progressed to more noisy surroundings (creating more acoustic competition in the left ear). He was encouraged to practice this more formally at home by talking to family members on the phone and regulating the level of an acoustic competition presented to the left ear. When possible he was to try to integrate this into his work environment. The final intervention was humming and singing along with music. This musical training was to be done whenever possible to help improve temporal processing (see Kraus and Chandrasekaran, 2010).

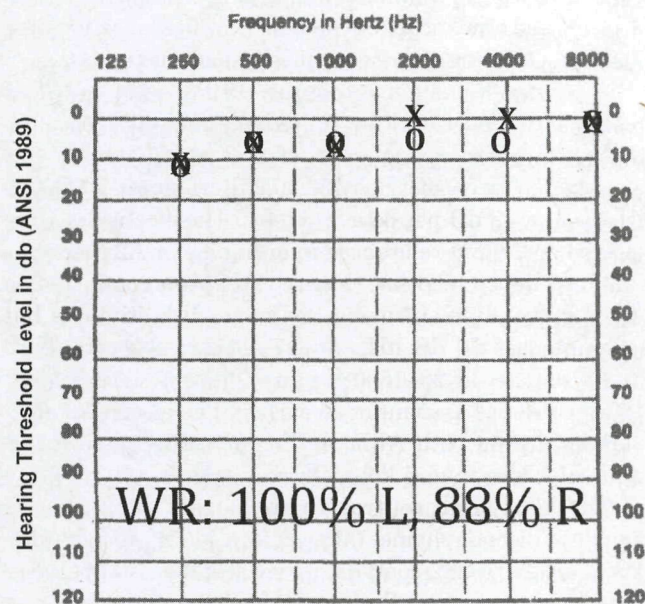


Figure 3. Pure-tone air conduction thresholds and word-recognition (WR) scores for left and right ears.

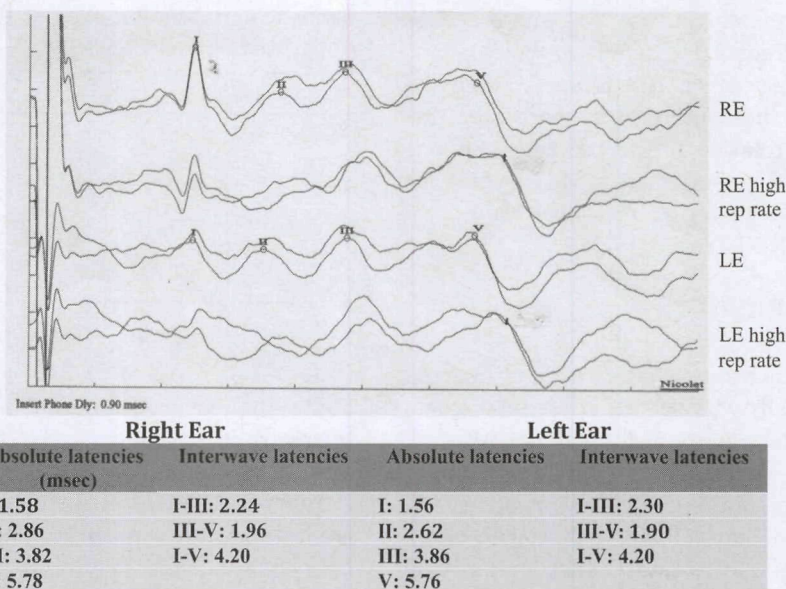


Figure 4. Auditory brainstem response (ABR) waveforms obtained with insert phones for left and right ears at low and high repetition rates. Absolute and interwave measures for both ears are displayed below the ABR tracings.

Follow-Up Evaluation

During a follow-up appointment, the patient reported that subjectively his hearing had improved and claimed that he functioned well in everyday situations. However, he admitted that he still depends more on his left ear than his right ear, but he appears to have accommodated well in doing this. Central auditory testing during follow-up indicated an improvement in right ear performance on dichotic digits and competing sentences to within the normal range, although results on other test procedures (behavioral and electrophysiological) have not changed. It is of interest to note that because the patient was doing reasonably well, he was not as motivated to follow therapy directives—although the recommendations offered at the time of his follow-up evaluation remained similar to those offered at his earlier evaluation and were strongly advocated by clinicians.

DISCUSSION

The central auditory tests were consistent with the lateralized symptoms of the patient while the pure-tone thresholds failed to provide insight either into the patient’s symptoms or to the lateralization of those symptoms to the right ear. As was noted in the introduction, individuals with neurologically based central auditory disorders generally do not specifically complain of loss of hearing (sensitivity) and usually do not lateralize their symptoms as in the case described here. A similar case, described in the introduction, is worthy of mention here. Both that case (Baran et al, 2004) and the present case were of a similar age; both had normal pure-tone audiograms; and both had compromise of Heschl’s gyrus

secondary to strokes. However, the previous case did not lateralize symptoms to a specific ear. There also was a difference in the extent of the lesions in these two cases, with the lesion in the earlier case involving most of Heschl’s gyrus, whereas the lesion in the present case involved only the medial aspect of Heschl’s gyrus.

It may be useful to theorize about the underlying mechanisms in this case—especially the subjective impression of hearing loss in the right ear with normal pure-tone thresholds but severe deficits for that ear on central auditory tests. This discussion will address two areas, although they certainly could be related: (1) misrepresentation of intensity (loudness) at the cortical level and (2) the possibility of partial auditory neglect and related issues affecting interhemispheric interactions.

Since the present patient with a confirmed auditory cortex lesion reported the perception of hearing loss in one ear but not the other, a logical starting point for discussion is the processing of intensity and/or its subjective counterpart, loudness. Intensity representation at the level of the cortex is related to neuronal firing rate (i.e., the greater the intensity, the higher the firing rate), the number of neurons stimulated (i.e., the greater the intensity, the more neurons respond), and the interaction between excitatory (E) and inhibitory (I) neurons. Damage to the auditory cortex could reduce the firing rate and number of viable fibers, which in turn, could alter the perception of loudness at the cortical level (see Musiek and Baran, 2007, for review; Phillips et al, 1994). For example, if more I neurons are damaged than E neurons, then E fibers dominate and the sound is perceived as louder and vice versa. On the other hand, if more E fibers are damaged than I fibers, then the perception of loudness would be decreased. Therefore, neuronal

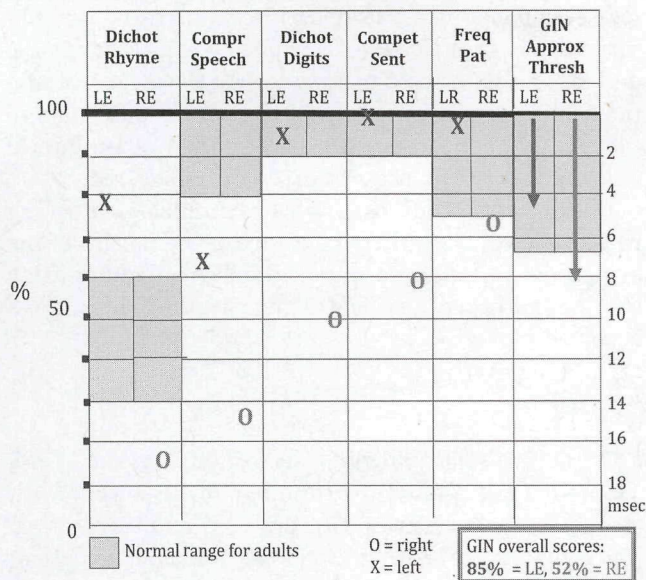


Figure 5. Central auditory behavioral test results for left and right ears. Dichot = dichotic; Compr = compressed; Compet Sent = competing sentences; Freq Pat = frequency patterns; GIN Approx Thresh = gaps in noise approximate threshold.

response to intensity from this damaged area in one hemisphere compared to the other will be reduced. This notion is supported by Young's (2010) comprehensive review of level and spectrum, which relates that in the auditory cortex neurons have a wide range of thresholds to stimulus level in the normal condition. Young also mentions that these neurons may be well tuned for both level and spectrum. Therefore it would follow, similar to the E and I interaction, that if a high proportion of high or low threshold neurons were damaged, the result would be an imbalance among the intensity neurons, which would likely influence the perception of loudness at the cortical level. Of course, the other possibility is that if approximately an equal number of E and I and/or high and low threshold fibers are damaged, then the asymmetry of loudness may not be appreciated. This may be why not all individuals with cortical lesions report the same lateralized hearing loss symptoms as does the present case.

Then why in the present case are the pure-tone thresholds normal and symmetrical? It is well known that a pure tone is a simple stimulus that requires little cortical substrate to be processed. Even with damage, there is likely sufficient cortical processing power to preserve good thresholds for pure tones and other simple acoustic stimuli. This may be demonstrated in a very practical manner. Though the patient has normal pure-tone thresholds, few individuals listen to pure tones often during a typical day. There are many complex acoustic stimuli and situations in everyday life that could put greater demands (than pure tones) on the auditory cortex, and if it is damaged it may not cope with these demands, resulting in a

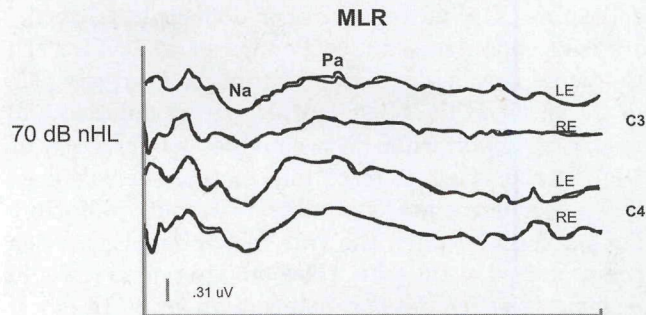


Figure 6. Middle latency response (MLR) waveforms for left and right ears at C3 and C4 electrode sites.

subjective impression of hearing loss for these complex acoustic signals and conditions. Certainly the central auditory tests in this case yielded major deficits, and they are, by design, complex and demanding.

The next theoretical consideration is that of partial or mild auditory neglect. Though neurological neglect is common in the neurological literature, it is seldom discussed in audiology (see Musiek, 2009, for review). However, some reports of auditory neglect have been published and are relevant here (Musiek, 2009). Auditory neglect is usually demonstrated by very poor performance from one ear such as in dichotic listening or errors in localization primarily for sound originating from one side (Marshall, 2001, Bellman et al, 2001). As previously proposed, this is likely a combination of deficits in hemi-attention and central auditory processing (Clarke and Thiran, 2004; Musiek, 2009). Though the present patient certainly does not have a full expression of auditory neglect, the interpretation of hearing loss at the right ear could possibly be a mild form of neglect. The patient does show right ear deficits on all dichotic tasks. Interestingly, in patients with auditory neglect, pure-tone thresholds may not be affected, but deficits are manifested on more complex procedures (i.e., localization, dichotic listening) (Musiek, 2009). It was reported that increasing the intensity of various stimuli has no effect on the degree of auditory neglect in patients who have suffered a stroke. This noneffect of intensity aligns well with the theory that sound intensity is represented in the central nervous system as encoded information instead of a linear representation (Bamiou et al, 2012). This kind of intensity dysfunction could play a role in the present patient's perception of hearing loss. Though trials of intensity increments were not applied in the evaluation, the report by Williams and Coleman opens the door to the possibility that improper loudness perception can be a factor in auditory neglect. This partial auditory neglect could be rooted in, or at least associated with, interhemispheric dysfunction.

Cortical damage in auditory areas can influence the transfer of information from one hemisphere to the other. That is, if an area of the cortex is damaged, the fibers from that area that project across the corpus

callosum (CC) will be absent or damaged; hence the transfer compromised (see Pandya et al, 1971). The CC is responsible for not only transfer from one side of the brain to the other but also for modulation and mediating equilibrium between the hemispheres as well as the integration of information (Kinsbourne, 2003). Perhaps the CC's ability to reach equilibrium for loudness between the two hemispheres has been compromised by the cortical lesion. Therefore, it would be logical to consider this interaction between hemispheres (comparison of intensity representation on one side versus the other) as a probable function for interpreting loudness at this high brain level. It therefore seems possible that these two areas of discussion—aberrant cortical representation of intensity and mild auditory neglect—could separately or in some combination play a role in this patient's subjective complaints and objective auditory findings. If not, they should at least stir interest in further discussion and investigation of these kinds of clinical problems.

There is one other topic pertaining to this case that is worthy of some comment. Why is there such a paucity in the literature of individuals with central auditory lesions and lateralized, subjective hearing loss like in the case discussed. Is this condition really a rare auditory profile? What are some possible reasons for this? Perhaps the appropriate questions about lateralization and the perception of hearing loss in central cases are not asked by the examiner. Often many patients with neurological involvement are not seen by audiologists, and therefore, it is possible that detailed questions about hearing symptoms simply are not asked in the patient interview. In a recent study, it was reported that a number of patients with strokes of the central auditory system did not report hearing difficulty unless they were asked detailed and specific questions about their hearing. When they were asked these more specific questions, various auditory symptoms came to light (Bamiou et al, 2012). Also, it maybe that lateralized hearing loss symptoms are present in neuroaudiologic cases but simply are not reported in the literature. Again, depending on the background of the investigator/author, specific information about hearing may be overshadowed by other findings and therefore not reported. Sometimes subtle auditory symptoms take a backseat to other more acute neurologic symptoms in regard to both the patient and the clinician (see Musiek et al, 1994, pp. 29–30). Another factor to consider may be semantics. At times *hearing loss* may have different meanings to different people. To most people and clinicians, hearing loss is usually linked to decreased sensitivity or acuity (i.e., something very restrictive), but possibly for some, it may mean practically any kind of hearing problem. From our own experience, it seems that those with central auditory problems seldom report having hearing

loss—they usually use descriptors such as *hearing difficulties, problems understanding, distorted hearing*, and so forth, as opposed to *hearing loss*. However, “semantic” factors could influence how often important and informative symptoms, such as the perceived unilateral hearing loss in the present case, are uncovered.

It is hoped that this case will alert audiologists seeing these kinds of patients to be aware of the nature of the symptoms reported so more audiological information can be gathered to shed light on the phenomena reported here.

SUMMARY

This case study provides an insight into the symptoms and related test findings for a patient with a well-defined lesion of the primary auditory cortex. The symptoms of hearing loss as described by the patient were lateralized to the right ear, which was consistent with the central auditory test findings for this patient but not with his pure-tone audiogram. The patient's perception of a “unilateral hearing loss” is unusual, especially since it was accompanied by a normal, symmetrical pure-tone audiogram. In our experience, distinct perceptual lateralization of hearing loss is rarely reported in central auditory disorders that are neurologically based. Demonstrated here is the potential value of central auditory testing to not only corroborate a patient's symptoms but also to define the laterality of these symptoms. In this report, two theoretical mechanisms were entertained as the bases for the current patient's symptoms and test results. First, the symptom of hearing loss for the right ear could be secondary to poor representation of intensity at the cortex due to insufficient neural substrate (number and firing rate of fibers) and/or an imbalance of available excitatory and inhibitory fibers. The second possible mechanism could be a mild form of auditory neglect that disallows the patient to fully appreciate intensity cues for the right ear. These proposed mechanisms could individually or in some combination be related to the subjective symptoms and objective test findings in this case. It is indeed difficult to know for sure. However, what is clear is that, as has been reported before, the audiologist must carefully listen to the patient, and if their symptoms are not faithfully reflected in basic tests of auditory function, then further testing should be completed. In this case, the further testing included a central auditory test battery that helped to define the problem and corroborate the patient's subjective complaints. It also served as a guide for counseling and designing a therapy plan for the patient.

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