Limitations of Pure-Tone Audiometry in the Detection of Nonorganic Hearing Loss: A Case Study

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ABSTRACT: Detection of a nonorganic hearing loss (NOHL) can pose a challenge for the clinical audiologist, especially if the diagnosis is dependent solely on behavioral testing. Detection can become particularly difficult if the NOHL is manifested at an unconscious level, as presented in this case study of a 48-year-old woman who was diagnosed with a bilateral severe-to-profound sensorineural hearing loss and fitted with monaural amplification based on pure-tone audiometric data. Several years after the initial diagnosis, a nonorganic component was detected based on physiologic test procedures and confirmed by a psychiatric evaluation. This case demonstrates the importance of the use of physiologic measures such as otoacoustic emissions, the middle ear muscle reflex, and auditory brainstem response testing as a cross-check for behavioral assessment and the incorporation of these tests in routine clinical practice.

KEY WORDS: adjustment disorder, auditory evoked potentials, conversion disorder, middle ear muscle reflex, nonorganic hearing loss, otoacoustic emissions

Pure-tone audiometry continues to be the mainstay of modern audiology and the most used procedure to diagnose auditory disorders. Hearing sensitivity levels obtained with pure-tone audiometry using appropriate psychophysical protocols and analysis procedures are generally reliable. One of the factors that can affect test reliability of pure-tone audiometry, however, is the interrelationship with client performance; if the client cannot or does not want to cooperate, the test results can be questionable. The reliability of pure-tone audiometry becomes particularly vulnerable when dealing with a nonorganic hearing loss (NOHL). An NOHL appears greater in magnitude than may be explained on the basis of any known auditory pathology (Martin, 2002). The presence of an NOHL, in most instances, is related to some kind of personal gain, which may be the expectation of monetary compensation, a need to gain attention from others, or, especially in children, an excuse for poor academic performance (Rintelmann & Schwan, 1999).

Although the classic literature suggests that there are no characteristic audiometric patterns that typify NOHLs (Chaiklin, Ventry, Barrett, & Skalbeck, 1959; Ventry & Chaiklin, 1965), one aspect of NOHL that is widely accepted in the literature is the high incidence of a true underlying hearing loss among adults diagnosed with NOHL (Chaiklin & Ventry, 1963; Gelfand & Silman, 1985, 1993). Further, in most cases of NOHL, it seems that these individuals use an internalized loudness-based anchor to present with the “exaggerated” hearing loss (Gelfand & Silman, 1985, 1993; Martin, Champlin, & McCreery, 2001). Typically, the exaggerated behavioral responses will show discrepancies between the different test results, which is not the case for most organic hearing losses. Common discrepancies include lack of reliability across test sessions; wide threshold ranges; speech recognition threshold (SRT) pure-tone average (PTA) disagreement, with the SRTs being considerably better; half-spondee responses; bone conduction thresholds poorer than air conduction thresholds; and lack of cross hearing (Gelfand & Silman, 1993; Martin et al., 2001).
Audiologic assessment of an NOHL can pose a major challenge for the clinical audiologist, with the diagnosis becoming even more problematic when that hearing loss is manifested at an unconscious level as a symptom of a psychological disturbance or disorder. An example of such a condition that is cited most commonly in the literature is a conversion disorder or a psychogenic hearing loss. Hysterical deafness is an older term used to describe a conversion disorder (Martin, 1986). These terms imply that the patient may have an emotional or psychological disorder resulting from a psychological conflict or need (Wolf, Birger, Shoshan, & Kronenberg, 1993).

Although there is some disagreement within the profession regarding conversion hearing loss, whether it is a true clinical entity (Beagley & Knight, 1968; Cohen et al., 1963; Wolf et al., 1993) or whether every NOHL is feigned (Goldstein, 1966), the psychiatric literature recognizes the presence and nature of conversion disorders. According to the American Psychiatric Association’s (APA’s, 1994b) Diagnostic and Statistical Manual of Mental Disorders (DSM–IV), a conversion disorder is a somatoform disorder with the symptoms or deficits affecting voluntary motor or sensory function, including hearing, that suggest a neurological or other general medical condition. The symptoms do not conform to known anatomical pathways or physiological mechanisms but instead follow the individual’s conceptualization of a condition. The DSM–IV (APA, 1994b) definition indicates a temporal relationship between a psychologically stressful situation and the appearance or exacerbation of the symptom. The symptom or deficit may be acute or chronic, often it is recurrent, and it is not intentionally produced or deliberately feigned. An associated feature of conversion disorder is la belle indifference (the relative lack of concern about the nature or implication of the symptom). It is more prevalent in rural populations, in individuals of lower socioeconomic status, and in individuals less knowledgeable about medical and psychological concepts. Conversion disorders appear more frequently in women, with reported ratios varying from 2:1 to 10:1, and often with an accompanying personality disorder of the histrionic type. In women especially, the symptoms appear much more commonly on the left side than on the right side of the body. In men, conversion disorder is often seen in the context of industrial accidents or the military, in which case it must be carefully differentiated from malingering (APA, 1994b).

As a group, individuals who manifest an NOHL demonstrate certain emotional and psychosocial characteristics. According to Trier and Levy (1965), they tend to score lower on all measures of socioeconomic status. They also score lower on tests of verbal intelligence and demonstrate a greater degree of clinically significant emotional disturbances. In an early study, Gleason (1958) reported that these individuals, as a group, typically tend to display emotional immaturity, instability, and an inadequate response to social demands. They also tend to manifest a reliance on denial mechanisms and appear to have a diminished sense of confidence in their abilities to meet the needs of daily living (Martin, 2002).

The majority of individuals with NOHL are diagnosed within a relatively short time or the functional component resolves when the expectation of the personal gain has been met. There have been some cases cited in the literature, however, where the NOHL was not diagnosed and these individuals were referred for a cochlear implant. The NOHL component was eventually identified by the cochlear implant assessment team with considerable time and expense spent on the diagnosis (Spraggs, Burton, & Graham, 1994).

Although the literature is replete with examples of cases of NOHL, the present case is somewhat unique because the individual had been in the health care system for at least 6 years before discovery of the NOHL. During that period, she had several audiologic evaluations and had been diagnosed with a profound sensorineural hearing loss in the right ear and a moderately severe sensorineural hearing loss in the left ear, had been fitted with monaural amplification, and had been under the care of an otolaryngologist—without the nonorganic nature of the hearing loss being detected.

### CASE STUDY

In 1998, JD, a 48-year-old homeless woman, was referred to a Midwest university’s speech and hearing clinic by her case manager at the local vocational rehabilitation (VR) district office. In 1992, JD had been fitted with an in-the-ear hearing aid in the left ear by an audiologist working in a local otolaryngology clinic. During the 9 months before referral to the university clinic, the VR case manager had referred JD to four hearing aid dispensers for free hearing evaluations. A pure-tone threshold assessment had been performed at all four sites. The test results indicated a profound bilateral sensorineural hearing loss with no response to pure tones at output limits of the equipment. The VR case manager also had referred JD to a local otolaryngologist who had evaluated JD in 1996. In his report, the otolaryngologist had stated that JD’s current audiogram showed a bilateral sensorineural hearing loss with no response to pure tones, which was not in agreement with her 1996 audiogram when she had presented with a profound sensorineural hearing loss in the right ear and a moderately severe sensorineural hearing loss in the left ear. The otolaryngologist reported no change in her medical history and no medical indication for the significant change in hearing. He added that JD had “normal” speech but responded to his questioning through a sign language interpreter. He recommended repeating the pure-tone test. No physiologic assessment other than tympanometry, which was completed only at the otolaryngology clinic, was performed at any of the test sites. The VR case manager had reliability concerns regarding JD’s behavioral test results because she did not believe that JD’s speech was consistent with a profound bilateral sensorineural hearing loss. She had, therefore, referred JD to the university clinic for physiologic audiometric assessment.

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1JD is a pseudonym used to protect the client’s privacy.
JD arrived for her appointment at the university clinic an hour late dressed in a night shirt. Her face was devoid of affect and she spoke in a barely audible whisper. JD was able to follow directions and answer questions at normal conversational levels without difficulty and without visual cues, including at one point when both her ears were plugged by immittance probe tips and the clinician’s back was to her. She reported a history of severe and recurrent ear infections as a child and poor hearing in her right ear for at least 20 years. She attributed the poor hearing to either childhood ear infections or sinus infections later in life. She described the current hearing ability in her right ear as “plumb gone.” She did not report any symptoms of a balance disorder such as dizziness, tinnitus, or vertigo. The rest of the medical history was unremarkable. The client was currently unemployed and stated that the hearing loss did not prevent her from working but that sometimes “it was frustrating.” She reported some noise exposure for 2 years while working as a seamstress at a local aircraft manufacturing plant. JD stated that she wanted the VR office to provide her with a new hearing aid for the left ear because her hearing had become worse in that ear. She did not have her hearing aid with her and was vague as to its whereabouts. During the initial interview and all through the evaluation, JD exhibited an affect that was flat and appeared indifferent to the severity of her reported disability. 

All tests were performed in a double-walled IAC sound-treated booth (Industrial Acoustics Company, Inc., Bronx, NY) with equipment calibrated according to American National Standards Institute (ANSI, 1996) S3.6-1996 guidelines. Otoscopy revealed normally appearing pinna, ear canal, and tympanic membrane bilaterally. Immittance testing was performed with a GSI immittance bridge (Grason Stadler, Inc., Fitchburg, WI). Tympanometry was performed with a single-component (Y-226 Hz) probe tone. The tympanogram obtained from both ears showed normal peak compensated static admittance (peak $Y_{tm}$), equivalent ear canal volume ($V_e$), tympanometric width (TW), and tympanometric peak pressure (TPP) (American Speech-Language-Hearing Association, 1997; Margolis & Heller, 1987; Shanks & Wilson, 1986; Shanks, Lily, Margolis, Wiley, & Wilson, 1988; Wiley et al., 1996), which was consistent with normal middle ear function. Tonal middle ear muscle reflex (MEMR) levels were within normal limits with ipsilateral and contralateral stimulation at 500, 1000, and 2000 Hz, with no response elicited at 4000 Hz in the left ear, which is not an uncommon occurrence at 4000 Hz for middle-aged and older individuals (Fria, LeBlanc, Kristensen, & Alberti, 1975; Gelfand, 1984, 1994, 2002). Tonal MEMR thresholds, however, were slightly worse for the left ear than for the right ear (see Table 1). Acoustic reflex decay with contralateral stimulation was negative for each ear at 500 and 1000 Hz.

An attempt was made to establish SRT using a GSI 10 diagnostic audiometer (Grason Stadler, Inc., Fitchburg, WI) with TDH-50 headphones (Telephonics, Huntington, NY) and MX 41/AR cushions. JD did not respond to any of the spondaic word stimuli in either ear even at maximum audiometric output. Pure-tone behavioral testing was attempted next, with repeated instructions to the client to respond if she heard or felt anything. JD did not respond to the air conduction tonal stimuli even at the maximum output of the audiometer at 1000 and 2000 Hz bilaterally, nor did she indicate any signs of discomfort or pain at the highest intensity level (120 dB HL). JD did respond to the clinician talking to her through the audiometer talk-forward up to an output level of 65 dB HL, but stopped responding when the intensity was decreased. At that point, behavioral testing was abandoned. Because of the lack of behavioral responses and also reliability concerns regarding the behavioral assessment, transient-evoked otoacoustic emission (TEOAE) testing was performed. TEOAEs are frequency-dispersive responses arising within the cochlea that can be measured in the external ear canal in response to brief stimuli such as clicks or tone bursts (Kemp, 1978; Norton, 1993; Prieve, Gorga, & Neely, 1996). TEOAEs are a noninvasive, rapid, and reliable assessment measure of the peripheral auditory system up to the outer hair cell level. A substantial body of literature exists describing the relationship of TEOAEs to hearing loss, with the generally accepted consensus being that the presence of TEOAEs is consistent with a hearing loss no greater than 30 to 40 dB HL (Glattke, Pafitis, Cummiskey, & Herer, 1995; Harrison & Norton, 1999; Hussain, Gorga, Neely, Keefe, & Peters, 1998). Reproducibility scores of the TEOAEs in the frequency bands from

**Table 1.** Tonal middle ear muscle reflex levels with ipsilateral (ipsi) and contralateral (contra) stimulation.

<table>
<thead>
<tr>
<th>Middle ear muscle reflex</th>
<th>Test frequencies</th>
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<tbody>
<tr>
<td></td>
<td>500 Hz</td>
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<tr>
<td><strong>Stimulus right</strong></td>
<td></td>
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<tr>
<td>Ipsi</td>
<td>80</td>
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<tr>
<td>Contra</td>
<td>95</td>
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<tr>
<td>Decay (contra)</td>
<td>Negative</td>
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<tr>
<td><strong>Stimulus left</strong></td>
<td></td>
</tr>
<tr>
<td>Ipsi</td>
<td>85</td>
</tr>
<tr>
<td>Contra</td>
<td>95</td>
</tr>
<tr>
<td>Decay (contra)</td>
<td>Negative</td>
</tr>
</tbody>
</table>

**Note.** NR = no response.
1.0 to 5.0 kHz, especially the reproducibility score at 2.0 kHz, appear to be the most efficient measure in separating normal and impaired ears, regardless of frequencies affected (Glatteke et al., 1995; Harrison & Norton, 1999). The TEOAEs in JD’s ears were evoked by an 88 dBpclick stimulus in the right ear and an 87 dBp click stimulus in the left ear generated by the commercially available ILO88/otoacoustic emission measurement system (Otodynamics, Ltd., Herts, UK). Based on current criteria (Hall, 2000; Hurley & Musiek, 1994), emissions were present at all five frequency bands from 1.0 to 4.0 kHz in both ears, including an overall waveform reproducibility of 96% in the right ear and 98% in the left ear (see Figure 1).

The auditory brainstem response (ABR) test was performed next as a cross-check to the TEOAEs and to rule out a retrocochlear pathology using a Traveler Express SE evoked potential system (Bio-logic Systems Corp., Mundelein, IL). The ABR generated from a normal otologic and neurologic auditory system consists of a sequential series of 5 to 7 peaks occurring within approximately 10 ms after stimulus onset. Auditory evoked potentials, in general, and the ABR, in particular, are extremely useful in evaluating the hearing sensitivity of clients who cannot or will not provide valid reliable hearing thresholds using behavioral methods. In addition, the ABR is most robust in identifying tumors of the eighth nerve greater than 1 cm in diameter (Chandrasekhar, Brackmann, & Devgan, 1995; Telian, Kileny, Niparko, Kemink, & Graham, 1989), with somewhat less success reported in diagnosing diffuse demyelinating conditions such as multiple sclerosis (Hood, 1998).

The ABR test with JD was performed with four runs in each ear. Two runs with a rarefaction and two runs with a condensation click stimulus polarity at 85 dB nHL also were completed to rule out the rare chance of an existing auditory neuropathy (dys-synchrony) even though the MEMRs were within normal limits (Berlin, 1999). ABR absolute latencies (the time interval between the stimulus onset and the peak of a waveform) and interwave latencies (the time interval between peaks of a waveform) for both ears were within normal limits (Hall, 1992b) (see Table 2). It is important to remember that the ABR is a test of synchronous neural function and not a test of hearing and thus cannot represent true, conscious, behavioral hearing (Hood, 1998). It is possible, however, to use this measure to make inferences about hearing sensitivity based on the presence of responses to sound stimuli at various levels of intensity by plotting the latencies of wave V as a function of intensity and, thus, obtain a latency-intensity function. The latency-intensity function in this case could have been useful in providing an estimation of JD’s actual hearing sensitivity, but, unfortunately, it was not determined because JD was getting restless, resulting in an increased number of artifacts and decreased reliability of the ABR test results. Table 3 provides an overview of the results of the diagnostic tests performed at the university clinic, demonstrating the discrepancies between the behavioral test results and the results of the physiologic assessment measures of the auditory system.

At the completion of the evaluation, JD was told that the results did not indicate as severe a hearing loss as had been demonstrated by the previous tests and that she may have to return for additional testing. She responded to this statement with no change in affect and appeared indifferent to the outcome of the evaluation. JD’s VR case manager was informed of the results and suggested a psychiatric evaluation rather than further audiological evaluations.

The preliminary diagnosis of the subsequent mental status evaluation was an adjustment disorder with mixed emotional features. An adjustment disorder is a maladaptive reaction to an identifiable psychosocial stressor, typically occurring within 3 months after the onset of the stressor and not meeting the criteria for any other specific diagnosis. Symptoms, though usually acute and transient, may last for years if the stressor continues (APA, 1994a). The differential diagnosis should be made from well-delineated disorders such as conversion disorder and depression (Gregory & Smeltzer, 1983). The psychologist reported that JD demonstrated “slightly flattened” facial expression and affect and spoke in a “very soft” voice but was able to communicate with him without the aid of a sign language interpreter. He categorized her intellectual function in the low “normal” range with memory that was intact and within normal limits for short-term auditory recall. JD reported to the psychologist that she had spent some time in a children’s home during her adolescence and was treated at the state mental hospital during her teen years. After the psychological evaluation, JD did not report back to the VR office, and, therefore, no follow-up psychological assessment or treatment was performed. Because of her homeless status, it was not possible to contact her to schedule follow-up audiological testing, despite repeated efforts.

**DISCUSSION**

Martin (2002) suggested that the function of the audiologist is to determine the true extent of the organic component, if present, rather than to determine the precise reason for the nonorganic component. Audiologists, however, also need to be aware of conditions other than a hearing loss that may bring an individual to their clinic and when the need for diagnostic tools beyond a pure-tone audiogram may become necessary. This case exemplifies the importance of physiologic measures such as the tonal MEMR, OAEs, and auditory evoked potentials such as the ABR as a cross-check for behavioral audiological thresholds.

One possibility for the delay in diagnosis of the NOHL in JD’s case may have been the lack of behavioral responses. Because of the absence of behavioral responses, the typical discrepancies between behavioral test results were not exhibited nor could behavioral diagnostic tests for NOHL be performed. Behavioral diagnostic tests for NOHL include the Stenger test when a unilateral NOHL is suspected. It is based on the Stenger principle, which states that when auditory stimuli are presented to both ears simultaneously, but with the stimulus presented to one ear being louder, only the louder sound will be perceived by the listener (Monro & Martin, 1977; Stenger, 1907; Taylor, 1949). A useful test for suspected bilateral NOHL is the
Figure 1. The upper half of the figure displays the transient-evoked otoacoustic emission (TEOAE) data from the right ear; the lower half displays the TEOAE data from the left ear. In the upper half of the figure, the top left box shows the temporal waveform of the click stimulus. The top right center “Response SNR” box displays the shaded waveform of the frequency spectrum of the emission as a cross-power correlation from the two emission waveforms. The straight band across the box is the associated noise spectrum and is the difference between the two waveform recordings. The left middle “Response Waveform” (QuickScreen) box displays the two overlapped emission waveforms (A and B) as a time spectrum. In the middle right “Power Analysis” box, the first solid line represents the frequency response of the stimuli, which was flat, as desired, across the frequency range from 0 to 4 kHz. The middle shaded waveform displays the frequency spectrum of the TEOAE response or the echo rising above the noise floor, and the lower shaded waveform displays the noise floor. The far right panel lists the stimulus and response parameters numerically. The top subsection, Noise, indicates that the average noise level in the external ear canal during TEOAE measurement was low (32.9 dB) and that 96% of the 260 stimuli were presented when the noise was lower than the rejection level of 47.3 dB. The middle subsection, Response, displays high overall waveform reproducibility of 96% as well as high reproducibility values (represented in %) across the individual octave bands from 1 to 4 kHz and a high signal-to-noise-ratio (SNR) for the TEOAEs (represented in dB) versus the noise floor. These results indicate that TEOAEs were present from 1 to 4 kHz based on the response parameters of adults with normal hearing sensitivity. Similar results were obtained for the left ear (lower panel), indicating the presence of TEOAEs from the 1 to 4 kHz frequency region with an overall waveform reproducibility of 98%. For a detailed analysis of OAE stimulus and response parameters, please refer to Hall (2000).
Doerfler-Stewart test (Doerfler & Stewart, 1946), which is performed by obtaining an SRT accompanied by background noise, with the intensity of the background noise increased systematically. The delayed auditory feedback (DAF) test is another behavioral test used to diagnose NOHL. It is based on the phenomenon that audible acoustic signals fed back to the listener with a delay of approximately 200 ms disrupt ongoing listening behavior that manifests as a significant change in vocal behavior (Ruhm & Cooper, 1964). Another behavioral assessment measure for NOHL is the Lombard test, which is based on the observation that in the presence of background noise, listeners will reflexively increase the intensity of their speech in order to be heard (Shoup & Roeser, 2000). Most of these tests were useful because they could identify the NOHL by disrupting or distorting the internal loudness anchor used by individuals with NOHL to decide which stimulus level they want to respond to (Gelfand & Silman, 1985, 1993; Martin et al., 2001). Now, however, with the exception of the Stenger test, these tests are almost obsolete because although they identified an NOHL, they failed to quantify its degree.

JD’s lack of behavioral responses to tonal stimuli was inconsistent with the results of the physiologic tests performed; the tonal MEMR levels were within normal limits with ipsilateral and contralateral stimulation, TEOAEs were present bilaterally, and ABR waveforms showed absolute and interwave latencies within normal limits at 85 dB nHL. An ABR latency-intensity function would have been beneficial in determining approximate hearing sensitivity levels. The presence of TEOAEs, however, argues strongly and objectively for normal cochlear integrity, at least up to the level of the outer hair cells. Although the actual hearing sensitivity was not established, and it is possible that JD may have had an organic peripheral hearing loss, the presence of TEOAEs (with the exception of individuals with auditory neuropathy) is consistent with a peripheral hearing sensitivity that was probably no worse than a mild sensorineural hearing loss (30–40 dB HL) for the stimulus frequencies (Hurley & Musiek, 1994; Lonsbury-Martin, Cutler, & Martin, 1991; Lonsbury-Martin & Martin, 2001; Stover & Norton, 1993). The audiologic testing performed at the university clinic

| Table 2. Results of the auditory brainstem response test with 85 dB nHL click stimulus with rarefaction polarity. |
|---|---|---|---|---|---|
| Ear | I | III | V | I–III | III–V |
| Right | | | | | |
| Absolute latency (ms) | 1.88 | 3.60 | 5.08 | 1.72 | 1.48 | 3.20 |
| Interwave latency (ms) | +.15 | +.17 | +.15 | | | |
| Amplitude (µV) | | | | | | |
| Left | | | | | |
| Absolute latency (ms) | 1.72 | 3.76 | 5.32 | 2.04 | 1.56 | 3.60 |
| Interwave latency (ms) | +.12 | +.14 | +.19 | | | |
| Amplitude (µV) | | | | | | |

Note. Results with the condensation polarity were similar and, therefore, were not presented in order to maintain clarity of the data.

| Table 3. Overview of the bilateral behavioral and diagnostic tests performed. |
|---|---|---|
| Test procedure | Stimuli used | Results |
| Tympanometry | 226 Hz Y | Normal |
| Tonal middle ear muscle reflexes (MEMRs) | 5000–2000 Hz with ipsilateral and contralateral stimulation | Within normal limits |
| Acoustic reflex decay | 500 and 1000 Hz with contralateral stimulation | Negative |
| Speech recognition threshold (SRT) | Spondee words presented at levels up to output limits of the audiometer | No response |
| Pure-tone air conduction | 1000 and 2000 Hz up to 120 dB HL | No response |
| Otoacoustic emissions (OAEs) | Click-evoked TEOAEs from 1000–4000 Hz | Present |
| Auditory brainstem response (ABR) | 85 dB nHL click stimulus with rarefaction and condensation polarity | Absolute and interwave latencies within normal limits |
did not rule out a neurological disorder beyond the auditory brainstem. The presence of such a condition, however, was unlikely given that no additional symptoms had been reported or documented since 1992. In addition, JD appeared to have no difficulty hearing or understanding speech even without visual cues. JD's psychosocial profile was consistent with that cited in the literature for individuals most likely to present with an NOHL. Further, the preliminary diagnosis of an adjustment disorder was more consistent with a primary psychological disorder rather than with an auditory disorder.

Although the diagnosis of NOHL may not be difficult for most clinicians, the challenge lies in determining the actual hearing sensitivity and confronting the client with that information in a manner that is not insulting and that allows for a graceful retreat on the part of the client. The NOHL diagnosis becomes more complicated when the purported hearing loss is manifested at an unconscious level, especially if clinicians are not experienced or trained to anticipate the existence of such a condition. In the absence of an appropriate diagnosis, individuals with NOHL may even be referred as candidates for a cochlear implant, as one hearing aid dispenser had recommended for JD, with the diagnosis consuming valuable time and expense (Spraggs et al., 1994).

The diagnosis of NOHL is often an exclusionary one made after other medical causes of the hearing loss have been ruled out. In the case of psychological conditions such as adjustment and conversion disorders, it is the absence of expected medical and laboratory findings that suggest and support the diagnosis (APA, 1994b). Identification of recent psychosocial stressors also can aid in the diagnosis. During the course of the evaluation, for example, JD had mentioned several times to the clinician her concerns regarding the whereabouts of her daughter and granddaughter, which may have been the current psychosocial stressor. Referral source, a good case history, and behavioral observations can be useful in raising the index of suspicion for an NOHL.

When responses to behavioral audiometric assessments are not reliable, or when the results of the different components of the basic assessment battery are not in agreement, then physiologic tests must be performed. Tonal MEMRs are an effective nonbehavioral tool for identifying or substantiating the presence of an NOHL. Part of the desirability of using tonal MEMRs comes from their routine clinical nature, compared to other physiologic measures such as cortical evoked potentials, which may address a wider range of hearing disorders but are rarely used in routine clinical practice because of well-established practical limitations (Gelfand, 1994). Several investigators have found that excellent agreement exists between the results of MEMR levels, reflex decay measures, and the ABR in distinguishing cochlear and retrocochlear site of lesion, and they serve as a reliable cross-check (Hayes & Jerger, 1981). When all three measures appear normal, then the presence of a pathology, at least up to the level of the auditory brainstem, would be highly unlikely. Gelfand (1994) reported that although the MEMR is an effective nonbehavioral tool for detection of an NOHL, it cannot identify a functional component if the volunteered thresholds are ≥60 dB HL because the MEMR would normally be present with a cochlear hearing loss of that magnitude. Even with this limitation, immittance measures should be performed initially for all routine audiologic assessments because they can discourage pseudohypacusis (Martin, 2002) and alert the clinician to the possible presence of an NOHL.

OAEs are another physiologic measure that can aid in the diagnosis of an NOHL. Equipment to determine OAEs is now available in most audiology clinics and can be used to provide additional valuable information regarding the status of the auditory system. The relationship of OAEs to other physiologic measures of auditory assessment, such as MEMR and ABR, substantiates the value of this procedure for the diagnosis of an NOHL (Kemp, Ryan, & Bray, 1990; Musiek, Bornstein, & Rintelmann, 1995; Robinette, 1992). The presence of emissions obtained with volunteered thresholds greater than approximately 30–40 dB HL suggests an NOHL or a neural basis for the hearing loss because evidence of OAEs is typically incompatible with a sensory hearing loss exceeding a mild loss range, if the loss is secondary to outer hair cell dysfunction (Musiek et al., 1995). Limitations of TEOAEs include prevention of recording of responses because of attenuative influences of the external and middle ear on reverse energy transmission and their insensitivity to disorders affecting inner hair cells or higher levels of the auditory pathway (Norton et al., 2000). The TEOAEs’ amplitude appears to decrease with increasing age: Adults have an overall lower TEOAE amplitude than children, and children have a lower amplitude than infants (Glattke et al., 1995; Prieve, Fitzgerald, & Schulte, 1997).

Auditory evoked potentials also are a useful tool for the diagnosis of an NOHL. The ABR test can be used as a cross-check to volunteered responses as well as to rule out a neurologic basis of the hearing loss, which may not be possible through behavioral or OAE testing alone. Abnormal ABR findings indicate the possibility of a pathological condition of the auditory nerve and brainstem pathways but not the type of the pathology. For example, the ABR abnormality cannot diagnose a tumor versus a vascular lesion (Arnold, 2000). Middle and long latency auditory evoked responses can be beneficial in effectively ruling out neurologic conditions beyond the level of the brainstem before establishing a diagnosis of NOHL. Middle latency auditory evoked potentials (MLAEPs) consist of a series of auditory evoked responses that occur between 10 and 80 ms following the onset of an acoustic stimulus. By definition, MLAEPs follow the ABR and precede the long latency auditory evoked potentials (Kraus, McGee, & Stein, 1994). Clinical uses of MLAEPs include the estimation of frequency-specific hearing thresholds, especially in the lower frequencies (Musiek & Geurink, 1981; Scherg & Volk, 1983); assessment of auditory pathway function, including ruling out a central pathology when an NOHL is suspected and localization of auditory pathway lesions (Buchwald, Erwin, Read, Van Lancker, & Cummings, 1989; Jacobson & Grayson, 1988; Kileny, Paccioretti, & Wilson, 1987; Kraus & McGee, 1988); and assessment of cochlear implant function (Kileny & Kemink, 1987; Miyamoto, 1986). The MLAEPs are affected by low-frequency muscle
artifacts, specifically postauricular muscle (PAM) activity (Kileny, 1984); sleep stages, especially in children (Kraus, McGee, & Comperatore, 1989); sedation (Schwender et al., 1996); and neuropsychiatric history (Baker et al., 1990; Siegel, Waldo, Mizner, Adler, & Freedman, 1984). The utility of MLAEPs as a test of central auditory function is limited by an incomplete understanding of the MLAEP generating system (Kraus et al., 1994), the considerable variation in normal waveform morphology, and the pattern of abnormalities that may vary with differing recording conditions (Arehole, Augustine, & Simhadri, 1995).

The long latency auditory evoked potentials (LAEPs) are recorded in a time period from approximately 50 to 250 ms after acoustic stimulation by a transient sound such as a click, toneburst, or speech element at a relatively slow rate (approximately 1 to 2 stimuli per second) and include both exogenous and endogenous potentials (Scherg, Vajsar, & Picton, 1989; Velasco, Eelasco, & Velasco, 1989). Exogenous auditory responses are those responses that are generated primarily by the physical characteristics of the stimulus; endogenous auditory responses are those that are produced after internal processing of the stimulus has occurred. The spectral power of the typical LAEP response is between 1 and 15 Hz and is most evident at approximately 5 Hz. Unlike the ABR waveforms, amplitude measures are always obtained for cortical evoked potentials. Compared to earlier evoked potential responses, the amplitude of the LAEP response is large at approximately 3 µvolt and sometimes even larger (Hyde, 1994). The precise location of the LAEP neural generators is unclear, but they probably overlap and include the areas in and around the primary auditory cortices (Vaughan & Ritter, 1970), the auditory association cortex in the superior temporal gyrus (Wolpaw & Penry, 1975), and the frontal motor and/or premotor cortex under the influence of the reticular formation and the ventral lateral nucleus of the thalamus (Naatanen & Picton, 1987).

LAEP responses demonstrate complexity of interaction among stimulus conditions (Stapells, 2002), attention (Naatanen & Picton, 1987), alertness and sleep (Osterhammel, Davis, Wier, & Hirsh, 1973; Picton & Hillyard, 1974), drugs (Hall, 1992a), and maturation (Kurtzberg, 1989; Sharma, Kraus, McGee, & Nicol, 1997). The LAEP can be a useful clinical tool in the estimation of hearing sensitivity of individuals when a nonbehavioral, frequency-specific, and sensitive measure is needed, such as for medicolegal and compensation assessments and the evaluation of a suspected NOHL (Hyde, 1994). Other potential clinical applications include nonbehavioral measurement of frequency and temporal discrimination, localization, speech perception, and the diagnosis of auditory processing disorders and attention deficit hyperactivity disorders (Hyde, 1994). Clinical applications of the LAEPs are, however, limited by considerable inter- and intrasubject variability, state of participants, drugs, age, and the lack of knowledge regarding precise location of neural generators (McPherson & Ballachandra, 2000). These responses are, however, still considered important and promising in assessing the functioning of the higher levels of the central auditory system.

Another cortical auditory evoked potential is the P300 response, which is essentially a component within an extended LAEP time frame. It is an endogenous cognitive auditory response that is nonsensory specific, that is, it occurs in response to both unimodality and multimodality sensory stimulation (McPherson & Ballachandra, 2000). It is recorded under special stimulus conditions, the simplest of these conditions being an infrequent or rare stimulus presented randomly within a series of frequent, predictable stimuli. This response is sometimes referred to as the P300 because it is often observed in the 300 ms region, although it has been recorded in listeners with normal hearing sensitivity as early as 250 ms and as late as 400 ms (Hall, 1992c).

In the literature, the amplitude and latency of the P300 has been reported as abnormal in several types of neuropsychiatric and behavioral disorders. Clinical applications of the P300 are related to the task-relevant nature of this response, and, therefore, it has potential for studying higher level auditory processing skills (McPherson & Ballachandra, 2000). It has been used successfully in the study and assessment of children with attention deficit hyperactivity disorders (Salamat & McPherson, 1999), cochlear implant users as a means of determining cognition and discrimination (Kileny, 1991), cerebral lesions in adults (Hurley & Musek, 1997), auditory deprivation (Hutchinson & McGill, 1997), and dementia and Alzheimer’s disease (Polich, 1989; St. Clair, Blackburn, Blackwood, & Tyrer, 1988). Currently, only a small percentage of clinicians are routinely using MLAEPs and LAEPs (Martin, Champlin, & Chambers, 1998), probably because of the available equipment, experience of clinicians, and third-party reimbursement issues.

Physiologic assessment measures, when used together with behavioral observation and behavioral assessment, can improve the diagnostic criterion of NOHL. Unfortunately, in JD’s case, no such assessments had been performed despite repeated audiologic evaluations over several years, and, thus, the possible underlying psychological disorder was not diagnosed. Early identification of the NOHL may have led to appropriate medical intervention that could have improved her quality of life. Audiologists are not responsible for determining the underlying reason for an NOHL, nor should they be judgmental of those who present with it. It is, however, the responsibility of the audiologist to diagnose a nonorganic component and to determine the extent of a true organic hearing loss that may coexist with the NOHL. It also is important for audiologists to be informed that psychogenic hearing loss does exist and that clinicians have an obligation to provide clinical services that will benefit clients with this disorder, including appropriate and timely medical referral when indicated.

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