Clinical Forum

Otoacoustic Emissions, Audiometric Sensitivity Loss, and Speech Understanding:
A Case Study

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Abstract
The clinical measurement of otoacoustic emissions can assist in differentiating between peripheral and central explanations for deficits in speech understanding. We present audiometric and distortion-product emission data in a case with sensorineural hearing loss and a deficit in speech understanding. The presence of evoked emissions argues against attributing the speech audiometric loss to cochlear defect.

Key Words: Otoacoustic emissions, sensorineural hearing loss, speech understanding, peripheral hearing loss, central hearing loss, central auditory processing disorders

The interpretation of speech audiometric deficits is often limited by inability to separate the effects of peripheral sensitivity loss from the effects of central processing disorder (Helfer and Wilber, 1990; Humes and Roberts, 1990; Humes and Christopherson, 1991). If a patient has both a speech audiometric deficit and depressed audiometric sensitivity, especially in the frequency range above 1000 Hz, it may be difficult to determine how much of the speech understanding deficit should be attributed to central processing disorder versus how much should be attributed to peripheral (i.e., cochlear) hearing loss. Even in the case of patients with normal audiograms one cannot rule out the possibility of a subtle cochlear defect, sufficient to impact measures of speech understanding, but not sufficient to cause audiometric sensitivity loss for pure-tone signals (Bredberg, 1968). As long as the pure-tone audiogram served as the sole index of cochlear normality there was no easy solution to this problem.

With the advent of clinically viable methods for measuring otoacoustic emissions, especially distortion-product emissions, however, the distortion-product audiogram provides invaluable complimentary information about cochlear status. It is now widely accepted that, with only rare exceptions (e.g., Prieve et al, 1991), the presence of otoacoustic emissions at normal levels indicates normal outer-hair-cell function (Lonsbury-Martin and Martin, 1990; Norton and Widen, 1990). This places a powerful interpretive tool at our disposal. If it can be shown that a patient with a speech audiometric deficit has normal otoacoustic emissions, then it cannot be easily argued that the speech understanding deficit is attributable to cochlear deficit. This will be true irrespective of the level of the pure-tone audiogram.

In the present paper we report findings in such a case. The patient had speech audiometric deficits, depressed behavioral audiograms, but normal distortion-product emissions. We argue that the presence of emissions supports a central rather than a peripheral interpretation of the deficit in speech understanding.

METHOD

Distortion-product emissions were measured by means of commercially available system consisting of a microcomputer (Dynova 286), an Ariel board, an Etymotic ER10B probe...
microphone system, two Etymotic ER2 tube-
phones, and software developed by Jont Allen.
For pairs of probe tones, $f_1$ and $f_2$, the amplitude,
in dB SPL, of the cubic distortion product, $2f_1 - f_2$, was measured at the frequency $f_2$. Over the range from 1000 to 8000 Hz, 14 pairs of probe tones at approximately equally spaced logarith-
mic units of frequency were presented. The intensity levels, $L_1$ and $L_2$, corresponding to the probe-tone frequencies $f_1$ and $f_2$, were always 65
dB SPL. Thus the $L_1/L_2$ ratio was always 1.0. The $f_2/f_1$ frequency ratio was always 1.2. Normal values for the noise floor in this report are based on the distribution of test results in ten ears of five young adults with normal hearing and no hearing complaints. In subsequent figures the dashed lines denoting the normal limits for the noise floor encompass 90 percent of the ex-
pected normal range ($\pm 1.65$ standard devia-
tions).

Conventional audiometric data were ob-
tained during the course of routine clinical audiometric assessment. For pure-tone audiograms we used the Virtual 320 system, controlled by a MacIntosh II microcomputer. For speech audiometric measures tape-recorded phoneti-
cally balanced (PB) word lists and synthetic sentence identification (SSI) materials were delivered through the Virtual system. Immit-
tance measures were obtained with an Amplaid 720 system.

**CASE REPORT**

Patient LS, a 29-year-old woman, had shown symptoms of multiple sclerosis (MS) for approximately 10 years. A diagnosis of definite MS was made 3 years prior to the present audiologic evaluation. Magnetic resonance imaging (MRI) of the brain, with and without contrast, revealed white matter lesions near the lateral ventricles, an extensive region of abnormality in the midline involving the entire superior margin of the corpus callosum, and a lesion in the deep left temporal white matter. She was referred to us with the complaint of progressive hearing loss in the right ear. Figure 1 summarizes the results of the conventional audiometric assessment. The left ear showed a mild low-frequency sensitivity loss, but was well within normal limits over the frequency range from 2000 to 8000 Hz. At no frequency on the left ear did the pure-tone threshold level exceed 20 dB HL. The score for PB words, presented at 80 dB HL, was 100 percent, and the maximum score for SSI at 0 dB MCR was 90 percent.

Results from the right ear, however, showed significant deficits. The pure-tone audiogram showed only a mild, relatively flat, sensitivity loss. The poorest threshold, at 3000 Hz, was 25 dB HL. In spite of this relatively mild sensitivity loss, however, speech understanding showed a marked deficit. The maximum PB score was only 60 percent, and the maximum SSI score was only 40 percent. In addition, there was significant rollover of the performance versus intensity (PI) function for SSI. The score de-
creased from 40 percent at 60 dB HL to 10 percent at 80 dB HL. Scores on the Dichotic Sentence Identification Test (DSI) in the fo-
cused attention mode were 100 percent on the left ear and 20 percent on the right ear.

Middle-ear function, as assessed by tympan-
ometry, was normal bilaterally. With signals to the left ear, acoustic reflex thresholds, in both the crossed and uncrossed modes, varied from 80 to 95 dB HL over the frequency range from 500 to 4000 Hz. With signal to the right ear, however, neither crossed nor uncrossed acoustic reflexes could be elicited at any test fre-
quency at intensity levels up to 110 dB HL.
The auditory brainstem response (ABR) was recorded from a conventional four-electrode montage. The active electrode was at the vertex (Cz), reference electrodes at the earlobes, and the ground electrode on the forehead. The ABR was elicited by 100-microsecond pulses presented at the rate of 21.1 per second. The EEG was filtered from 150 to 3000 Hz. A total of 2,048 individual sweeps were averaged to define each ABR waveform.

Figure 2 shows that the ABR was within normal limits for clicks delivered to the left ear, but for right ear stimulation there were no repeatable waves beyond wave I.

The poor speech audiometric scores for the right ear are, of course, well outside the limits to be expected from patients with cochlear deficits at the 20 to 25 dB level (Yellin et al, 1989). Although extremely unlikely, one cannot rule out the possibility that the right ear's sensitivity loss might reflect a subclinical cochlear defect sufficient to account for the reduction in PB and SSI scores. If this were the case, however, we would expect that otoacoustic emissions would be reduced or absent from the right ear. Figure 3 shows that this was not the case. Across the range from 1000 to 8000 Hz the distortion-product emissions were robust from both ears. We cannot, therefore, account for the poor speech audiometric scores from this patient's right ear on the basis of a right cochlear defect in spite of the slight apparent reduction in pure-tone sensitivity on the right side. Clearly, a processing deficit at a more central site in the auditory system must be invoked.

**COMMENT**

This case (LS) illustrates how otoacoustic emissions can be useful in ruling out subtle cochlear defect as an explanation for deficits in speech understanding. There was a marked asymmetry in speech audiometric scores along with a slight asymmetry in pure-tone sensitivity levels. The presence of distortion-product otoacoustic emissions from both ears argues for a more central locus of the speech processing disorder.

Of course, this argument rests on the assumption that, in persons with sensorineural hearing loss, outer hair cell loss, evoked otoacoustic emissions, and audiometric threshold levels are highly correlated measures. It is possible, however, that the combination of substantial sensitivity loss and good otoacoustic emissions is the result of selective loss of inner hair cell function, with preservation of outer hair cells and the otoacoustic emissions generated by them. This was, indeed, the mechanism suggested by Prieve et al (1991) to explain their unusual results in a subject with severe sensorineural loss and an apparently normal tone-burst evoked emission. This is not, however, an attractive hypothesis to explain results in the present case. Although wave 1 of the right-ear ABR were abnormal, there was a robust wave 1 at levels of 60 and 80 dB nHL.

**Figure 2** Auditory brainstem responses for the 29-year-old woman with multiple sclerosis.

**Figure 3** Distortion-product emissions are robust from both ears, in spite of pure-tone and speech-audiometric deficits on the right side.
We conclude, therefore, that the asymmetric speech audiometric deficit reflects central rather than peripheral asymmetry in auditory processing.

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REFERENCES


