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Clinical Applications of Otoacoustic Emissions

Tutorial

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Editor's Note: *Otoacoustic emissions have received considerable attention in the contemporary audiology literature because of recent advances in auditory psychophysiology. New knowledge regarding cochlear physiology in particular suggests that measures of otoacoustic emissions may hold promise in diagnostic audiology. We are pleased that Drs. Lonsbury-Martin, Whitehead, and Martin have prepared this tutorial paper to discuss the bases and potential clinical applications of these measures.*

On the basis of recent advances in auditory physiology, new tests of cochlear function have been developed using measures of otoacoustic emissions. In the present report, the clinical potential for each of the four basic emission types is examined. In addition, the practical advantages of examining the ear with two specific types of evoked emissions, transiently evoked and distortion-product otoacoustic emissions, are reviewed in detail. Finally, the future role of tests of otoacoustic emissions in the diagnosis of hearing impairment is discussed. The current view is that evoked emissions hold promise as an essential part of the clinical examination of the auditory system.

KEY WORDS: spontaneous otoacoustic emissions, stimulus-frequency otoacoustic emissions, transiently evoked otoacoustic emissions, distortion-product otoacoustic emissions, outer hair cells, clinical applications

Sound stimulation of the ear sets up mechanical vibrations of cochlear structures. In the perception of sound, the fundamental role of cochlear inner hair cells is to transduce these mechanical disturbances into neurochemical events. The neurochemical processes, in turn, initiate a sequence of steps that gives rise to impulses from fibers of the auditory nerve. Recent studies have demonstrated that within the organ of Corti, an active mechanical process makes use of metabolic energy to create additional microvibrations that enhance the sound-induced motion of cochlear structures and increase the sensitivity and frequency selectivity of the ear (Davis, 1983; Johnstone, Patuzzi, & Yates, 1986; Sellick, Patuzzi, & Johnstone, 1982). Thus, the cochlea actively produces energy as a part of the normal hearing process. Some of this added energy propagates towards the base of the cochlea, to the stapes footplate, through the ossicles, and into the external ear canal, where it can be detected by a sensitive microphone (Kemp, 1978; Kemp, Bray, Alexander, & Brown, 1986; Wilson, 1980a). The sounds produced in this manner are called otoacoustic emissions (OAEs). The results of a considerable number of experimental and theoretical studies of OAEs, carried out since their discovery by Kemp (1978), indicate that emissions are produced as a normal by-product of the micromechanical actions of the "cochlear amplifier" (for reviews, see Brownell, 1990; Kemp, 1986; Wilson, 1984).

The "cochlear amplifier" is thought to be situated in the outer hair cells (OHCs), which have been shown in vitro to be motile in response to high frequencies of electrical stimulation (Brownell, Bader, Bertrand, & de Ribaupierre, 1985). Consistent

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with this notion are the results of animal experiments that have provided evidence that activation of the cochlear-efferent system, known to synapse preferentially on OHCs, modifies OAEs, thus indicating that emissions are generated by the OHC system (Guinan, 1986; Mountain, 1980; Siegel & Kim, 1982). The cochlea's mechanical amplifier appears particularly vulnerable to physiological and physical trauma, such as anoxia and exposure to ototoxins or loud noises (Anderson, 1980; Johnstone et al., 1986; Sellick et al., 1982). In actuality, many forms of hearing loss are caused by a deficiency in the action of the cochlear amplifier, resulting in a corresponding reduction in the mechanical vibrations transduced by the inner hair cells and, thus, a loss of hearing sensitivity. The results of a number of contemporary studies indicate that a reduction or loss of function of the cochlear OHCs is typically reflected as a reduction or absence of OAEs in the ear canal (Johnsen & Elberling, 1982; Kemp, 1978, 1982, 1988; Kemp & Brown, 1984; Lonsbury-Martin, Probst, Coats, & Martin, 1987; Zwicker, 1983a). Because hearing impairment and OAE responses are associated, and because the measurement of OAEs is both objective and noninvasive, OAE procedures represent a valuable technique in diagnostic audiology.

Our discussion on the usefulness of OAEs in clinical practice begins with defining and classifying these measures. The basic properties of each OAE type will then be described. Following a discussion of the suitability of the four types of OAEs for use in the detection of cochlear dysfunction, representative examples of the clinical application of evoked emissions will be presented. Finally, the results obtained using these techniques will be evaluated, and the future development of OAEs as a clinical tool will be discussed.

Classification of Otoacoustic Emissions

Otoacoustic emissions can be separated into two general categories: spontaneous and evoked emissions. Spontaneous OAEs (SOAEs) occur in the absence of any deliberate stimulation of the ear. They can be detected in about 50% of all ears with normal hearing (Dallmayr, 1985; Lonsbury-Martin, Harris, Hawkins, Stagner, & Martin, 1990a; Schloth, 1983; Whitehead, Baker, & Wilson, 1989; Zurek, 1981) by sealing a sensitive miniature microphone into the external ear canal. Evoked emissions occur in response to the presentation of acoustic stimuli to the ear. Consequently, a sound source must also be sealed into the ear canal to present the sounds necessary for eliciting evoked emissions. On the basis of the stimuli used to elicit them, evoked emissions can be usefully categorized into three different subtypes. Transiently evoked OAEs (TEOAEs) are elicited by an acoustic transient such as a click or toneburst; stimulus-frequency evoked OAEs (SFOAEs) are elicited by a single, continuous pure tone; and distortion-product OAEs (DPOAEs) are generated in response to two continuous pure tones, separated in frequency by a prescribed difference (in Hz). These three types of evoked OAEs are found in essentially all normally hearing ears. The categorization of the four forms of emissions according to stimulus type is briefly summarized in Table 1. In addition, a schematic drawing depicting the typical configuration of the devices commonly utilized to measure evoked emissions, along with the general features of the ear that are involved in the generation and propagation of OAEs, is shown as Figure 1.

Basic Properties of Otoacoustic Emissions

Spontaneous Otoacoustic Emissions

Spontaneous otoacoustic emissions (SOAEs) are typically detected, in the absence of deliberate sound stimulation, by spectral analysis of the amplified output of a miniature microphone sealed in the ear canal. In the resulting amplitude spectrum, SOAEs appear as narrow peaks above the noise floor. An example of three SOAEs in one ear is given in Figure 2A. Spontaneous emissions are low-level (i.e., usually <20 dB SPL) narrow-band sounds that are typically continuously present at one or more frequencies in about half of the ears of normally hearing persons (Dallmayr, 1985; Lonsbury-Martin et al., 1990a; Schloth, 1983; Whitehead et al., 1989; Zurek, 1981). The prevalence of SOAEs is similar in infants, children, and adults (Bargones & Burns, 1988; Strickland, Burns, & Tubis, 1985), but there is a strong gender difference in their occurrence, with approximately twice as many females demonstrating SOAEs as males (Bilger, Matthies, Hammel, & Demorest, 1990; Strickland et al., 1985; Whitehead et al., 1989).

Spontaneous emissions appear to be produced by the same active process that generates TEOAEs and SFOAEs (see below). Because of middle-ear conduction properties, only a portion of the evoked OAE energy escapes from the cochlea and is detected by the microphone in the ear canal (Kemp, 1979a). The remainder of the energy is reflected back into the cochlea by the stapes footplate, which represents an impedance mismatch in the propagation path for otoacoustic energy leaving the cochlea. It is hypothesized that this internally reflected energy acts as a stimulus in its own right, producing a secondary forward-travelling wave that restimulates the emission generator. If enough energy is reflected back into the cochlea, and the phase relation of the reflected energy and the generator output is appropriate, this reflection results in the continuous stimulation of the emission generator, thus leading to a sustained oscillation (Kemp, 1979b, 1981). In this schema SOAEs can be thought of as continuously self-stimulating evoked OAEs that can occur at those frequencies where the emission-generator output is

TABLE 1. Classes of otoacoustic emissions.

Otoacoustic emission	Notation	Stimulus	Prevalence (by ear)
Spontaneous	SOAE	None	~50%
Evoked:			
Transiently evoked	TEOAE	Click/toneburst	~100%
Stimulus-frequency	SFOAE	Continuous pure tone	~100%
Distortion-product	DPOAE	2 continuous pure tones	~100%

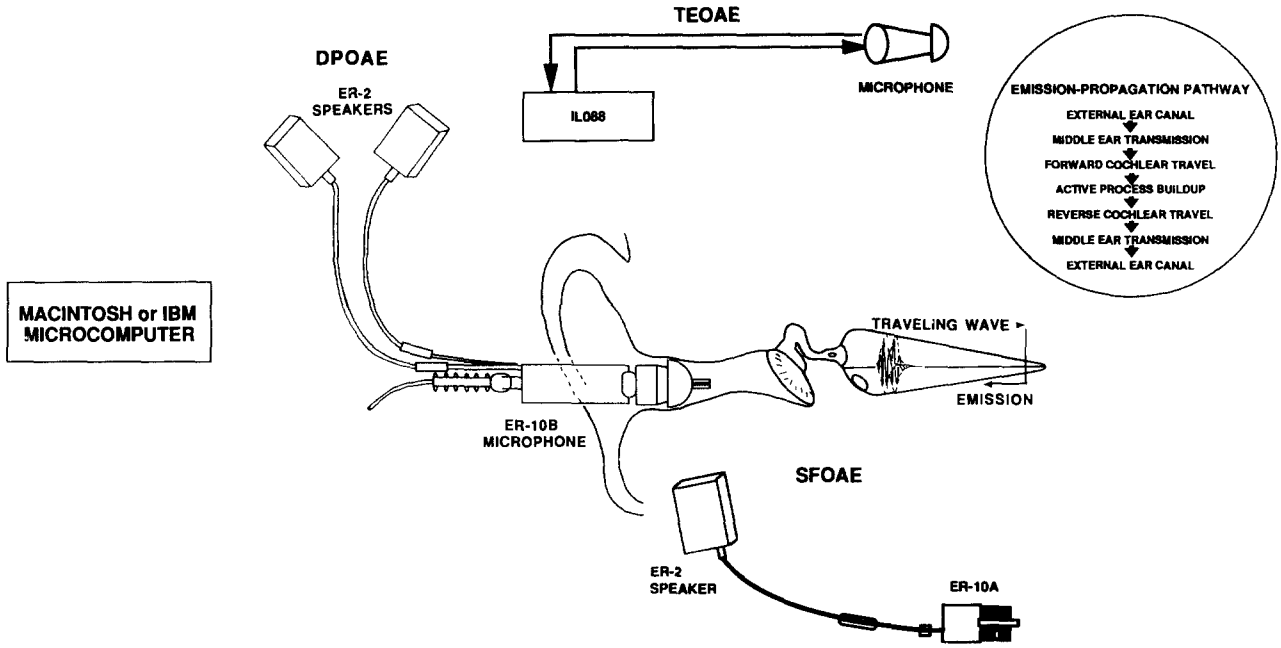

FIGURE 1. Schematic of various equipment and microphone/speculum components used to measure evoked OAEs. The microcomputers at the left support the current commercially available systems that control the devices and procedures for eliciting, detecting, and analyzing OAEs. In the center, a typical configuration of a speaker/sensor system for measuring bitonal-evoked DPOAEs is illustrated. This layout shows the acoustic speculum, containing the sound-delivery tubes and an often used microphone device (ER-10B) designed specifically to fit infant ears, in relation to the propagation pathway of the eliciting stimuli and returning emissions. Above, a commonly used TEOAE set-up based on the computer-controlled Otodynamic Analyzer (Otodynamics Ltd., ILO88) is shown, along with its microphone assembly which contains a miniature speaker to deliver the click or toneburst stimuli. Below, a commercially available microphone system designed for adult ears (ER-10A) is depicted in an arrangement that permits the measurement of tonal-evoked SFOAEs. The inset at the top right presents the details of the emission-propagation pathway.

positively fed back into its input. Consistent with this view is the observation that SOAEs are found only in frequency regions associated with a strong evoked response, where the feedback-dependent or loop gain of the reflected energy is sufficient to sustain the oscillation. The need for the loop-gain and phase relationship of the internal reflection to be appropriate may explain why SOAEs are present in only about half of all healthy ears, whereas TEOAEs and SFOAEs can be detected in essentially all ears with normal hearing. Spontaneous emissions are restricted to low amplitudes by the compressive nonlinearity of the OAE generator, which essentially limits the maximum output of the cochlear amplifier. Like the other OAE types, SOAEs are most commonly detected in the 1- to 2-kHz region, where reverse transmission through the middle ear is most efficient (Kemp et al., 1986).

As would be expected from their origin in the mechanism responsible for evoked OAEs, SOAEs demonstrate vulnerability to insults similar to that of TEOAEs and SFOAEs (see below), and they are not found in frequency regions for which hearing thresholds exceed about 20 dB HL (Bonfils, 1989; Lonsbury-Martin, Cutler, & Martin, 1991). However, because SOAEs are not ubiquitous and because they occur only at a few idiosyncratic frequencies for those ears in which they are found, their utility as tools for the detection of hearing impairment is severely limited. (Table 2 includes a review of some of the strengths and weaknesses of SOAEs with respect to their clinical usefulness.) Additionally, it should be emphasized that the existence of an SOAE implies only that the OHC system is functioning normally at the frequency of the SOAE (Bonfils, 1989). Further, it is also important to note that a few normal-amplitude SOAEs have been detected at the edges of audiometric notches (Ruggero, Rich, & Freyman, 1983; Wilson & Sutton, 1981). Thus, even ears with hearing loss may have SOAEs within their normal-frequency regions.

The results of early emission studies indicated that the frequencies of SOAEs in healthy hearing regions were highly stable (e.g., Zurek, 1981). In spontaneously emitting ears, it was thus expected that SOAEs would make useful longitudinal detectors of subsequent auditory dysfunctions. However, the amplitudes of these emissions vary by as much as 30 dB between measures on different days, making their utility for chronologic studies of hearing problematic (Wilson, 1986a; Wit, 1985).

When SOAEs were discovered, it was also anticipated that they would provide some insight into the pathologic mecha-

TABLE 2. Clinical utility of spontaneous otoacoustic emissions.

Strengths	Weaknesses
Stimulus generation unnecessary	Low prevalence
Stable frequencies over time	Genetic preference for females
	Limited frequency range
	Variable amplitudes over time
	Few idiosyncratic frequencies/ear
	Cannot test in ears w/>20 dB HL

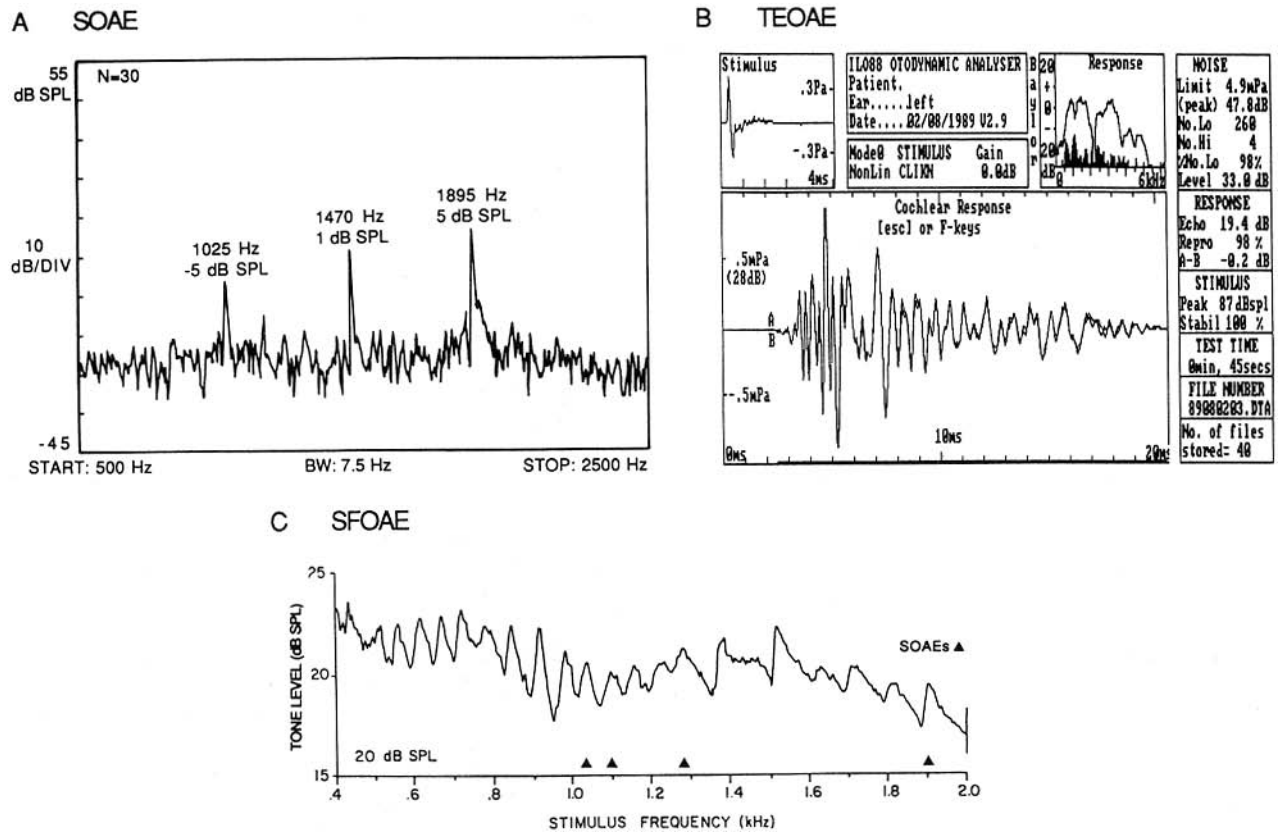

FIGURE 2. Examples of three types of otoacoustic emissions. A: Spectral average (n = 30) of an ear-canal signal from one ear of a normally hearing human in which three SOAEs were detected. B: A standard record depicting TEOAEs measured with an Otodynamic Analyzer (ILO88). Plot information includes the acoustic waveform of the click stimulus recorded in the ear canal (top left), the amplitude spectrum of the emission (top right) and associated noise (filled region), and two independent time waveforms (A and B) of the TEOAE superimposed on one another (below). Data describing the patient and mode of stimulation are itemized at the top center of the record. The panel at the far right lists several useful values regarding the noise level (A − B) during the recording and the preset value of the noise-rejection level (NOISE), the strength (Echo in dB) and reliability (Repro in %) of the emission (RESPONSE), details concerning the stimulus (STIMULUS), and other information. C: Record of sound pressure level in the ear canal evoked by a continuous pure tone of 20 dB SPL, swept slowly (i.e., over 150 s) in frequency, from 0.4–2 kHz. Arrowheads delineate the frequencies of associated SOAEs in this ear. Ripples in the trace represent the interaction in the ear canal of the SFOAE with the swept tone.

nisms underlying tinnitus. Unfortunately, the association between SOAEs and tinnitus is somewhat obscure (Norton, Schmidt, & Stover, 1990; Penner & Burns, 1987; Wilson, 1986b; Wilson & Sutton, 1981; Zwicker, 1987). Whereas many normally hearing people can detect one or more of their SOAEs as tonal tinnitus, especially when they are made aware of their emissions, this is usually not a source of annoyance, and many spontaneously emitting subjects claim not to hear their SOAEs. In addition, surveys of SOAE incidence in ears of tinnitus sufferers have typically revealed a lower incidence of SOAEs than in normal ears. These results are consistent with the fact that tinnitus is often associated with impaired hearing, whereas SOAEs are generally measured in regions of normal hearing. There have been rare cases, however, in which annoying tinnitus has been shown to be caused by SOAEs (e.g., Penner, 1988, 1989a, 1989b).

Transiently Evoked Otoacoustic Emissions

Transiently evoked otoacoustic emissions (TEOAEs) occur in response to acoustic transients (e.g., clicks, tone-bursts) presented to the ear. In humans, they can be detected in essentially all normally hearing ears (Bonfils, Bertrand, & Uziel, 1988; Kemp, 1978; Kemp et al., 1986; Probst, Coats, Martin, & Lonsbury-Martin, 1986; Stevens, 1988). The stimulus transient is delivered by a miniature loudspeaker sealed into the ear canal, and the amplified microphone output is typically sampled for about 20 ms following the stimulus presentation and averaged in order to enhance the signal-to-noise ratio. The averaging procedure is time-locked to the presentation of the stimulus much as it is in measurements of the electrically recorded auditory brainstem response.

Transiently evoked emissions in response to click stimuli consist of a delayed, nonlinear, frequency-filtered "echo" of the stimulus (Kemp, 1978). Across ears, the observed frequency filtering is highly idiosyncratic. This feature of TEOAEs can be appreciated upon inspection of the amplitude spectrum of the response, which is usually determined by fast-Fourier transformation (FFT) of the time-domain waveform. Because of the ear's innate capability for generating and conducting OAEs, a healthy ear typically demonstrates several regions of strong evoked response, between 0.4 and 6 kHz, each several hundred Hertz wide, and

separated by narrower regions of reduced response (Sutton, 1985; Wilson, 1980a). An example of the time waveform and amplitude spectrum of a TEOAE is shown in Figure 2B. The frequency regions of strong response vary greatly among ears, but, for any one ear, they are stable over years (Kemp, 1978, 1982). The middle ear is inefficient at transmitting sound out of the cochlea to the ear canal at low and high frequencies, resulting in the low- and high-frequency limits of TEOAE detection. The latency of TEOAEs, that is, the time between presentation of the stimulus and detection of the response, is typically 5–20 ms in humans and tends to decrease as the frequency of the OAE component increases. These delay times suggest a cochlear origin of TEOAEs. Further, the frequency dependence of the latency suggests that the place of TEOAE origin moves basally in the cochlea with increasing frequency. Thus, the OAE generator is apparently distributed along the cochlea's frequency-place axis. Consequently, frequency-specific components of the click-evoked TEOAE response can be selectively excited by the use of tonebursts near the component frequency as the eliciting stimuli (Norton & Neely, 1987; Probst et al., 1986; Wilson, 1980b).

Transiently evoked emissions may be detected in response to stimulus levels well below the threshold of hearing of the evoking signal (Probst et al., 1986; Zwicker, 1983b). These observations indicate that there is little opportunity for neural involvement in their generation, because it is well established that auditory nerve-fiber thresholds closely approximate behavioral thresholds. A sensory origin for TEOAEs is further indicated by the precise inversion of the response waveform with stimulus polarity (Anderson, 1980) and the lack of the neural phenomenon of adaptation that has been shown for click-evoked OAEs (Kemp, 1982; Rutten, 1980). Kemp and Chum (1980a) determined from the amplitude of some TEOAEs that more energy could be emitted by the ear than was present in the evoking stimulus, suggesting that an active element providing added energy is involved in their generation. Transiently evoked emissions grow linearly with stimulus levels below about 10 dB SPL but exhibit a strong saturating nonlinearity at higher stimulus levels such that they rarely evidence growth above stimulus levels of 20–30 dB SPL. This compressive nonlinearity (see Pickles, 1988, for a thorough discussion of the nonlinearities of cochlear function), which describes the relatively smaller emissions at high intensities, provides a valuable way of distinguishing TEOAEs from other artifacts in the time waveform of the sound in the ear canal following an acoustic transient. Ringing of the sound source and middle-ear effects, for example, generally grow linearly with increasing stimulus intensity (see below).

Factors known to cause sensorineural hearing loss have also been found to reduce or abolish TEOAEs. Experiments in normal human ears have shown that both administrations of salicylate (Johnsen & Elberling, 1982) and brief noise exposures (Kemp, 1982; Zwicker, 1983a) that result in temporary threshold shifts can cause reversible reductions in TEOAE amplitudes. Patients known to have hearing losses primarily of cochlear origin (see below; Bonfils & Uziel, 1989; Kemp, 1978; Probst, Lonsbury-Martin, Martin, & Coats, 1987) demonstrate reduced or absent TEOAEs. Animal experiments have shown that hypoxia (Zwicker & Manley, 1981) and the ototoxic loop diuretics furosemide and ethacrynic acid (Anderson, 1980) also reduce TEOAE amplitudes.

The prevalence and basic properties of TEOAEs in the ears of infants and neonates appear to be similar to those in adults (Johnsen, Bagi, Parbo, & Elberling, 1988; Stevens, Webb, Smith, Buffin, & Ruddy, 1987). However, TEOAEs are larger in infants and neonates, at least partly because of the smaller volumes of their ear canals (Bray & Kemp, 1987; Norton & Widen, 1990), and perhaps because of differences in the resonance of the middle ear.

The ubiquity of TEOAEs in normally hearing human ears and their reduction by factors known to cause sensorineural hearing loss suggest that TEOAEs may be well suited for the detection of cochlear disorders. (The strengths and weaknesses of TEOAEs as the basis for a clinical test of cochlear function are summarized in Table 3). The observation that there is no neural involvement in their generation further suggests that emissions may be particularly valuable as specific indicators of the sensory versus the neural component of sensorineural hearing loss. Because TEOAEs are easily measurable with simple averaging techniques, are distinct from their evoking stimuli in time, and can be distinguished from artifacts on the basis of their compressive nonlinearity, much attention has been focused, with some success, on developing practical tests of hearing impairment utilizing TEOAEs (Kemp et al., 1986; Kemp, Ryan, & Bray, 1990). The only commercial instrument that is presently available for recording TEOAEs (see Kemp et al., 1990), the Otodynamic Analyzer (Otodynamics Ltd., ILO88), takes advantage of the beneficial features described above in its measurement of these responses.

Stimulus-Frequency Otoacoustic Emissions

From the findings described above, it is clear that brief, broad-band stimulation of the ear results in a transient emission of low-level sound from the ear at certain frequencies and that components of the transient response can be specifically elicited by the use of tonebursts near the component frequency (Probst et al., 1986; Wilson, 1980a). Thus, it follows that continuous tonal stimulation at such frequencies causes a continual reemission of low-level sound from the ear at the stimulus frequency (Kemp & Chum, 1980b; Wilson, 1980a). The stimulus-frequency otoacoustic emissions (SFOAEs) are more difficult to study than TEOAEs,

TABLE 3. Clinical utility of transiently evoked otoacoustic emissions.

Strengths	Weaknesses
Present in normal ears	Tests only innate frequencies
Measured using standard averaging	Variable amplitudes over time
Stable frequencies over time	Restricted to <5 kHz
Temporally separate from stimulus	Limited dynamic range
Stimulus-related frequency pattern	Cannot test in ears w/>30 dB HL

however, because it is necessary to separate them from the stimulus tone that is simultaneously present in the ear canal and larger than the reemission. (Table 4 summarizes the strengths and weaknesses of SFOAEs as clinical measures).

The existence of SFOAEs is revealed by slowly sweeping the frequency of the stimulus tone across a prescribed region encompassing up to 1–2 kHz. As the frequency of the stimulus increases, the phase lag of the reemission relative to the stimulus also increases, resulting in a physical interaction between the stimulus and the emitted response in the ear canal. At frequencies of strong evoked response, this process results in a ripple in the otherwise smooth frequency response of the sound in the ear canal at low stimulus levels, as the stimulus and the reemission move alternately in and out of phase (Wilson, 1980a). At high stimulus levels, the size of the emission relative to the stimulus is very small because of the compressive nonlinearity of its generator. Thus, the physical interference that causes the expression of SFOAEs in the ear canal is no longer detectable. In Figure 2C, a trace of the amplitude of the sound pressure recorded in a sealed human ear canal for a low-level stimulus is provided to demonstrate SFOAEs. The peaks and troughs in this amplitude spectrum occur when the stimulus and the SFOAE interact.

Although SFOAEs have been studied much less completely than TEOAEs, they show characteristics similar to those of transient emissions, a fact that is consistent with the notion that SFOAEs arise from the same generator as TEOAEs. For example, SFOAEs are present in virtually all normal human ears (Lonsbury-Martin et al., 1990a), are measurable in those frequency regions in which TEOAEs are strong (Zwicker & Schloth, 1984), and can be detected at stimulus levels well below the threshold of hearing (Wilson, 1980b). Like TEOAEs, SFOAEs are reduced or eliminated in frequency regions of sensorineural hearing loss. It is probable that SFOAEs could provide the same information as TEOAEs in tests of hearing impairment. However, because the detection of SFOAEs is more complicated and time-consuming than the measurement of TEOAEs, SFOAEs have not been incorporated into clinical tests of cochlear function.

Distortion-Product Otoacoustic Emissions

To evoke distortion-product otoacoustic emissions (DPOAEs), two stimulus tones of moderate level (55–75 dB SPL), separated in frequency, are presented to the ear. Nonlinear processes that are innate to the healthy cochlea result in the creation of responses at frequencies not present in the two-tone input. Although distortion products in the form of audible combination tones have been known to exist for many years, Kemp (1979a) was the first to demonstrate DPOAEs in human ears. Acoustic-distortion products have not been studied as extensively as TEOAEs. However, according to the findings of several systematic studies (Harris, 1990; Harris, Lonsbury-Martin, Stagner, Coats, & Martin, 1989; Lonsbury-Martin, Harris, Hawkins, Stagner, & Martin, 1990b), they appear to be a property of all normally hearing individuals over a frequency range extending from about 0.5 to 8 kHz. (Table 5 summarizes the strengths and weaknesses of DPOAEs as the basis for a clinical test of cochlear function).

The strongest DPOAE in human ears occurs at the cubic-difference frequency described by the algebraic expression $2f_1 - f_2$, in which f_1 represents the lower frequency stimulus or primary tone and f_2 the higher frequency primary. In humans, DPOAEs are low in amplitude, usually about 60 dB lower than the levels of the eliciting primary tones. Consequently, it is essential that the generating and measuring equipment have linear-response properties and a dynamic range of at least 80 dB over the frequency and amplitude ranges of these emissions. To prevent distortion generation in the transducer, the two primary tones are typically presented through separate speakers. The outputs of these transducers pass through sound tubes that connect to an acoustic speculum sealed snugly within the external ear canal where the two primary stimuli are acoustically mixed (see Figure 1). The measurement of DPOAEs at selected frequencies is achieved by spectral averaging of the microphone output. In Figure 3A, a typical FFT-based spectrum of the ear-canal signal is shown for which an emission at the $2f_1 - f_2$ frequency of 2.5 kHz is evident. Several earlier studies focused on determining the frequency region that makes the most significant contribution to the generation of the $2f_1 - f_2$ DPOAE (Brown & Kemp, 1984; Martin, Probst, Scheinin, Coats, & Lonsbury-Martin, 1987). The outcomes of these investigations support the notion that the cochlear place primarily responsible for generating this emission is closely approximated by the geometric mean of the primaries, $(f_1 \times f_2)^{0.5}$.

It is well established that DPOAE amplitudes depend critically on both the level and frequency of the primary stimuli. In addition to the frequencies and levels of the eliciting tones, crucial variables include the difference between the levels of f_1 and f_2 (i.e., $L_1 - L_2$) and their frequency ratio (i.e., f_2/f_1). The frequency separation between the two

TABLE 4. Clinical utility of stimulus-frequency otoacoustic emissions.

Strengths	Weaknesses
Present in normal ears	Complex to measure and interpret
Stable frequencies over time	Tests only innate frequencies
	Limited frequency/amplitude ranges
	Variable amplitudes over time
	Provide same results as TEOAEs
	Cannot test in ears w/>20 dB HL

TABLE 5. Clinical utility of distortion-product otoacoustic emissions.

Strengths	Weaknesses
Present in all normal ears	Dual-stimulation set-up
Tests 1–8 kHz range	Difficult to test <1 kHz
Tests selectable frequencies in detail	Cannot test in ears w/>55 dB HL
Precise extraction of test frequency	
Dynamic range of 40–50 dB	

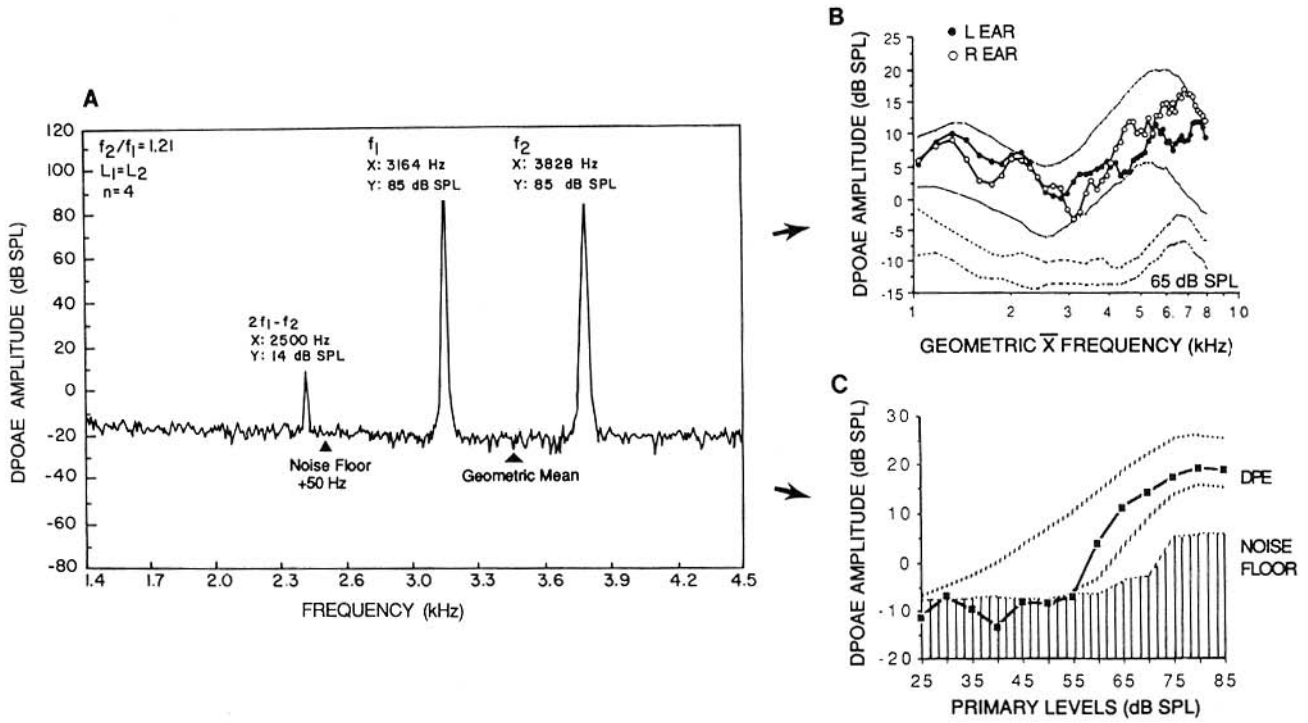

FIGURE 3. Spectral average (n = 4) of a typical $2f_1 - f_2$ DPOAE at 2.5 kHz recorded from a normally hearing individual. Details of the stimulus parameters are noted at the top left of the record and in association with each spectral peak representing the DPOAE, f_1, or f_2. The solid triangles represent the frequency 50 Hz above the emission (left), at which the related noise-floor level was measured, and the geometric mean of f_1 and f_2 (right). An emission is assumed to be present if its amplitude at $2f_1 - f_2$ is >3 dB above the level of the corresponding noise floor at $(2f_1 - f_2)$ + 50 Hz. To the right are examples of the two DPOAE response forms, with the audiogram (top) displaying emission amplitude in response to equilevel primary tones, as a function of the geometric mean of the primaries, and the response/growth function (below) showing emission magnitude at one frequency as a function of systematic increases in the level of the primaries. The geometric mean of f_1 and f_2 is used for plotting purposes, because the DPOAE is believed to be generated around the cochlear place corresponding to the primary frequencies. The average range ($M \pm 1$ SD) of emission amplitudes for normally functioning ears is depicted by the top pair of broken lines in each plot, whereas the corresponding noise floors are represented by the lower pair of dashed lines in the audiogram and the vertical-striped lines in the growth function.

primary tones is a principal factor in determining DPOAE amplitude. Kemp and Brown (1983) reported maximal DPOAEs using f_2/f_1 values of 1.25, and a similar optimal ratio of about 1.22 was determined later by others (Gaskill & Brown, 1990; Harris et al., 1989) as the most effective stimulus for eliciting DPOAEs from 1 to 4 kHz. In the latter studies, it was noted that maximal DPOAE amplitudes for low-frequency emissions were evoked with larger (1.26) f_2/f_1 values or at relatively high stimulus levels, whereas the largest high-frequency DPOAEs were noted for smaller (1.19) f_2/f_1 ratios when low-level primaries were used.

Although a great deal is known about the optimal frequency distance between f_1 and f_2, the effects of systematically varying the level difference between the primary tones (i.e., $L_1 - L_2$) have been less thoroughly examined. The findings of several recent studies determined that DPOAEs are maximal when L_1 is about 10 dB or more greater than L_2 (Gaskill & Brown, 1990; Hauser & Probst, 1990; Probst & Hauser, 1990). The results of detailed studies in rabbits further showed that the optimal $L_1 - L_2$ decreases with increasing stimulus intensity (Whitehead, Lonsbury-Martin, & Martin, 1990). It is not presently known whether human DPOAEs are produced by distinct low- and high-level generators, depending on stimulus level, as appears to be the case in rabbits (Whitehead et al., 1990). Because of the lack of a complete knowledge concerning the testing of DPOAEs in humans, the levels of the primaries are typically equated ($L_1 = L_2$) and restricted to <90 dB SPL (e.g., Harris, 1990; Harris et al., 1989; Kimberley & Nelson, 1989; Lonsbury-Martin et al., 1990b; Martin, Ohlms, Franklin, Harris, & Lonsbury-Martin, 1990; Smurzynski, Leonard, Kim, Lafreniere, & Jung, 1990).

In addition to the factors discussed above, the amplitudes of DPOAEs are dependent on the confounding influence of SOAEs, TEOAEs, and SFOAEs. For example, Wier, Pasanen, and McFadden (1988) showed that if the DPOAE frequency coincides with an SOAE, the DPOAE amplitude can be enhanced so that the acoustic-distortion product for low-level primary tones is only 10–20 dB smaller than the level of the eliciting primaries. However, this potentiation effect is limited to a narrow DPOAE frequency range of about 50 Hz around the SOAE.

Acoustic-distortion products are commonly measured using two protocols, the results of which are illustrated at the right of Figure 3 as a DPOAE audiogram (B) and as a response/growth or input/output (I/O) function (C). In the DPOAE audiogram, the frequency pattern of an ear's ability to generate acoustic-distortion products is established by

measuring emission amplitudes as a function of the geometric mean of the two primary frequencies. In this manner, DPOAEs are measured for primary tones maintained at a constant level (e.g., $L_1 = L_2 = 55$, 65, or 75 dB SPL), and emission frequency is increased in regular intervals of 10 steps per octave. In the response/growth protocol, a series of I/O functions are determined at geometric-mean frequencies that are related to the conventional audiogram (i.e., at 1, 2, 3, 4, 6, 8 kHz) by varying the primary-tone levels in 5-dB steps between 25 and 85 dB SPL. Several quantitative features of the emitted response can be determined from the resulting curves, including detection threshold (i.e., the lowest stimulus level at which the DPOAE is >3 dB re the related noise floor), maximum amplitude, dynamic range, and slope, which relates the rate at which the emission grows as a function of increased primary-tone levels. From a clinical perspective, both analyses of acoustic-distortion products are useful, because the DPOAE audiogram appears to reflect the frequency configuration of the standard audiogram (e.g., Martin et al., 1990), whereas the detection threshold of the I/O function apparently has a systematic relation to hearing level in many cochlear-based diseases (e.g., Ohlms, Lonsbury-Martin, & Martin, 1991).

Clinical Applications of Evoked-Emission Tests

It is important to emphasize that for OAEs to be measured, the middle ear must be essentially normal (Rossi, Solero, Rolando, & Olina, 1988). For other tests of the auditory pathway such as the auditory brainstem response (ABR), the middle ear must be normal to ideally support the forward transmission of acoustic signals. However, the healthiness of the middle ear is even more crucial for measuring OAEs, because emissions testing depends on the ability of the middle ear to transmit sound energy into the ear at levels that are typically lower than the transient stimuli traditionally used to elicit ABRs. Moreover, to record emissions in the outer ear canal, even lower level energy, which is propagated in the reverse direction, must be detected. In fact, there is some evidence from experiments in rabbits that factors that modify the conduction capability of the middle ear (e.g., activation of the acoustic reflex) have a greater reducing effect on the magnitude of the reversely transmitted DPOAE than on the level of the forward-traveling stimulus (Whitehead, Lonsbury-Martin, & Martin, 1991).

Some of the significant strengths of the OAE techniques reviewed above are that evoked emissions can be measured objectively and noninvasively by placing a small microphone in the outer ear canal. Additionally, with microcomputer technology, evoked emissions can be measured rapidly, accurately, and with high resolution with respect to the primary stimulus features of level and frequency, thus permitting a very fine analysis of these properties. Another important aspect of evoked OAEs is that they are present in the ears of essentially all normally hearing subjects and are reduced or absent in those affected by cochlear disorders. Because of these advantages, it is clear that evoked emissions in the form of TEOAEs and DPOAEs, both of which can be measured in a relatively straightforward manner, have considerable potential as clinical tools to assess the contribution that OHC dysfunction makes to a patient's hearing impairment. Here, examples are provided of clinically determined hearing levels re the average normal threshold of hearing (American National Standards Institute, 1989) and TEOAEs and DPOAEs measured in patients representative of those visiting a typical audiology clinic. In these cases, an attempt is made to illustrate the particular features of TEOAEs and DPOAEs that make them potentially important to the diagnosis and treatment of cochlear-based hearing impairments.

In the examples, the TEOAEs were determined with the microcomputer-based Otodynamic Analyzer (ILO88), operated in the nonlinear click mode. The important clinical information provided by this analysis includes the automatically determined reproducibility of the emission (REPRO) reported in percent, which describes the degree of correlation between two separately determined temporal averages (A and B) of the click-evoked emission (see Kemp et al., 1990), and the emission (ECHO) amplitude and noise (difference between the response waveforms, i.e., A − B) level in dB. Thus, from the latter two values, emission-to-noise ratios can be estimated. These values are displayed on the video monitor as shown in the RESPONSE panel, at the right of Figure 2B. On the basis of experience with TEOAE testing, the standard interpretation of these factors is that if the reproducibility value is greater than 50%, and the emission (ECHO) amplitude is at least 5 dB greater than the noise (A − B), a reasonable conclusion is that the patient's hearing level is better than 25–30 dB HL for frequencies at which emissions are present, that is, typically between 0.7–4 kHz.[1]

In the DPOAE examples, acoustic-distortion products were measured routinely both in the form of audiograms and as a series of I/O curves, acquired at audiometric frequencies, with the computer-controlled laboratory instrumentation described previously by Lonsbury-Martin et al. (1990b). For both measures, each patient's findings are related to the average range of values determined previously for a population of normally hearing subjects (Martin et al., 1990). In the illustrations provided, the normal range (±1 *SD*) of DPOAE amplitudes is depicted as the upper pair of dashed lines, and the measuring system's noise floor is represented as the bottom pair of dashed lines. In some I/O plots, vertical-striped lines indicate the beginning of the upper 1 *SD* of the normal noise floor.

One pragmatic issue of evoked-emissions testing concerns the test/retest reliability of these new measures of cochlear processing. In Figure 4, an example of the test/retest reliability of evoked OAEs is illustrated for the right ear of JL, a 19-year-old woman with excellent hearing. The emitted responses were collected during a number of test

[1]The criterion for identifying an emission, i e., the relative difference between the emitted response and the related noise floor, is smaller for DPOAEs (3 dB) than for TEOAEs (5 dB). This difference in determining detection threshold probably arose from the understanding that the DPOAE frequency is accurately specified according to the algebraic expression $2f_1 - f_2$, whereas the TEOAE comprises many frequencies. Thus, the broad frequency spectrum of the TEOAE requires a more conservative definition to allow for chance increases in the noise floor at one or more frequencies.

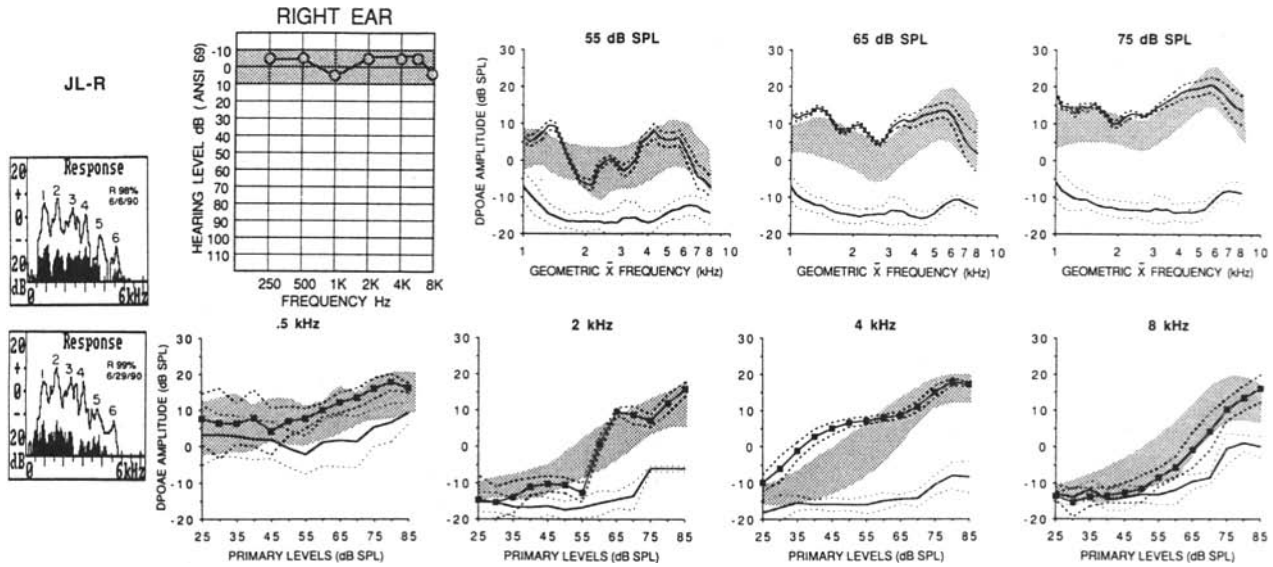

FIGURE 4. Test/retest determinations for two assessments, 23 days apart, of TEOAEs and seven determinations of DPOAEs, over 3 weeks, obtained for the right ear of a normally hearing 19-year-old woman. Note the excellent repeatability of the TEOAEs (left) and DPOAE audiograms (top right) and I/O functions (bottom), especially for the frequencies between 1 and 4 kHz. For DPOAEs, the mean (solid line: audiogram; solid squares: I/O curves) levels (± 1 *SD*) are plotted. The shaded regions on the DPOAE plots represent the average ranges for young normally hearing adults between the ages of 20 and 30 years (Lonsbury-Martin et al., 1990b).

sessions distributed over about a 3-week period. The two TEOAE spectra illustrated at the left exhibit the six-peak frequency pattern and high reproducibility that were characteristic of this patient over seven test periods. Similarly, the DPOAE audiograms (top right) elicited by low- to high-level primaries illustrate the mean (±1 *SD*) DPOAEs calculated for the seven sessions, with the shaded region representing the average range of amplitudes for young, normally hearing subjects (Lonsbury-Martin et al., 1990b). Note the excellent repeatability of these measures, especially for frequencies

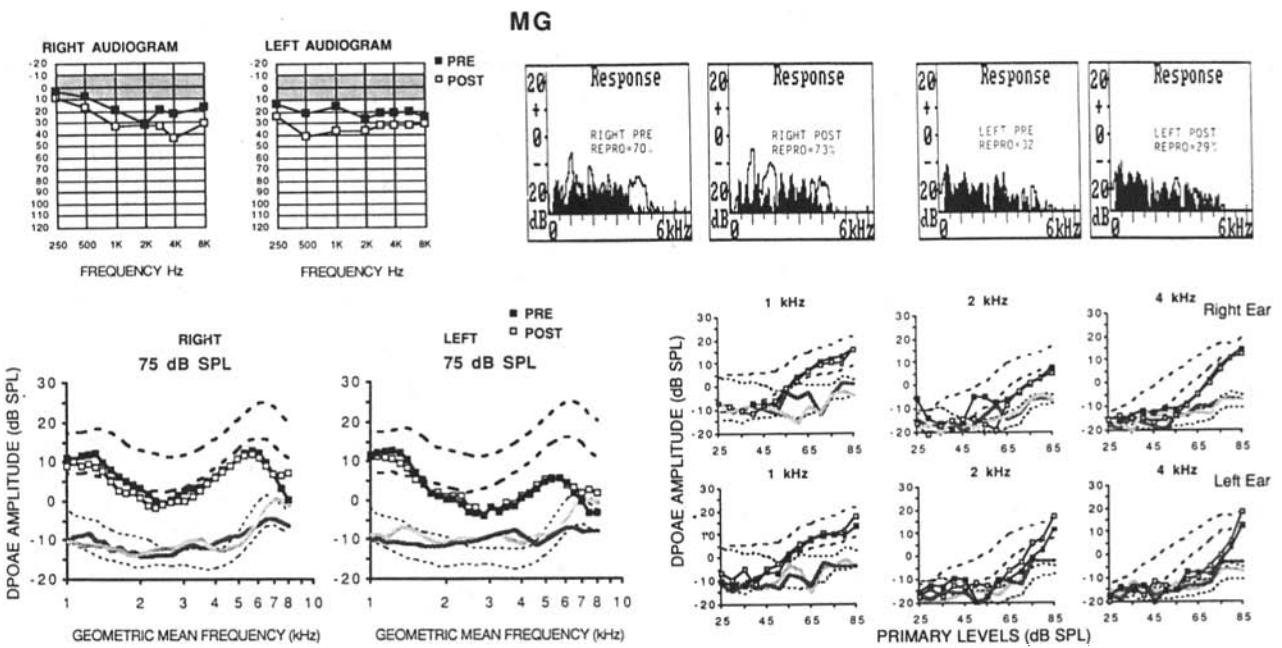

FIGURE 5. Audiometric and emission data measured before (solid symbols) and 3 hrs following the administration of glycerol (open symbols) to a 51-year-old woman suspected of having Ménière's disease. The behavioral audiograms (top left) indicate that, in general, postglycerol hearing (open squares) was worse than predrug (solid squares) levels by as much as 20 dB, depending on the test frequency. In contrast, the TEOAEs (top right) and the DPOAE audiograms (bottom left) and I/O functions (bottom right) corresponding to the pre- and postglycerol measures showed little change between the two separate determinations. The gradual decrease in the magnitudes of the high-frequency emissions of this patient is a frequent observation in aging ears (Lonsbury-Martin et al., 1991).

<4 kHz. In the lower portion of the figure, similar comparisons are performed for I/Os selected to encompass the range of audibility. The good reproducibility of the I/O measures also is clearly apparent, especially at the middle frequencies.

As Figure 4 demonstrates, one important benefit of otoacoustic emissions is their ability to yield objective and, consequently, very reliable test/retest measures. The potential importance of this capability is illustrated in Figure 5. In this case, the goal of the clinic visit was to provide evidence that would rule out the presence of Ménière's disease in MG, a 51-year-old woman with a 1-year history of bilateral hearing loss, tinnitus, and the sensation of fullness or pressure. Toward this end, testing of both emission types and hearing were performed before and at 3 hr following the administration of the hyperosmotic agent, glycerol. At the time of retesting, the patient complained of a severe headache and responded poorly during conventional threshold audiometry. The postglycerol hearing levels (top left: open squares) were less sensitive than the preglycerol measures by 10 dB or more. The finding that hearing was more impaired following the ingestion of glycerol was contrary to the osmotically induced functional improvement commonly observed in the early stages of Ménière's disease. In contrast to the varied audiometric results, both the TEOAEs (top right) and DPOAEs (audiograms: lower left; I/Os: lower right) evidenced little change between pre- and postglycerol responses. On the basis of the combined outcomes, it was concluded that MG's diverse behavioral-hearing results appeared to be unrelated to the presence of endolymphatic hydrops and were most likely caused by a headache-induced inattentiveness during the postglycerol test. In this case, the objectivity of the emissions examination was not affected by unfavorable extraneous influences that are sometimes associated with subjective testing under less than ideal conditions.

Because evoked OAEs are detected optimally with moderate-level stimuli, an additional advantage of these measures is that they are sensitive to relatively small amounts of hearing impairment. In Figure 6, the effects of a sudden, fairly flat hearing loss of about 20 dB on TEOAEs (top left) and DPOAEs (audiogram: top right; I/Os: below) are illustrated for the right ear of BS, a 60-year-old woman. The spectral plot reveals a rather restricted frequency of the TEOAEs com-

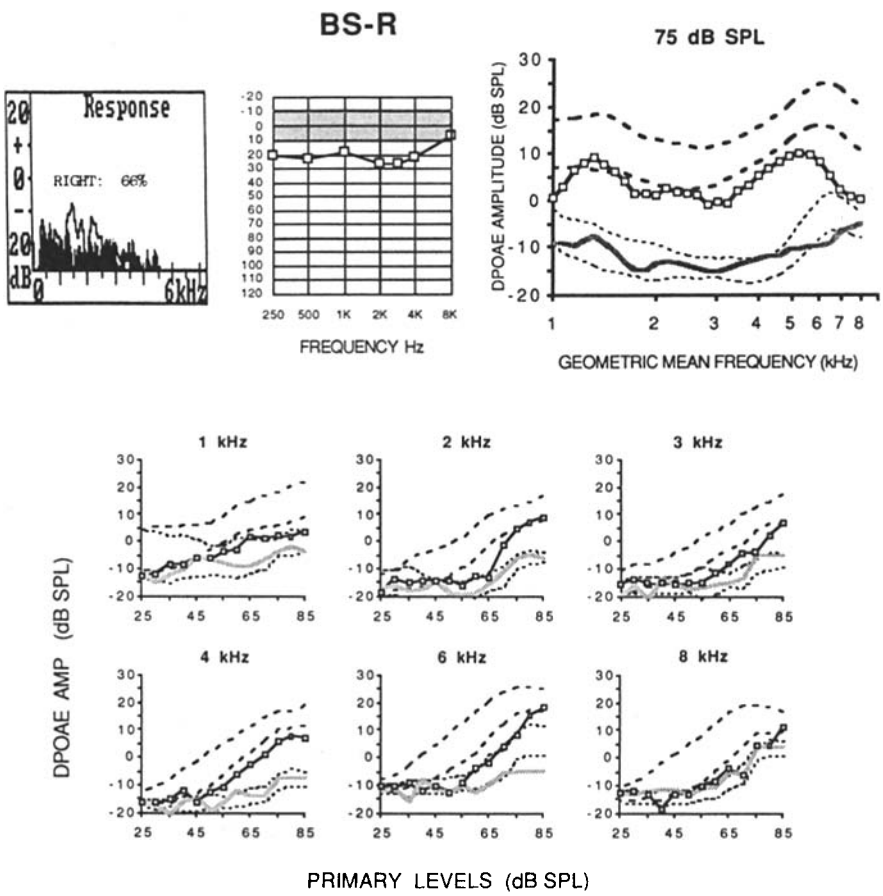

FIGURE 6. Results of hearing and emissions testing for the right ear of a 60-year-old woman who recently developed a hearing loss in this ear. Note that the 15- to 25-dB HL responses depicted on the standard audiogram (top center) were associated with reduced TEOAEs (top left), compared to the robust response of the normally hearing individual of Figure 2B, and lower than average DPOAEs as measured by both the audiogram (top right) and I/Os (below), which also displayed threshold shifts of about 20–30 dB.

pared to the pattern illustrated in Figure 2B for a typical normally hearing individual. The scant TEOAEs were complemented by the below-normal levels of the DPOAEs (squares) observed in the audiogram. The abnormally small DPOAEs were also evident in the I/O curves, which displayed detection thresholds that, depending on the test frequency, were elevated by 10–25 dB compared to average thresholds, even though the patient's (shaded lines) measured noise floors were within the normal limits designated by the lower pair of dashed lines.

The information in Figure 6 also illustrates the unique advantages of each of the two evoked emissions types, TEOAEs and DPOAEs, for assessing the cochlear reserve of ears exhibiting functional difficulties. According to the pattern of TEOAE activity, hearing sensitivity between about 1.5 and 2.5 kHz should be better than 30 dB HL. This prediction, based upon a brief test that was performed in <1 min, is supported by the results of the pure-tone audiometry. However, for this particular patient, the mechanisms responsible for either the generation or the expression of TEOAEs were not robust enough to permit an evaluation of lower or higher frequency ranges. In contrast, the DPOAE findings describing lower than normal levels reflected the mild hearing impairment that encompassed the major portion of the audiometric-test range. In this case, the capacity of DPOAEs for eliciting emission activity at deliberately selected frequencies was an advantage over the TEOAE test, which depends on the inherent ability of the cochlear partition to resonate at particular frequencies in response to brief acoustic stimulation. The benefits of using DPOAEs compared to TEOAEs in clinical testing await more systematic analyses.

Another positive feature of evoked emissions that makes them clinically useful is their specificity for testing the micromechanical activity of OHCs. Because OHCs are the auditory receptors that are usually most sensitive to the damaging influences of bacterial or viral pathogens, external agents such as excessive noise or ototoxic drugs, and inherited factors that cause deafness (see Schucknecht, 1974), the evoked emissions are extremely sensitive to many common cochlear disorders. In addition, the clinical utility of evoked OAEs as objective tests of auditory function is greatly enhanced by their ability to test discrete, frequency-specific regions of the cochlea so that frequency areas of impaired hearing can be adequately distinguished from regions of normal function. Clear illustrations of the frequency specificity of evoked OAEs combined with their ability to test OHC activity specifically are provided by instances of hearing loss caused by exposure to excessive sound, which is known, especially in the early stages, to damage primarily the OHCs. In such cases, the frequency boundary between normal and abnormal hearing is usually well demarcated.

The data in Figure 7 for OM, a 64-year-old man, illustrate

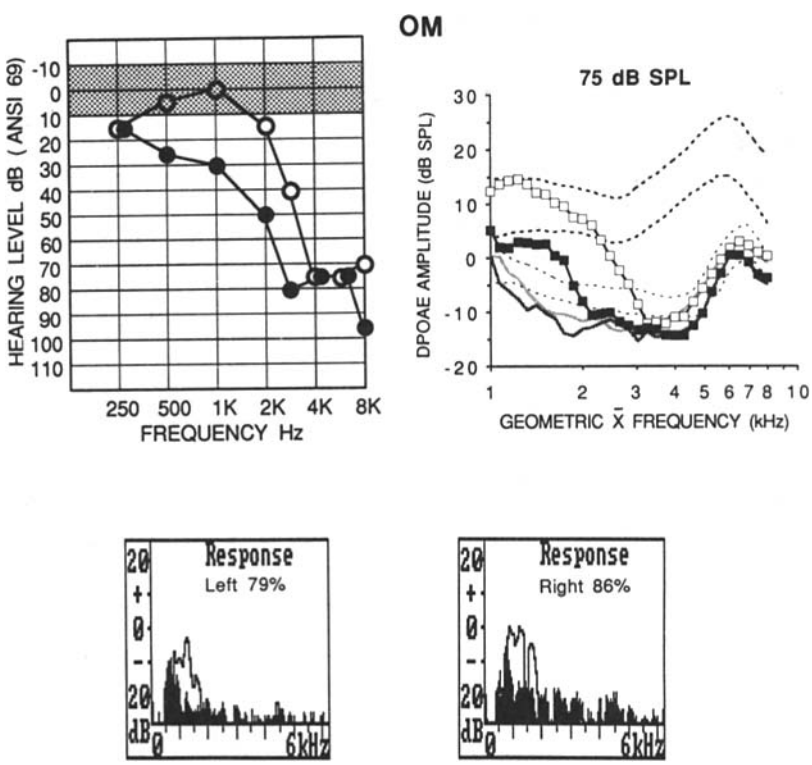

FIGURE 7. Example of the effects of a lengthy (almost 40-year) exposure to industrial noise for a 64-year-old man. The TEOAE findings (below) are consistent with the >30 dB hearing level and presumed lack of OHC activity above about 2 kHz. Because DPOAE methods have a larger dynamic range and can deliberately test discrete frequencies, details of the asymmetry of the resulting audiometric thresholds (top left) were accurately revealed by the DPOAE audiograms (top right).

the consequences of exposure to excessive sound on hearing and on the generation of OAEs. Because of the patient's age, it is probable that presbycusis contributed significantly to his high-frequency hearing loss. However, it is reasonable to assume that the majority of OM's low- to mid-frequency loss is attributable to his 38-year exposure to factory noises. The behavioral audiogram shows the classic pattern of hearing loss exhibited by an individual who operated a noisy metal lathe in that the head-shadow effect protected, in this case, the right ear, which was directed away from the machine's motor. The TEOAE spectra (below) are consistent with the elevated hearing thresholds in that no emissions were detected for either ear above about 1.5–2 kHz. Similarly, the DPOAE audiograms (top right) depict the capability of these emissions to effectively track the different patterns of hearing loss exhibited for the two ears, with the left ear displaying dysfunction at a lower frequency than the less impaired right ear.

The hearing impairment due to aging alone can be caused by defects involving a number of cochlear sites, including the sensory cells, the stria vascularis, and the basilar membrane. The example shown in Figure 8 illustrates an instance of progressive presbycusis in BH, a 54-year-old woman. In this case, all three measures of auditory function—the clinical audiogram, the TEOAEs, and the DPOAEs—indicate a significant dysfunction at frequencies above 2 kHz. Note the symmetric pattern of hearing loss between ears, which is clearly mimicked by the DPOAE audiograms and I/O functions.

The ability of evoked OAEs to measure OHC function also permits emitted responses to exhibit another benefit that is compatible with clinical goals: Specifically, evoked OAEs can decisively isolate the sensory component of a sensorineural hearing loss. This capacity of evoked OAEs is illustrated in Figure 9 for the left ear of BS. This ear had been previously diagnosed with Ménière's disease on the basis of an otologic history that included dizziness, tinnitus, a fluctuating hearing loss, and a feeling of fullness. At the time of testing, the patient exhibited a fairly flat hearing loss of about 40–50 dB. The spectral pattern at the left of Figure 9, which displays the outcome of the transient-emissions testing, is consistent with threshold hearing levels no better than 25–30 dB; that is, no TEOAEs were detectable. However, given observations that DPOAEs evoked by primaries <80 dB SPL are typically absent in cases of sensorineural hearing loss resulting in hearing levels of 40–50 dB (Martin et al., 1990), the clearly reduced, but measurable, emission audiogram (top right) and I/Os (below) are more consistent with hearing levels of about 30 dB. Thus, the results of emissions testing for the left ear of BS could be interpreted to imply that the sensorineural hearing loss resulting from Ménière's disease had two components. According to this line of reasoning, approximately 30 dB of the elevated threshold findings was likely caused by the disease's adversely affecting OHC function, whereas the remaining impairment was probably due to the dysfunction of critical cochlear elements located central to the OHC system, that is, the inner hair cells or the dendritic endings of the auditory nerve. This example illustrates one of the important advantages of evoked-OAE testing: its ability to isolate the sensory component of a sensorineural disease. It is quite conceivable that this beneficial feature of emissions testing will eventually provide better insights into the underlying

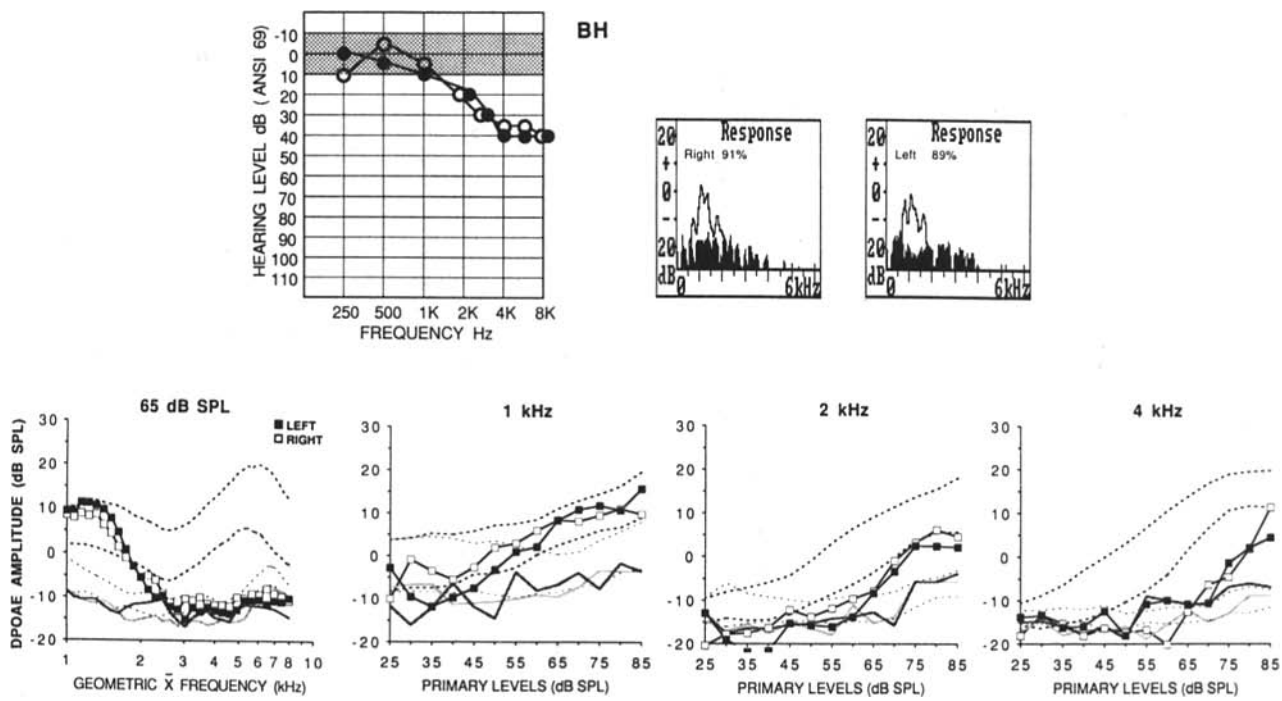

FIGURE 8. Example of presbycusis in a 54-year-old woman who was unaware of her hearing loss. The symmetrical pattern of the high-frequency hearing loss depicted by the clinical audiograms (top left) was faithfully tracked by the DPOAE audiograms and reflected in selected I/Os (below), at 2 and 4 kHz, showing DPOAE threshold elevations. Spectra for measures of TEOAEs (top right), for each ear, also revealed the lack of emitted responses for frequencies above about 2 kHz.

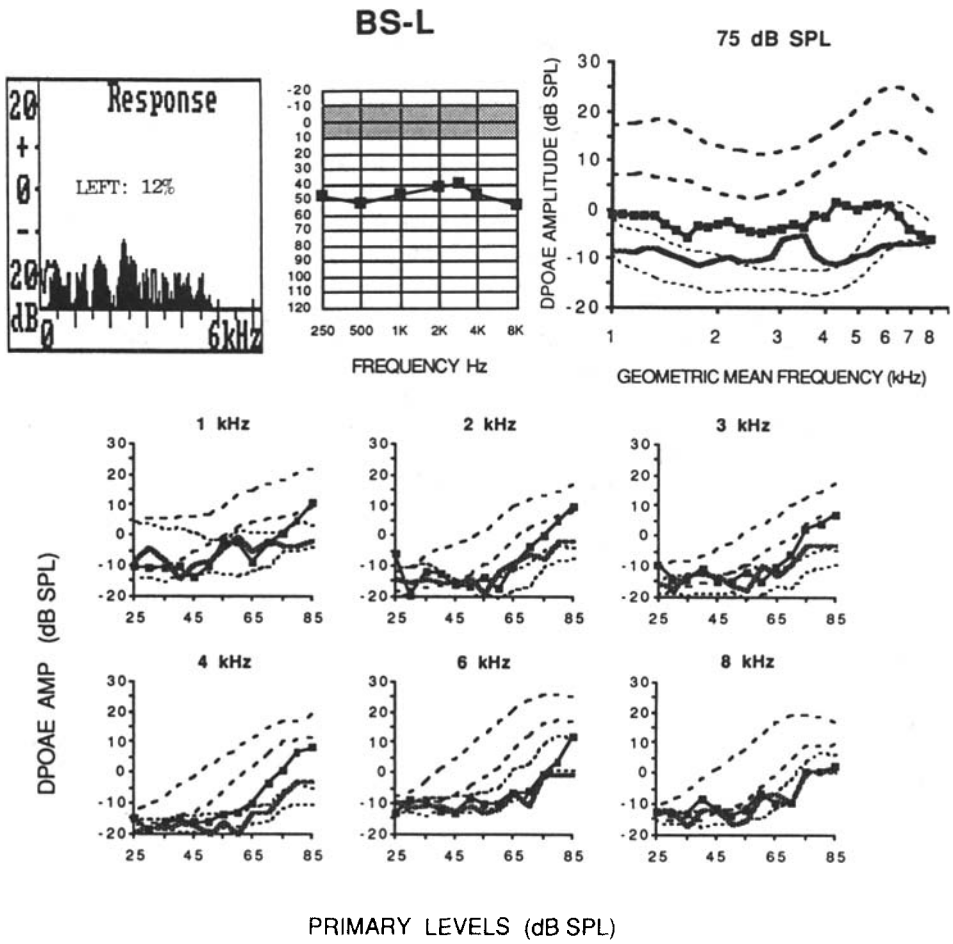

FIGURE 9. Audiometric and emission data for the left ear of the patient depicted in Figure 6. The hearing loss in this ear was previously diagnosed as being caused by Ménière's disease according to classic symptomatology. The TEOAE and DPOAE results were consistent with the proposition that the initial amount of threshold shift (i.e., ~30 dB) depicted in the clinical audiogram (top center) was probably due to damage to the OHC system. However, the remaining degree of threshold elevation was likely caused by disease-induced injury to cochlear structures that were more central to the OHCs, because DPOAEs elicited by primary tones <75 dB SPL would normally be absent in association with hearing levels of 40–50 dB.

basis of complicated sensorineural hearing disorders like Ménière's disease. Such greater understanding may aid in the development of improved prophylactic and restorative treatments of this typically difficult-to-manage affliction.

One practical issue of evoked-emissions testing concerns the assessment of hearing in difficult-to-test patients. The evoked OAE data illustrated in Figure 10 show a rather unique application of this technique in TM, a 3-year-old boy. The "before" responses demonstrate absent TEOAEs and minimal DPOAEs during a time when both otoscopic and tympanometric evidence indicated the presence of active middle-ear disease in the form of a fluid line on the tympanic membrane and a type-B tympanogram. In contrast, the "after" data illustrate the benefits gained from a combination of myringotomy surgery, in which a purulent exudate was aspirated from the middle-ear space and tympanic ventilation was achieved by inserting a polyethylene (PE) tube through the tympanic membrane. From the information displayed in these plots, it is clear that, following surgery, hearing improved and TEOAEs became measurable, with an 82% reproducibility factor compared to the −18% presurgery value. In addition, DPOAEs increased from near noise-floor levels to normal magnitudes for frequencies <3 kHz. In this particular case, the lack of high-frequency responses suggest that either some small amount of remaining serous fluid impeded the middle-ear's conduction capability at these frequencies, or that the repeated treatment of the young patient for recurrent disease with topical antibiotics caused ototoxicity. In any case, in instances such as this, which involve a compromise in the middle ear's capacity to conduct OAEs (e.g., serous otitis media), emitted responses can be used to evaluate the adequacy of the palliative treatment, whether surgical or medical.

The plots of Figure 11 reinforce the acknowledgment that emissions testing should be considered as a beneficial adjunct to, but not a replacement for, standard diagnostic tests. These data were determined for RB, a 56-year-old man, who complained of a noise-induced hearing loss that had been present since military service in an armored tank division, some 30 years previously. In addition, this patient had a long history of eardrum

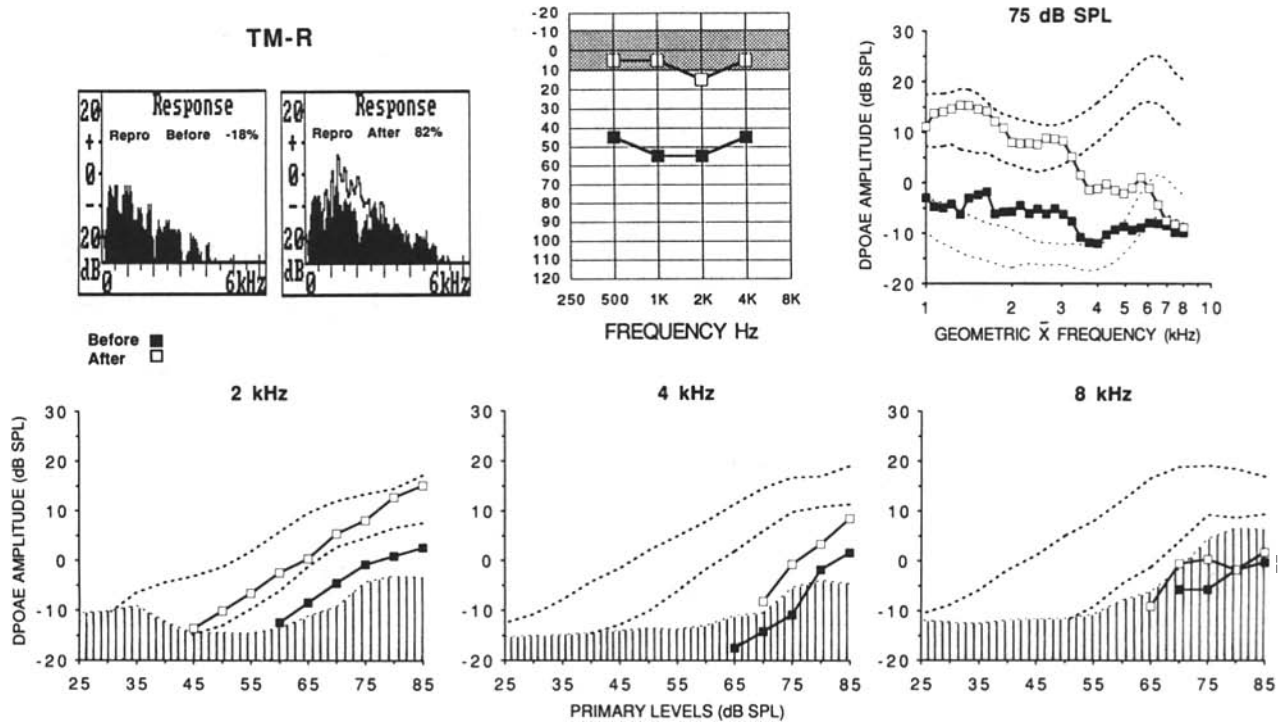

FIGURE 10. Example of the utilization of evoked OAEs to test the integrity of the middle-ear transmission system. The right ear of this 3-year-old boy had a long history of repeated middle-ear infections. Before surgery (closed symbols) was performed to clear the middle ear of a viscous exudate and to insert a ventilating tube in the tympanic membrane, essentially no TEOAEs (top left) and very low-level DPOAEs (top right and below) were measurable. However, following surgery (open symbols), both TEOAEs and DPOAEs regained reasonably normal levels, at least up to about 4 kHz, which matched the recovered hearing levels.

perforations on the left side, due to repeated childhood ear infections and several deep-diving incidents during his teenage years, respectively. The tympanograms at the top left were consistent with normal middle-ear function in the right ear and with the established diagnosis of a monomeric tympanic membrane on the left side. The clinical audiograms depicted in the top middle plot were consistent with a bilateral high-frequency hearing loss, probably due to the combined effects of noise exposure and aging, along with a mild mid-frequency loss in the left ear. The essentially absent emissions for the left ear indicate the deleterious effects of the middle-ear disorder on the expression of both TEOAEs and DPOAEs. In contrast, the right ear, which displayed good middle-ear function and better hearing at the low and middle frequencies, exhibited both types of evoked OAEs, up to the frequency at which behavioral thresholds rapidly deteriorated to 80 dB or greater. These data illustrate the vulnerability of the reverse-transmission process to middle-ear disease and, thus, support the necessity of performing a complete audiologic assessment in order to accurately and thoroughly describe the status of the peripheral-hearing apparatus.

Discussion

The examples presented above support the proposition that both TEOAEs and DPOAEs have a beneficial clinical application. Other positive features not illustrated include the ability of evoked emissions to accurately track dynamic changes in OHC-based disease. This longitudinal tracking capability is useful in monitoring cochlear function in some forms of sudden idiopathic sensorineural hearing loss and in the early stages of Ménière's disease upon either a challenge with an osmotic agent or in instances involving naturally occurring spontaneous fluctuations in sensory function (Martin et al., 1990; Ohlms, Lonsbury-Martin, & Martin, 1990). Other applications include the ability of TEOAEs and DPOAEs to definitively depict the more steady progressive deteriorations in hearing commonly observed in hereditary impairments and in cases of congenitally related disease (e.g., asymptomatic cytomegaloviral disease). Further, in cases for which it can be safely assumed that OHC damage is primarily involved in the confirmed hearing impairment (e.g., noise-induced hearing loss), ongoing studies suggest that abnormal hearing levels can be estimated from the magnitude of the remaining DPOAEs (Lonsbury-Martin & Martin, 1990; Ohlms et al., 1990). Finally, with respect to the important clinical application of hearing screening, TEOAEs have already proven useful in the newborn population (Kemp et al., 1990; Norton & Widen, 1990; Stevens et al., 1989). Similarly, TEOAEs and DPOAEs also may prove useful as a hearing screener in industrial hearing-conservation programs, in monitoring the progressive hearing loss caused by aging, and in identifying the onset stage of ototoxicity during treatment with certain antibiotic or antitumor agents.

Summary

A number of the properties of evoked emissions and resultant measures support the proposition that these newly developed tests of auditory functioning will make a significant

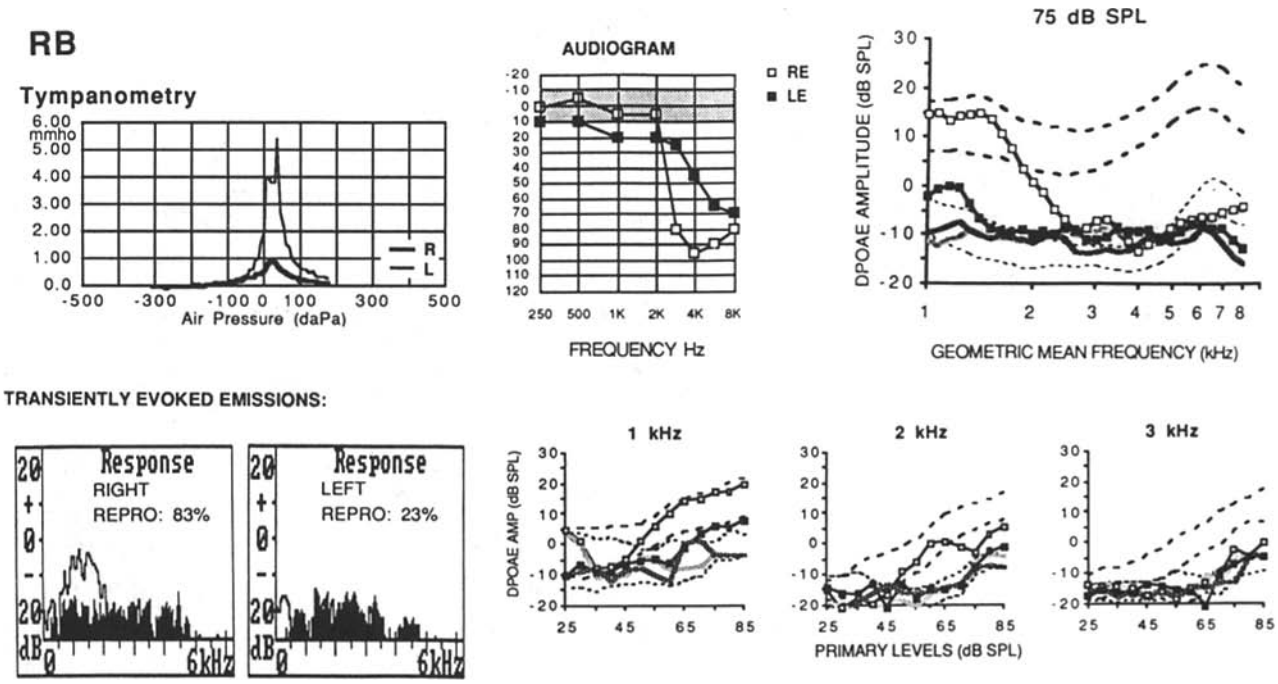

FIGURE 11. An example of the influence of middle-ear pathology on the expression of evoked emissions for a 56-year-old man. At the top left, the tympanogram patterns (admittance in mmhos as a function of air pressure in daPa) are consistent with a long-standing history of a normally functioning middle ear on the right side, along with the presence of a monomeric tympanic membrane on the left side. The clinical audiograms (top center) also reflect the patient's history of bilateral noise exposure during previous military service, in combination with a positive pathologic history for the left middle ear, which experienced a number of episodes of otitis media while the patient was a child and several eardrum perforations when he was a teenager. The corresponding evoked-OAE data show the capacity of these responses to efficiently track the abrupt mid- to high-frequency noise-induced loss evidenced by the right ear (open squares). However, the middle-ear dysfunction prevented the successful performance of emissions testing in the left ear, which exhibited only a mild hearing impairment over the low- to mid-frequency range. Although not tested during this session, previous bone-conduction testing revealed an air/bone gap of about 10 dB in the left ear.

contribution to the clinical practices that deal with hearing impairment. Some of the more general strengths of OAEs in applied settings include their objectivity, noninvasiveness, and compatibility with microcomputer-based control, which allows simple, accurate, rapid, and high-resolution measurements. In addition, evoked emissions are present in the ears of essentially all normally hearing individuals and are systematically reduced or absent in the ears of sensorineurally hearing-impaired patients (see Kemp et al., 1986; Kemp et al., 1990; Martin et al., 1990). Further, the ability to measure OAEs evoked by relatively low-level stimuli results in a capacity to detect early stages of hearing loss. Finally, the specificity of OAEs for measuring the micromechanical activity of OHCs allows them to characterize distinct frequency boundaries between regions of normal and impaired hearing in many cochlear-based disorders and to isolate the sensory component of a sensorineural hearing loss.

Other specific benefits of evoked emissions that offer clinical promise include their straightforward application in difficult-to-test patients; their high degree of test/retest reliability in ears with either normal or abnormal function; their sensitivity to the conductive state of the middle-ear system, which potentially makes them detectors of subclinical pathology in infants and children; their ability to track function that alters over time in dynamically changing ear diseases and in progressive conditions that cause hearing loss over longer periods; and, finally, the systematic relationship of certain evoked-OAE properties to hearing level.

It is important to note that despite the useful information that otoacoustic emissions can contribute toward the evaluation of hearing, a number of practical issues need to be resolved to increase our understanding of the meaningfulness of these measures. For example, the consequences of middle-ear disease on the reverse-transfer function of the middle ear, especially with respect to childhood diseases like otitis media and adult afflictions such as otosclerosis, need further study. With respect to our current knowledge about the influence of middle-ear factors on emission transmission, it is noteworthy that even basic information concerning the effects of perforations of the tympanic membrane on the recordability of emissions is not available. Other more normal conditions such as presbycusis, which appears to also affect the conduction of emissions from the cochlea to the ear canal, are just beginning to be methodically investigated (Bonfils et al., 1988; Lonsbury-Martin et al., 1991). One potential outcome of gaining a more detailed knowledge of the relation of the status of the middle ear to OAE properties may be the capability of combining immittance and emission findings to calculate accurate transfer functions for the conduction apparatus. In any case, experience to date with

evoked emissions in clinical settings and the availability of some normative databases on TEOAEs and DPOAEs make it clear that OAEs can be developed as a screening procedure and as a diagnostic tool, both of which have the potential of becoming important parts of the basic evaluation of hearing.

Acknowledgments

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