Transient Evoked and Distortion Product Otoacoustic Emissions in Traumatic Brain Injury

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Abstract

Behavioral audiometry may be of limited value for those patients who have incurred significant brain injury and remain unresponsive to auditory stimuli. Even the more "objective" tests such as auditory brainstem response (ABR) and acoustic reflex testing may be inadequate tools to assess peripheral auditory function when significant brainstem injury has occurred. Determination of peripheral hearing becomes important because rehabilitation strategies are anchored to the knowledge of sensory function prior to implementing appropriate plans of intervention. We present the usefulness of otoacoustic emission (OAE) testing to further evaluate the cochlear function in a patient with traumatic brain injury who was subsequently inconsistent in response to sound.

Key Words: Auditory brainstem responses (ABRs), distortion product otoacoustic emissions (DPOAEs), transient evoked otoacoustic emissions (TEOAEs), traumatic brain injury

While normal findings during auditory brainstem response (ABR) and acoustic reflex testing are dependent upon an intact peripheral mechanism and auditory brain stem, otoacoustic emissions (OAEs) are largely the result of preneural cochlear activity of the outer hair cells (OHCs) of the organ of Corti (Dallos, 1988). OAEs are thought to represent an active micromechanical process of the OHCs that results in an acoustic signal that can be recorded in the external auditory canal (Kemp, 1978; Brownell, 1990). Several investigators have demonstrated that OAEs are present in essentially all ears with normal middle ear and cochlear function (Bonfils et al, 1988; Probst et al, 1991; Robinette, 1992a). If the middle ear mechanism is functioning normally, then the absence of OAEs is usually associated with mild or greater cochlear (OHC function) hearing loss (Probst et al, 1987). However, some normally hearing individuals have demonstrated minimal or no OAEs for the frequency region at 3000 and 4000 Hz (Robinette, 1992a).

Transient evoked otoacoustic emissions (TEOAEs) are elicited with brief acoustic stimuli. They are most frequently measured between 500 and 4000 Hz (Elberling et al, 1985; Probst et al, 1986). The latency of TEOAEs is typically between 5 and 20 msec and varies inversely with the frequency of the OAE component (Kemp, 1978). The frequency content of a transient evoked emission is dependent upon the spectrum of the evoking stimulus. The amplitude grows nonlinearly as a function of stimulus level (Norton and Stover, 1994).

Distortion product otoacoustic emissions (DPOAEs) represent the acoustic energy elicited by the presentation of two long-lasting tonal stimuli (f_1 and f_2) to the ear with response frequencies predicted on the basis of the intermodulation distortion frequencies of the input stimuli. When f_1 and f_2 are presented to the ear, combination tones result due to interference or amplitude distortion. DPOAEs occur in the range of 500 to 8000 Hz and are dependent upon both the sound pressure levels and frequencies of f_1 and f_2 (Martin et al, 1990).

We present the usefulness of TEOAEs and DPOAEs to further evaluate cochlear function...
in a patient with traumatic brain injury (TBI) who was subsequently inconsistent in response to sound. A review of the patient's cognitive and language function will also be presented to highlight the inconsistency in auditory performance.

CASE REPORT

Medical History

The patient was a young adult female who suffered a severe closed head injury as the result of being an unrestrained passenger in a motor vehicle accident. At the time of the initial neurologic assessment, she was unresponsive, other than the presence of her heart beat and respiration. She was deeply comatose with nonreacting pupils and no response to deep pain. She later was felt to have a significant amount of hypoxic encephalopathy and showed intermittent seizure activity. Follow-up computed tomography scans showed a small hemorrhage in the right perimisencephalic cistern in addition to left frontal cerebral atrophy. The medical examination revealed no evidence of temporal bone fracture or tympanic membrane abnormalities.

Speech, Language, and Cognition

Overall, the patient displayed severe cognitive and communicative deficits 1 year post TBI. Auditory comprehension was severely impaired. Her ability to follow one-step, one-element commands and answer personally related yes-no questions accurately was inconsistent. Her responses were limited to shaking her head "no" and shrugging her shoulders, with occasional vocalizations noted. She was unable to follow simple conversation.

Verbal language formulation was severely impaired due to poor initiation and decreased intelligibility. Spontaneous verbalizations were noted on occasion. However, speech output was aphonie and difficult to interpret due to imprecise articulation. Reading comprehension was adequate at the simple sentence level and was the primary mode of communication. Written communication was essentially nonfunctional due to severe ataxia of the upper extremities. Cognitively, she was clearly in a state of post-traumatic amnesia and demonstrated poor sustained attention and diligence with therapeutic tasks.

Audiologic History

The patient was seen at Mayo Clinic Scottsdale for a second opinion regarding hearing. Initial behavioral audiometric testing conducted at another audiology center suggested a moderate-to-severe hearing loss in the right ear and a severe-to-profound hearing loss in the left ear but was inconclusive. An FM auditory trainer was tried but no change in her response was observable by her family or rehabilitation team. She appeared to reject the FM system by being upset when it was used. She frequently attempted to shake the headset from her head.

Audiologic Test Results

At the time of her visit, she was nonverbal but did show consistent yes/no responses to written material. In an attempt to estimate hearing sensitivity for speech, three-choice spondees were presented in 5-dB increments from 60 to 85 dB HL with only one correct response noted at 75 dB HL. She indicated that she did not comprehend at any other presentation levels. There were no other responses observable either by pure-tone or informal speech testing. Tympanometry using a Grason-Stadler GSI-33 middle ear analyzer indicated normal Type A tympanograms bilaterally with middle ear pressures of -10 daPa (peak compliance of .6 mL) and -5 daPa (peak compliance of .5 mL). Ipsilateral and contralateral acoustic reflexes were absent bilaterally.

ABR and OAE testing were conducted to further evaluate peripheral auditory function. ABRs were measured using a Nicolet Compact Four evoked potential unit. Stimuli were 100 μsec square wave pulses delivered through TIPProbe 300 ohm insert earphones (0.9 msec correction factor related to tube length) at a rate of 11.7 refreshation clicks per second. Filter settings were 150 Hz high pass and 1500 Hz low pass. At least 1000 sweeps were summed and responses were replicated at all intensity levels tested.

The results of ABR conducted at 90 dB nHL (118 dB peSPL) indicated abnormal conduction of auditory stimuli within the auditory brainstem pathways bilaterally (Fig. 1). The left interwave interval between wave I and wave V was 5.82 msec. This value exceeded 2 standard deviations of the mean for age (mean = 3.99 msec, SD = 0.22 msec). No identifiable ABR wave components could be established beyond wave III for the right ear. Further testing at lower intensities indicated minimal ABR responses at 55 dB
nHL and 75 dB nHL for the right and left ears, respectively. Accurate peripheral threshold measurements using ABR were precluded by the central abnormalities bilaterally, that is, the low amplitudes or absent waves I, III, and/or V bilaterally could not be used as indicators of ABR threshold.

TEOAEs were measured using the Otoacoustic Analyzer Transient Evoked ILO88 module and software (Kemp et al., 1986, 1990) controlled by an IBM PC. All OAE testing was conducted in a sound-treated double-walled test room. Stimuli were presented in the default mode (differential nonlinear test paradigm). Each run consisted of 265 blocks, 80 μsec rectangular pulses presented at 50/sec at 82 dB peSPL. The pulses were presented through a transducer in a probe fitted to the patient’s ear canal with an immittance probe tip. In an effort to improve data quality, several repetitions of each ear were conducted. This was necessary because of intermittent head movement of the patient due to her impaired motor control and the associated high level of recorded noise. The canal noise floor was 30.5 dB SPL for the right ear and 31.8 dB SPL for the left ear. The waveform reproducibility was 78 percent for the right ear and 66 percent for the left ear.

TEOAEs were present in both ears but relatively low in response amplitude (right = 2.0 dB SPL; left = −0.6 dB SPL). The presence of TEOAEs bilaterally indicated normal to near-normal cochlear function for the emission frequencies obtained (Fig. 2).

DPOAEs were measured using the Otodynamics Otoacoustic Analyzer ILO88 XP module and software. DPOAEs were measured at levels of 70 dB SPL for \( f_1 \) and \( f_2 \) with an \( f_2:f_1 \) ratio of 1.22. The DP Gram software provided a plot of the intensity of the 2\( f_1 - f_2 \) distortion product (Fig. 3). Averaging of the DP data was undertaken in the time domain. The noise data were averaged in the same way as the DP signal. The present data suggested the possibility of cochlear hearing loss at 2000 Hz, but DPOAE responses were otherwise present bilaterally from 0.1 to 6 kHz.

**DISCUSSION**

There is a gathering body of evidence revealing abnormal ABRs and present otoacoustic emissions (Lutman et al., 1989; Prieve et al., 1991; Robinette and Facer, 1991; Robinette, 1992b). Since absent or abnormal ABRs may reflect dysfunction at the level of the brain stem.
Figure 3  DPOAEs for right and left ears were measured at levels of 70 dB SPL. The dark and light shaded areas represent 1 and 2 standard deviations respectively above the mean level of the noise.

and obscure the assessment of cochlear function, OAEs hold promise in providing valuable additional information in the evaluation of patients who have brainstem injury and remain unresponsive during behavioral testing. The presence of otoacoustic emissions in the absence of valid ABR thresholds because of central abnormalities provides exclusive evidence for predominantly retrocochlear dysfunction. In most cases of conventional ABR measurements, the amplitude of wave I is insufficiently robust to be evident at ABR wave V thresholds (Hyde and Blair, 1981). In severe high-frequency sensorineural hearing loss, the shallow canal extratympanic electrodes may not be sufficient to resolve wave I (Ruth, 1994). Although tympanic or transtympanic electrocochleography can provide equivalent thresholds for wave I and wave V (Ferraro and Ferguson, 1989), it was not possible in this case because of the patient’s poor head control and associated unpredictable movement. The OAEs were conducted quickly and the measurement successfully provided an indication of normal to near-normal cochlear function for the emission frequencies assessed.

One option used to enhance auditory awareness and sound recognition was an assistive listening device. Although the initially recommended FM system had the advantage of improving the signal-to-noise ratio, the output and gain may have exceeded levels of comfort for this patient. The parents commented that they increased the volume control because of their daughter’s lack of auditory awareness and responsiveness at lower gain settings. Subsequently, she rejected the FM system by shaking her head to remove the headset. This underscores the importance of taking care to limit the output of any amplification device when auditory thresholds remain uncertain. The advantages of FM systems can be negated with overamplification, and, ultimately, rejection of the system can occur. Subsequently, the parents purchased a personal FM system (Comtek A72 with option D and headphones) with a maximum output limited to 100 dB SPL. The parents and speech pathologist, however, continued to see little improvement in auditory responsiveness. One year later, the OAEs remain unchanged.

SUMMARY

Despite the significant contribution of ABR and acoustic reflex testing in assessing peripheral auditory function in nonresponsive patients, these tests are largely inadequate in establishing normal cochlear function when brainstem dysfunction precludes an accurate assessment of threshold estimation. The value of OAEs in these cases adds a new dimension in the audiologist’s ability to separate preneural cochlear function from abnormalities at the level of the auditory nerve and brain stem.

Acknowledgment. This paper was presented at the 1993 American Speech-Language-Hearing Association Annual Convention, Anaheim, California.

REFERENCES


