Journal of Speech and Hearing Research, Volume 35, 1142–1159, October 1992

Distortion-Product Emissions and Auditory Sensitivity in Human Ears With Normal Hearing and Cochlear Hearing Loss

David A. Nelson
Barry P. Kimberley*
Department of Otolaryngology
University of Minnesota
Minneapolis

Distortion product emissions (DPEs) at $2f_1 - f_2$ frequencies were measured in 53 human ears; 21 of them exhibited cochlear hearing loss. DPEs were obtained as a function of stimulus level (DPE growth curves) at seven frequency regions between 707 Hz and 5656 Hz. Several distinctly different shapes or patterns of DPE growth curves were observed. These included single-segment monotonic growth curves with and without saturation at moderate and high stimulus levels, diphasic growth curves with nulls at moderate stimulus levels, and non-monotonic growth curves with negative slopes at high stimulus levels. Low-level, irregularly shaped segments were more frequent in normal-hearing ears, suggestive of normal low-level active nonlinearities from the outer-hair-cell subsystem. High-level, steeply sloped segments were frequent in hearing-impaired ears, suggestive of residual nonlinearities from a cochlear partition without functional outer hair cells. The stimulus level at which the DPE could just be distinguished from the noise floor, the DPE detection threshold, demonstrated moderate positive correlations (r's from 0.50 to 0.81) with auditory thresholds when all ears, both normal and impaired, were considered together. Those correlations were not strong enough to quantitatively predict auditory thresholds with any great accuracy. However, DPE thresholds were able to predict abnormal auditory sensitivity with some precision. DPE thresholds correctly predicted abnormal auditory sensitivity 79% of the time in the present study, and up to 96% of the time in previous studies. These results suggest that DPE thresholds may prove useful for hearing screening in cases where cooperation from the subject is limited or where corroboration of cochlear hearing loss is required. Different patterns of DPE growth curves suggest underlying micro-mechanical differences between ears, but the differential diagnostic value of those patterns remains to be determined.

KEY WORDS: distortion-product otoacoustic emissions, hearing loss, DPE thresholds, hearing screening

Otoacoustic emissions recorded from the occluded external auditory canal are low-intensity acoustic signals emanating from the cochlea, either spontaneously or as a result of acoustic stimulation. One of the stimulated otoacoustic emissions, the distortion-product emission (DPE), can be recorded from the ear canal at a frequency equal to $2f_1 - f_2$ during continuous stimulation with pure tones at f_1 and f_2 frequencies, where $f_1 < f_2$. The DPE has been linked to outer-hair-cell function (Dallos, Harris, Relkin, & Cheatham, 1980; Fahey & Allen, 1986; Furst, Rabinowitz, & Zurek, 1988; Horner, Lenoir, & Bock, 1985; Kim, Molnar, & Matthews, 1980; Norton & Mott, 1987; Ruggero, Rich, & Freyman, 1983; Siegel & Kim, 1982; Wiederhold,

*Currently affiliated with the University of Calgary, Alberta, Canada.

0022-4685/92/3505-1142$01.00/0

Mahoney, & Kellog, 1986; Zurek, 1985; Zurek, Clark, & Kim, 1982). For example, in a nude mouse model, Horner et al. (1985) reported that cochleas with intact outer hair cells but defective inner hair cells produced DPEs, whereas those with intact inner hair cells but absent outer hair cells produced no DPEs. This relation between DPEs and outer-hair-cell function may prove particularly useful for diagnosing cochlear hearing loss in human ears if a strong relation can be found between auditory threshold and some aspect of the DPE.

An association between stimulated otoacoustic emission measures and the presence of cochlear hearing loss was suggested in the initial research noting their discovery (Kemp, 1978). Emissions evoked by click stimuli were recorded from 21 ears with cochlear hearing loss. Evoked emissions were generally not present in ears with sensory thresholds exceeding 30 dB HL.

Subsequent studies have confirmed and extended these findings. Harris (1988, 1990) and Harris and Glattke (1988) found very good agreement between low DPE levels and high auditory thresholds in some of their subjects. However, in other subjects, that relationship did not hold. Martin, Ohlms, Franklin, Harris, and Lonsbury-Martin (1990) demonstrated similar findings. They reported strong negative correlations between DPE level and auditory threshold in subjects with noise-induced hearing loss. Gaskill and Brown (1990) reported that a subjective correspondence between DPE levels and behavioral audiograms existed in 80% of the ears they tested, but they also reported a statistically significant correlation between DPE levels and auditory thresholds across the audiometric frequency range in only 50% of the ears.

Unfortunately, correlations between DPE levels and auditory thresholds have not been strong enough to allow accurate predictions of auditory threshold from DPE level in many hearing-impaired ears. Whether this is due to differing cochlear micromechanical dysfunctions that reflect the same auditory thresholds, or to methodological limitations in the recording procedures, is not yet clear (see Martin, Probst, & Lonsbury-Martin, 1990, for a thorough review of these issues).

One reason why some subjects demonstrate strong negative correlations between DPE level and auditory threshold, while other subjects may not, might be understood from the nonmonotonic behavior of DPE growth curves in some ears under differing stimulus conditions. Wiederhold et al. (1986) demonstrated in animals that the growth of DPE level with stimulus level was nonmonotonic. Specifically, he found localized nulls in the DPE growth curve. At low stimulus levels, DPE level increased monotonically with stimulus level. Then, at stimulus levels around 55 dB SPL, DPE level dropped dramatically before rising again with further increases in stimulus level. To explain those nulls, they postulated the existence of multiple emission generator sources (or sites) with different phase characteristics. Presumably, when summed in the external auditory canal or within the cochlea, multiple emissions canceled one another at appropriate stimulus levels. Even earlier, Kim et al. (1980) had noted similar nonmonotonic growth behavior for DPEs from animals. Fahey and Allen (1986) studied this nonmonotonic behavior further. They showed that nulls in the DPE signal were dependent upon the ratio of f1 and f2 and upon the absolute and relative levels of the two primaries. Brown and Gaskill (1990) demonstrated the same types of nonmonotonicies in human ears, which are consistent with the existence of more than one generator site for DPEs within the cochlea.

If the existence of multiple emission generator sites, and the subsequent demonstration of nonmonotonic DPE growth curves is somewhat subject dependent, it is understandable that over a range of stimulus levels, in some subjects, DPE levels might very well decrease with stimulus level instead of increasing with stimulus level. This outcome could confound any attempt to find a strong correlation between DPE level and auditory threshold across subjects. Perhaps a different measure of DPE behavior, one not so sensitive to nonmonotonic DPE level growth, might provide a better indication of auditory threshold and presumed cochlear hearing loss. Such a measure appears to be the level of the stimulus required just to elicit a DPE larger than the noise floor of the recording system, which is essentially the stimulus level at the intercept between the DPE growth curve and the noise floor. This has been referred to as the *DPE detection threshold* (Lonsbury-Martin, Harris, Stagner, Hawkins, & Martin, 1990), which, for convenience here, we will call the *DPE threshold.* Although it is obviously dependent upon the noise floor of the measuring system, that noise floor appears to be consistent enough across subjects to allow useful information to be retrieved from the measurement of DPE thresholds.

Harris (1990) measured DPE thresholds and auditory thresholds over a frequency range between 750 and 8000 Hz from a group of 20 ears with high-frequency hearing loss and a group of 20 ears with normal hearing. She found that at higher frequencies where hearing losses existed, the number of ears with DPE thresholds better than 55 dB SPL was greater in the normal-hearing ears than in the hearing-impaired ears. She did not report a quantitative relation between DPE threshold and auditory threshold; however, her results indicated that a significant number of her normal-hearing ears had DPE thresholds greater than 55 dB SPL, which would make it difficult to predict elevated auditory thresholds from DPE thresholds.

Leonard, Smurzynski, Jung, and Kim (1990) measured DPE thresholds and auditory thresholds from 17 normal-hearing and 18 hearing-impaired ears at DPE frequencies of 1500 and 3000 Hz. Their normal-hearing ears demonstrated a wide range of DPE thresholds, between 37 and 67 dB SPL, even though auditory thresholds were better than 10 dB HL between 250 and 8000 Hz. No DPEs were evident whenever auditory thresholds were greater than 60 dB HL. When hearing loss ranged between 10 and 60 dB, 75% of the DPE thresholds were greater than seen in normal hearing ears. However, the number of different ears at any one condition was too few to draw any generalized conclusions about quantitative relations between DPE threshold and auditory threshold.

A more quantitative relation between DPE threshold and auditory threshold was reported by Kimberley and Nelson (1989, a, b). They compared DPE thresholds and auditory thresholds in normal-hearing and hearing-impaired ears and

found correlations as high as 0.86 at some frequencies. Similarly, Martin et al. (1990) compared DPE thresholds and auditory thresholds in a group of ears with noise-induced hearing loss. They reported correlations between DPE threshold and auditory threshold that ranged between 0.84 and 0.91 for frequency regions above 1000 Hz where sizable hearing losses existed.

This preliminary work suggests that the relation between DPE threshold and auditory threshold may be strong enough to allow quantitative predictions of auditory threshold from DPE detection thresholds. However, the work to date involves limited samples of hearing-impaired ears. Current results are from too few ears, with insufficient variety of cochlear pathology, to warrant generalizations about the predictability of auditory threshold from DPE threshold. Furthermore, very few details about individual DPE growth curves are known—details that might permit inferences about possible micromechanical cochlear dysfunctions in individual ears.

The present research examines individual DPE growth curves from both normal-hearing and hearing-impaired ears. It assesses the various forms of growth curves found in normal and impaired ears, evaluates an objective procedure for estimating DPE thresholds from DPE growth curves, and it looks for a relation between DPE threshold and auditory threshold that might be realized with such a procedure. It also examines the utility of DPE thresholds as a screening device for hearing loss.

Method

Subjects

Auditory thresholds and DPE thresholds were measured from 53 ears of 27 subjects. Of those 53 ears, 32 exhibited normal auditory thresholds [< 20 dB HL (see ANSI, 1969)] at all test frequencies, and 21 demonstrated a moderate hearing loss at one or more test frequencies. Subjects were selected without regard to age, sex, etiology of hearing loss, or audiometric pattern of sensory loss. Table 1 indicates the ages and etiologies of the subjects with significant sensorineural hearing losses. Subjects with conductive hearing losses, middle-ear pathology, or retrocochlear auditory pathology were not included in this study. Because we were interested in the various DPE growth-curve patterns that might exist in the general population of normal-hearing ears, none of the subjects were screened with a distortion audiogram for high levels of distortion prior to participation in this investigation (Gaskill & Brown, 1990).

DPE Measurement System

The two stimulating tones (f_1 and f_2) were computer synthesized through two separate 14-bit digital-to-analog converters (sample rate of 20 kHz) and low-pass filtered at 10 kHz before exciting two separate Etymotic 2-A transducers. The two stimulating tones were routed, via plastic tubing, through an Etymotic ER-10 ear-canal microphone system into the external ear canal where they were mixed acoustically in the occluded ear canal. An Etymotic ER-10 microphone system and its associated preamplifier were used to record acoustic emissions. A Hewlett-Packard 3561A signal analyzer was used to amplify the microphone output and perform frequency analysis. An IBM/AT computer synthesized the two stimulus tones with the aid of an associated TMS3020 microprocessor. It also controlled the programmable attenuators and the signal analyzer. Once an experiment began it proceeded automatically. Subjects were asked to simply sit quietly inside a sound booth during the DPE recording session.

TABLE 1. Ages and etiologies of hearing-impaired ears.

Ear code	Age	Etiology
High-frequency hearing loss		
BAH(L)	34	noise induced
BAH(R)	34	noise induced
CJL(L)	27	noise induced
CJL(r)	27	noise induced
EIP(1)	66	noise induced + Meniere's
EIP(R)	66	noise induced
PJB(L)	33	ototoxicity
PJB(R)	33	ototoxicity
SCG(L)	37	acquired/genetic
SCG(R)	37	acquired/genetic
TRC(R)	37	noise induced
WDW(L)	66	noise induced + sociocusis
WDW(R)	66	noise induced + sociocusis
Sloping hearing loss		
MGR(L)	29	Alport's
MGR(R)	29	Alport's
PHL(L)	51	unknown
PHL(R)	51	unknown
Flat hearing loss		
HIW(L)	54	Meniere's
JVE(L)	37	viral (scarlet fever)
JVE(R)	37	viral (scarlet fever)
SXW(R)	38	acquired

Experimental Procedure

Auditory thresholds were determined at seven test frequencies (707, 1000, 1414, 2000, 2828, 4000, and 5656 Hz) using TDH-49 headphones mounted in MX/41-AR cushions. A four-interval forced-choice adaptive procedure (using feedback, a 2-dB step size, and the last 6 of 12 reversals to specify threshold) was used to estimate the 71% correct detection level for 500-msec tone bursts that were gated with 10-msec rise and decay times.

DPE growth curves, also referred to as DPE input/output (I/O) functions (Lonsbury-Martin et al., 1990), were measured at seven frequency regions: 707, 1000, 1414, 2000, 2828, 4000, and 5656 Hz. Specifically, the frequencies of the two stimulating tones (f_1 and f_2) were chosen so that their geometric means were at the same test frequencies used to obtain auditory thresholds. The ratio between f_2 and f_1 was held constant at 1.2, so the actual DPE frequencies were 520, 730, 1030, 1470, 2060, 2920 and 4120 Hz. During a single experimental session, which lasted approximately 30

min, DPE growth curves were recorded at all seven frequencies.

A DPE growth curve consisted of measurements of the sound-pressure level of the DPE as a function of the sound-pressure level of the two stimulating tones. The levels of each of the two stimulating tones (primaries) were varied together in 6 dB steps, from 30 dB SPL to 78 dB SPL. Growth curves were always obtained in an ascending series to minimize adaptation or fatigue. At each level of the primaries, three measurements were made from the acoustic spectra in the ear canal: the level of the DPE at $2f_1 - f_2$, the level of the f_1 stimulating tone, and the level of the noise floor. The noise-floor measurement was taken as the level of the frequency components 20-Hz below the $2f_1 - f_2$ DPE frequency. All acoustic measurements were made by RMS averaging the acoustic spectra. Thirty spectra were averaged per measurement. The signal analyzer was set to a frequency-resolution bandwidth of 7.5 Hz. A complex algorithm was used to specify DPE thresholds for each growth curve (see Appendix).

Prior to DPE data collection, the frequency response of each subject's ear canal was measured with the Eytomotic ER-10 microphone assembly in place. During DPE measurements, stimulating tones were presented at sound pressure levels adjusted for minor differences in the frequency-response characteristics of each subject's ear canal. Ear canal sound pressures were specified by the ER-10 microphone voltages corrected by the response of the ER-10 system in a 2-cm^3 cavity terminated by a 1/2-inch condensor microphone (Harris, Lonsbury-Martin, Stagner, Coats, & Martin, 1989). Distortion at $2f_1 - f_2$ was more than 80 dB below the primaries.

Results

Individual DPE Growth Curves

DPE growth curves obtained from the left ear of a normal-hearing subject, DJF(L), are shown in Figure 1. Each panel shows the results at a different frequency region. Auditory thresholds were measured at the geometric frequency (Fgm) between f_1 and f_2, which is indicated in each panel. For example, for Fgm = 707 Hz, f_1 was at 650 Hz, f_2 was at 780 Hz, the DPE was measured at 520 Hz ($2f_1 - f_2$), and the level of the noise floor was measured at 500 Hz. DPE levels (filled circles) and noise-floor levels (NSF) are plotted as a function of the level of the two, equal amplitude, primary tones f_1 and f_2. Auditory threshold in dB SPL is given by the value of *A* in each panel.

The results for DJF(L) in Figure 1 revealed normal auditory thresholds (−6 to 12.7 dB SPL) at all frequencies. DPEs were well above the noise floor at every test frequency. For this ear, DPEs began to appear above the noise floor at stimulus levels around 30 dB SPL. As stimulus level increased, DPE level increased, but not always proportionately. In this ear a single-segment monotonic DPE growth curve was evident at 5656 Hz, and to a lesser extent at 2828 Hz where saturation began at 66 dB SPL. At other frequencies, the DPE growth curves exhibited departures

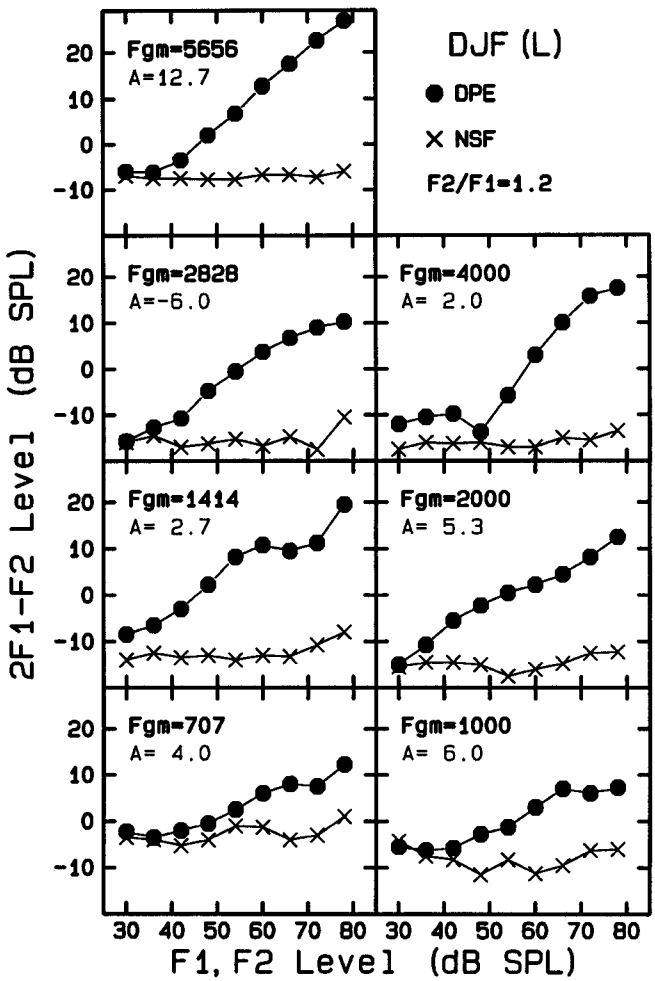

FIGURE 1. DPE growth curves at seven different frequency regions from the left ear of a normal-hearing subject. Level of the $2f_1 - f_2$ DPE (dB SPL) is shown as a function of the level of the F1 and F2 stimulating tones (dB SPL). Filled circles show the level of the DPE as a function of stimulus level. Xs show the level of the background noise floor measured for each stimulus level. The background noise floor (NSF) was specified as the level of the spectrum 20-Hz lower in frequency than the DPE frequency. Each panel is for a different frequency region. The frequency given in each panel (Fgm) is the geometric mean between F1 and F2, which is the frequency at which the auditory threshold was measured. Auditory threshold is given (in dB SPL) by the value of *A* in each panel.

from monotonicity. At 4000 Hz, a localized decrease in DPE level with increased stimulus level was seen at 48 dB SPL, and a steep rise in DPE level followed with further increases in stimulus level. For 707 through 1414 Hz, the DPE growth curves saturated at 60 dB SPL, with a second rising segment for stimulus levels above 72 dB SPL.

The DPE growth curves obtained from the left ear of a 51-year-old hearing-impaired subject, PHL(L), are shown in Figure 2. This ear exhibited a cochlear hearing loss that sloped from a mild loss at low frequencies to a moderate loss at high frequencies. As indicated by the *A* values in each panel, auditory thresholds varied from 43 dB SPL at 707 Hz to 62 dB SPL at 5656 Hz. The hearing loss was due to unknown factors, since there was a negative history of noise

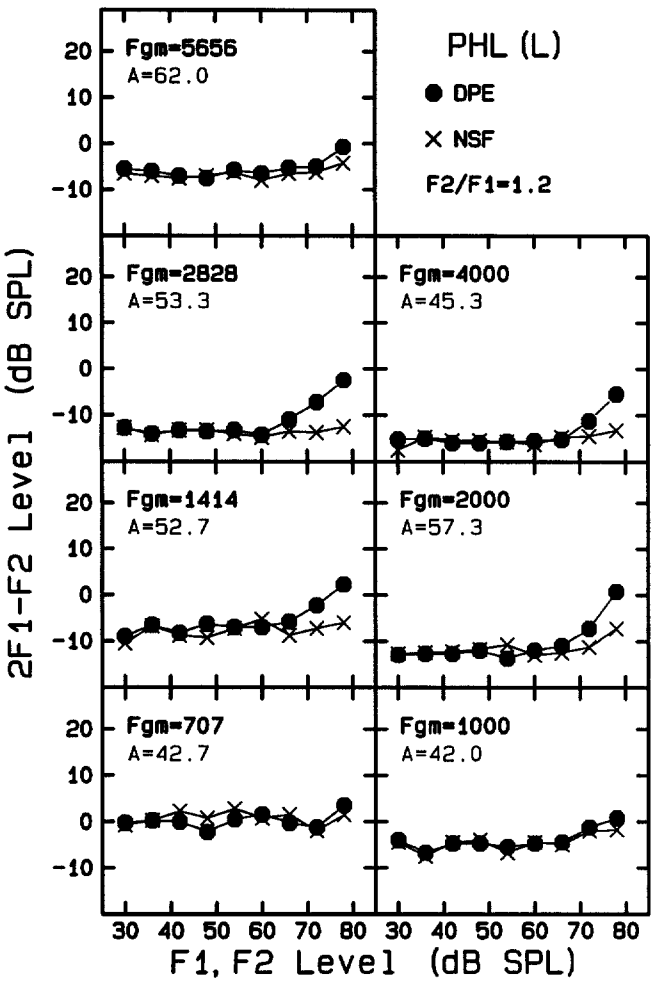

FIGURE 2. DPE growth curves from a subject with a sloping loss toward high frequencies. Legend as in Fig. 1.

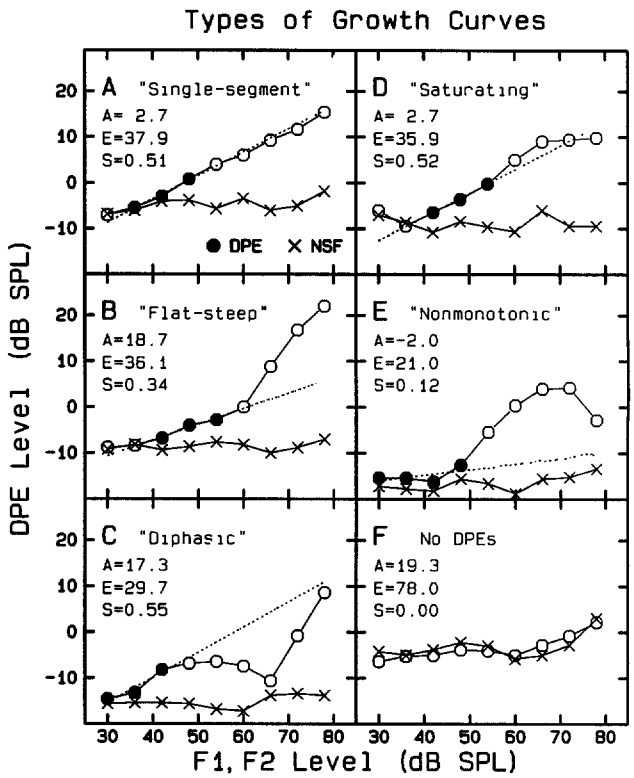

FIGURE 3. Examples of the different types of DPE growth curves that were observed. The DPE threshold estimation procedure is also illustrated. Filled circles indicate the DPE levels used by the threshold estimation procedure. Dotted lines through those DPEs are the best-fit lines used to extrapolate to the average noise floor. The slopes of those best-fit lines are given by the value of *S* in each panel. Auditory thresholds are given (in dB SPL) by the value of *A*. DPE thresholds are given (in dB SPL) by the value of *E*.

exposure and there were no medical indications of ear disease. DPE levels from this ear were considerably lower than seen in the previous figure for normal-hearing subject DJF(L). DPE levels were measurable above the noise floor for all of these frequency regions, but only at stimulus levels above 60 dB SPL.

The results from these two subjects are typical of others reported in the literature. The DPE growth curves from the normal-hearing ear in Figure 1 exhibit large DPEs at all test frequencies. DPE levels from the ear with hearing loss in Figure 2 are reduced in amplitude, and the DPE growth curves begin to rise above the noise floor at higher stimulus levels. However, many of the DPE growth curves obtained from our sample of normal-hearing ears were not as ideal as these examples. Many were not single-segment monotonic (straight-line) functions. Some were two-sloped, others were diphasic with evidence of nulls, some showed sloping saturation, and others were strongly nonmonotonic showing decreasing DPE levels with increasing stimulus level. Since one of our goals was to evaluate an objective procedure to estimate DPE thresholds, the different types of growth curves encountered should be considered.

Types of DPE Growth Curves

Subjective examination of DPE growth curves from both normal-hearing and hearing-impaired ears revealed that their growth curves could be divided into six distinguishable types. Figure 3 shows examples of five of the six different types of DPE growth curves obtained from normal-hearing and hearing-impaired ears, along with an example of a DPE growth curve that was not above the noise floor sufficiently to exhibit a DPE threshold. The frequency of occurrence of each type of growth curve is given in Table 2. In this figure and the next, one example was chosen to demonstrate each type of DPE growth curve. The results of the DPE threshold estimation procedure, which is described in detail in the Appendix, are also illustrated in Figure 3. The filled circles show the points chosen by the procedure used to estimate DPE thresholds. The noise-floor levels associated with those points were used to calculate the noise-floor level associated with each DPE threshold. The dashed line through those points, the *best fit line,* was extrapolated to the average noise floor to estimate DPE threshold. The slope of that line is given by the value of *S* in each panel. The DPE threshold is indicated in the legend by the value of *E*. As in the previous figures, auditory threshold is indicated by the value of *A*.

TABLE 2. Frequency of occurrence of different types of DPE growth curves observed in normal-hearing and hearing impaired ears.

	Frequency region (Hz)								
	707	1000	1414	2000	2828	4000	5656	Totals	%
Normal-hearing ears (n = 32)									
Single segment	14	11	5	3	5	10	12	60	27.6
Flat-steep	3	2	1	2	3	5	9	25	11.5
Diphasic	7	10	10	15	6	4	4	56	25.8
Saturating	3	6	13	9	13	12	3	59	27.2
Nonmonotonic	1	3	3	3	5	1	0	16	7.4
Two-point	0	0	0	0	0	0	1	1	0.5
Total DPE growth curves recorded								217	
No DPEs	4	0	0	0	0	0	3	7	3.1
Total cases								224	
Hearing-impaired ears (n = 21)									
Single-segment	7	4	8	6	6	6	2	39	42.9
Flat-steep	1	0	1	2	2	1	1	8	8.8
Diphasic	2	5	5	2	2	0	0	16	17.6
Saturating	1	1	1	1	0	1	0	5	5.5
Nonmonotonic	0	1	1	2	2	0	0	6	6.6
Two-point	1	1	2	3	2	2	6	17	18.6
Total DPE growth curves recorded								91	
No DPEs	9	9	3	5	7	11	12	56	38.1
Total cases								147	

The first type of DPE growth curve, called a *single-segment monotonic DPE growth curve,* is shown in Figure 3(A). Single-segment growth curves rose above the noise floor with a constant slope that was less than 1.0. In this ear the slope of the DPE growth curve was 0.51. DPE threshold estimation was straightforward for these types of growth curves, since only a single slope was involved. As indicated in Table 2, single-segment growth curves tended to occur somewhat less often at mid frequencies (1414, 2000, and 2828 Hz) than at lower (707, 1000 Hz) and higher (4000 and 5656 Hz) frequencies. Of all the DPE growth curves obtained from normal-hearing ears, 28% were single-segment growth curves. In ears with significant hearing losses, 43% of the DPE growth curves that could be recorded were single-segment growth curves.

A variant of the single-segment monotonic growth curve was what we refer to as a *flat-steep DPE growth curve.* An example is shown in Figure 3(B). The flat-steep growth curves were similar to single-segment curves in terms of monotonicity, but there was evidence of two segments to the curves: a flatter-sloped segment at lower stimulus levels followed by a steeper-sloped segment at higher stimulus levels. In this case the flatter segment had a slope of 0.34 and the steeper segment had a slope greater than 1.0. DPE threshold estimation for flat-steep growth curves utilized the flatter slope of the initial segment, since it would result in the lowest threshold estimate. As indicated in Table 2, flat-steep growth curves occurred less frequently than single-segment curves. The frequency of occurrence was about the same in normal-hearing and hearing-impaired ears, 12% and 9% respectively.

A somewhat more complex growth curve is shown in Figure 3(C). We refer to these as *diphasic DPE growth curves* because they appear to reflect two different underlying functions that interact to produce the null seen at moderate stimulus levels. The diphasic growth curves were characterized by a gradually sloped segment at lower stimulus levels, followed by saturation and a zero-slope or negative-slope region at moderate stimulus levels. Then an additional steeply sloped growth segment was evident at higher stimulus levels. In this case the slope of the lower segment was 0.55 and the slope of the steeper segment above the null was greater than 1.0. DPE threshold estimates for diphasic growth curves utilized the slope of the initial low-level segment. As indicated in Table 2, 26% of the DPE growth curves from normal-hearing ears and 18% from hearing-impaired ears were diphasic.

Figure 3(D) shows an example of a saturating DPE growth curve. These saturating DPE growth curves were characterized by proportionate growth at lower stimulus levels followed by saturation at higher levels. The saturation was evidenced by constant DPE levels over a range of at least 12 dB of stimulus intensity. These saturating DPE growth curves were differentiated from the diphasic growth curves by the lack of a negative slope region at moderate levels and by the lack of a subsequent steeply sloped segment at higher intensities. The DPE threshold estimation was straightforward with only a single slope. In normal-hearing ears, 27% of the curves were saturating growth curves. In the hearing-impaired ears, only 5% were saturating growth curves.

An interesting type of growth curve, not seen as frequently, was the strongly nonmonotonic DPE growth curve. Figure 3(E) shows an example. The nonmonotonic growth curve was characterized by a well defined negative slope region at higher stimulus levels, similar to some of the diphasic curves but without any additional positive sloped segment at higher intensities. In this particular example, an additional flat sloped segment was also seen at low stimulus levels (0.12 dB/dB), and the threshold algorithm utilized that flat sloped region to estimate DPE threshold. In both normal-hearing and hearing-impaired ears, nonmonotonic DPE growth curves accounted for around 7% of the measurable curves.

Figure 3(F) shows an example of a failure to record DPEs that were sufficiently strong to obtain a DPE threshold estimate (No DPEs). In this case, the auditory threshold was within normal limits at 19.3 dB SPL, yet the DPE levels were not above the noise floor except at 66 and 72 dB SPL. The algorithm used to estimate DPE threshold (see Appendix) rejected this function as a valid DPE growth curve. As indicated in Table 2, this outcome occurred in only 7 cases for normal-hearing ears, and only for the 707 and 5656 Hz frequency regions. However, for hearing-impaired ears it was the typical result when auditory thresholds were greater than 50 dB SPL.

The final type of DPE growth curve that was observed is referred to as a two-point DPE growth curve because it was characterized by DPE levels greater than the noise floor at only the highest two stimulus levels, 72 and 78 dB SPL. Examples of two-point DPE growth curves are shown in Figure 4 for different ears at each of six different frequency regions. This type of growth curve was common when DPE threshold estimates could be obtained from ears with auditory thresholds greater than 40 dB SPL. In the examples shown, auditory thresholds ranged from 43 to 69 dB SPL. Notice also in Figure 4 that the slopes of these two-point growth curves ranged from 0.60 up to 2.77. These slopes are steeper than seen in the previous figure. Only one two-point growth curve was observed from the normal-hearing ears. On the other hand, in hearing-impaired ears 19% of the recordable growth curves were two-point curves.

The examples of different types of DPE growth curves in Figure 3 illustrate the problems inherent in attempting to relate measures of DPE level to auditory threshold. At any single stimulus level, DPE level varied considerably across subjects, even though auditory thresholds were normal. For example, consider the variations in DPE level that might occur for a stimulus level of 66 dB SPL if a single-segment growth curve were evident in one subject and a diphasic growth curve were evident for another subject. From the examples seen here, a 20 dB difference in DPE level could easily occur with auditory thresholds that are normal. Given such DPE growth curves, it is understandable that attempts to find strong quantitative correlations between DPE level and auditory thresholds have not been forthcoming.

Correlations Between DPE Thresholds and Auditory Thresholds

As postulated earlier, one of the characteristics of DPE growth curves that might be less sensitive to nonmonotonicities than measures of DPE level is the intercept between the DPE growth curve and the noise floor—the *DPE detection threshold.* To explore that possibility, DPE thresholds were calculated for each of the DPE growth curves obtained from the 32 normal-hearing and the 21 hearing-impaired ears. An algorithm that extrapolated to the noise floor from the slope of each DPE growth curve was used to calculate DPE threshold (see Appendix).

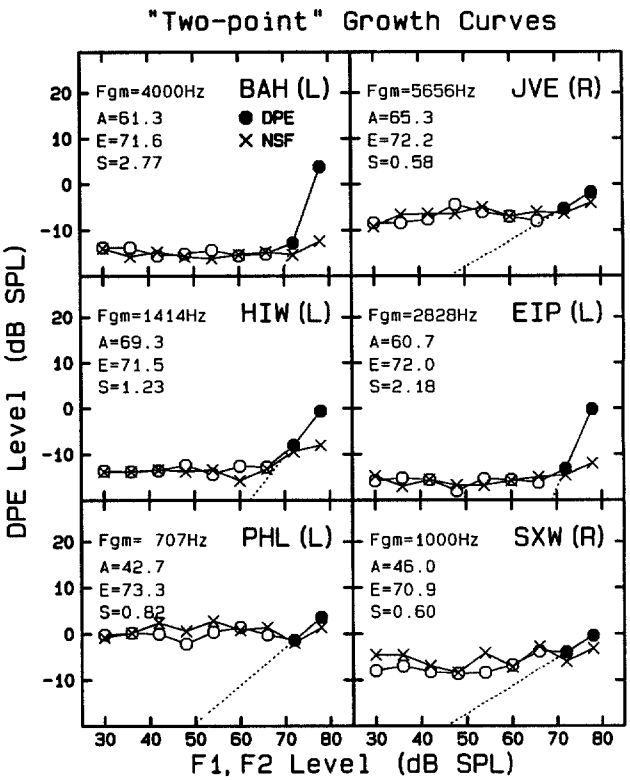

FIGURE 4. Examples of two-point growth curves obtained from ears with elevated auditory thresholds. Legend as in Fig. 3.

DPE thresholds obtained at each frequency region are compared with auditory thresholds in Figure 5. DPE thresholds are shown on the ordinate, and auditory thresholds are shown on the abscissa. Results for different frequency regions are shown in separate panels. Ears from which DPE thresholds could be measured are represented by filled circles for normal-hearing (NH) ears and by open circles for hearing-impaired (HI) ears. Those ears in which DPE thresholds could not be measured at any stimulus level (No DPEs) are indicated by Xs for normal-hearing ears and by open triangles for hearing-impaired ears.

Auditory thresholds from normal-hearing ears were clustered together. The vertical dotted lines at each frequency show the upper limits for normal auditory thresholds, specified here by 2 standard deviations above the group mean at each frequency region (means and standard deviations are given in Table 3). DPE thresholds from these normal-hearing ears, on the other hand, were scattered over a wide range, from below 0 dB SPL to above 60 dB SPL. In 7 cases, no DPE thresholds could be measured for stimulus levels up to

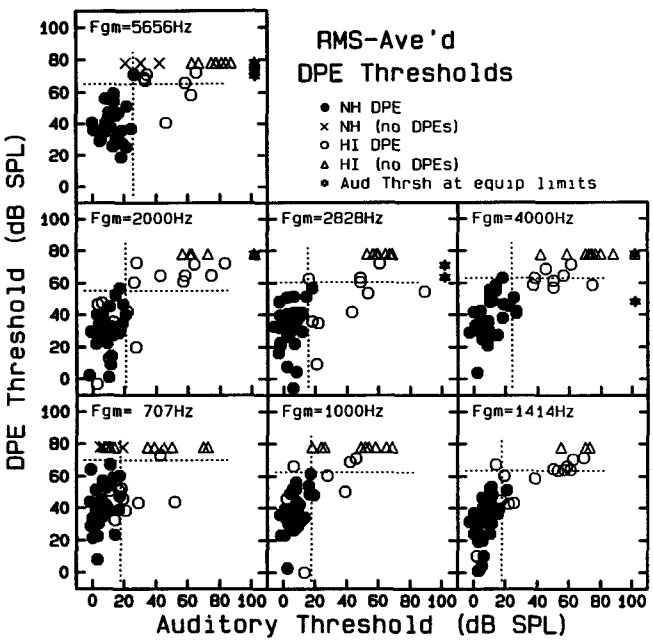

FIGURE 5. Scattergrams of DPE thresholds and auditory thresholds from normal-hearing and hearing-impaired ears. Each panel shows results for a different frequency region. DPE threshold is given on the ordinate and auditory threshold is given on the abscissa. DPE thresholds from normal-hearing ears are shown by filled circles, those from ears with significant hearing losses at one or more test frequencies are shown by unfilled circles. Cases in which no DPE thresholds could be estimated because DPEs were not stronger than the noise floor, at stimulus levels up to 78 dB SPL, are shown by Xs for normal-hearing ears and by triangles for hearing-impaired ears. The stars indicate cases where auditory thresholds were not measurable at the intensity limits of our equipment.

78 dB SPL. Those are shown by the Xs for the frequency regions at 707 and 5656 Hz.

There was a trend toward higher DPE thresholds with higher auditory thresholds in normal-hearing ears, but that trend was not strong. Least-squares linear fits to the normal-hearing data were accomplished to describe any linear relation that might exist between DPE threshold and auditory threshold among normal ears. The results of those fits are given in Table 4. Although there was a positive correlation between DPE threshold and auditory threshold at every test frequency, none of those fits (*r*'s from .29 to .50) accounted for more than 25% of the variance. These results indicate that the variation in DPE threshold among normal-hearing ears is large and is not strongly dependent upon small variations in auditory sensitivity.

Results from the group of 21 ears with elevated auditory thresholds at one or more frequency regions are shown by the unfilled symbols in Figure 5. In this sample of hearing-impaired ears, DPE thresholds did not closely follow auditory thresholds. For auditory thresholds above about 30 dB SPL, most DPE thresholds tended to be above 60 dB SPL, or they were unmeasurable at the testing limits (78 dB SPL). Linear least-squares fits were performed between the auditory threshold and DPE thresholds. Table 4 shows the results of that analysis. A strong quantitative relation between auditory threshold and DPE threshold was not apparent. Correlation coefficients between DPE thresholds and auditory thresholds in hearing-impaired ears ranged from 0.41 to 0.85. At 1414 and 2000 Hz the correlation coefficients were 0.85 and 0.77, accounting for 72% and 60% of the variance, respectively. However, at the other frequency regions the correlations were poorer, accounting for less than 46% of the variance at any one frequency region. These regression analyses included ears from which DPE thresholds could not be obtained (up to the limit of 78 dB SPL). As indicated in Table 2, that occurred in 38% of the tests of ears with significant hearing losses, especially at the higher frequency regions where the highest auditory thresholds existed (55% of the tests at 4000 and 5656 Hz). Without those ears included, the correlations were poorer at all frequency regions.

Noise-Floor Levels

The levels of the noise floors that were used to estimate DPE detection thresholds are shown in Figure 6. Noise-floor levels are plotted as a function of auditory threshold at each frequency region. The range of noise-floor levels from normal-hearing ears is indicated by dotted lines at 2 standard deviations above and below the group mean noise level (means and standard deviations given in Table 3). Noise-floor levels at the lower frequency regions (707 and 1000 Hz) demonstrated larger variability across ears than those for the higher frequencies (standard deviations were about twice as large at these levels as they were at the other five frequencies); this was most likely due to body noise and movement artifact. Noise-floor levels for those cases in which DPE thresholds could not be measured from normal-hearing ears, because the DPE levels were buried in the noise floor (Xs in Figure 6), were all higher than 2 standard deviations above the mean. This suggests that, in those cases, high noise floors may have precluded the measurement of DPE thresholds.

In ears with auditory thresholds greater than 30 dB SPL, over 50% of the noise-floor levels were greater than 2 standard deviation from the mean noise floor obtained from normal-hearing ears. Thus, the high DPE thresholds in hearing-impaired ears may represent measurement limitations as well as the effects of hearing loss. Observations of the raw data records indicated a frequent occurrence of movement artifacts from some subjects who had significant hearing losses. It became apparent that with our ascending series of test levels, subjects with significant hearing loss did not know when we were actually collecting data and became restless with the passage of time. To counter this problem we now indicate recording periods with warning lights.

Slopes of DPE Growth Curves

The slope values obtained by fitting each DPE growth curve with the threshold-estimating algorithm are shown in Figure 7. These are the slope values for the initial lowest-level segment of the DPE growth curves. The average slopes of the DPE growth curves ranged from 0.34 to 0.56 across the seven frequency

TABLE 3. DPE growth-curve parameters and auditory thresholds.

	Frequency region (Hz)						
	707	1000	1414	2000	2828	4000	5656
DPE thresholds: (dB SPL)							
NH *M*	41.7	38.1	31.1	30.4	32.8	38.2	40.9
NH *SD*	14.1	12.3	16.2	12.3	13.8	12.4	12.0
N	28	32	32	32	32	32	29
HI *M*	47.8	45.6	52.0	47.7	47.4	60.8	63.4
HI *SD*	10.4	20.2	17.2	23.1	17.2	8.5	11.0
N	12	12	18	16	12	9	7
Auditory thresholds: (dB SPL)							
NH *M*	6.5	7.0	6.8	9.0	4.4	9.6	12.8
NH *SD*	5.7	5.3	5.4	5.9	5.6	7.3	6.3
HI *M*	20.8	17.5	32.5	33.3	36.9	49.0	47.4
HI *SD*	13.9	16.5	23.3	26.6	24.4	14.3	14.4
Noise-floor levels: (dB SPL)							
NH *M*	−4.2	−8.5	−11.6	−15.0	−15.6	−16.7	−7.6
NH *SD*	2.4	2.4	1.1	0.9	1.1	1.3	1.1
HI *M*	−1.8	−5.3	−9.8	−13.1	−14.1	−15.1	−5.2
HI *SD*	3.4	3.1	2.1	2.0	1.9	1.7	1.9
Growth-curve slopes:							
NH *M*	0.34	0.49	0.55	0.41	0.46	0.56	0.41
NH *SD*	0.15	0.21	0.28	0.22	0.26	0.27	0.24
HI *M*	0.36	0.48	0.69	0.54	0.50	0.66	0.48
HI *SD*	0.19	0.28	0.35	0.41	0.57	0.82	0.34

Note: Parameters and thresholds are from ears with normal-hearing (NH) and from ears with significant hearing loss at one or more frequency regions (HI). Data include only those cases in which valid DPE thresholds could be obtained. The values of N given for DPE thresholds applies to the other measures as well.

regions. Those slopes varied considerably across ears, from near zero to greater than unity in some ears. In Figure 7, the dotted horizontal lines indicate 2 standard deviations above the mean slope values from normal-hearing ears (means and standard deviations given in Table 3).

There did not appear to be any consistent differences in slope values obtained from normal-hearing ears and those obtained from ears with elevated auditory thresholds (see Table 3). The average slope values for the two groups were 0.53 and 0.46 dB/dB, respectively. Of those ears with auditory thresholds greater than 30 dB SPL, only about 15% of them demonstrated slopes that were steeper than the confidence limits for slopes from normal-hearing ears as shown by the dotted lines in Figure 7.

TABLE 4. Correlations between DPE detection thresholds (y) and auditory thresholds (x).

	Frequency region (Hz)						
	707	1000	1414	2000	2828	4000	5656
Normal-hearing ears $n = 32$							
Slope (a)	1.19	1.15	1.08	0.78	0.71	0.84	0.92
Intercept (b)	40.0	30.0	23.7	23.4	29.7	30.1	31.0
R	0.39	0.49	0.36	0.38	0.29	0.50	0.50
Hearing-impaired ears $n = 21$							
Slope (a)	0.39	0.68	0.63	0.63	0.44	0.21	0.20
Intercept (b)	49.6	39.3	32.3	28.3	36.9	55.4	57.9
R	0.47	0.68	0.85	0.77	0.62	0.41	0.50
Hearing-impaired and normal-hearing ears $n = 53$							
Slope (a)	0.56	0.80	0.73	0.68	0.53	0.50	0.44
Intercept (b)	43.4	33.7	27.1	25.0	31.2	34.6	38.8
R	0.50	0.74	0.77	0.79	0.74	0.81	0.76

Note: Correlations use a linear regression equation: $y = ax + b$. Slope (a) and Intercept (b) are indicated for each frequency region. *R* is the correlation coefficient. *N* is the number of ears in each group.

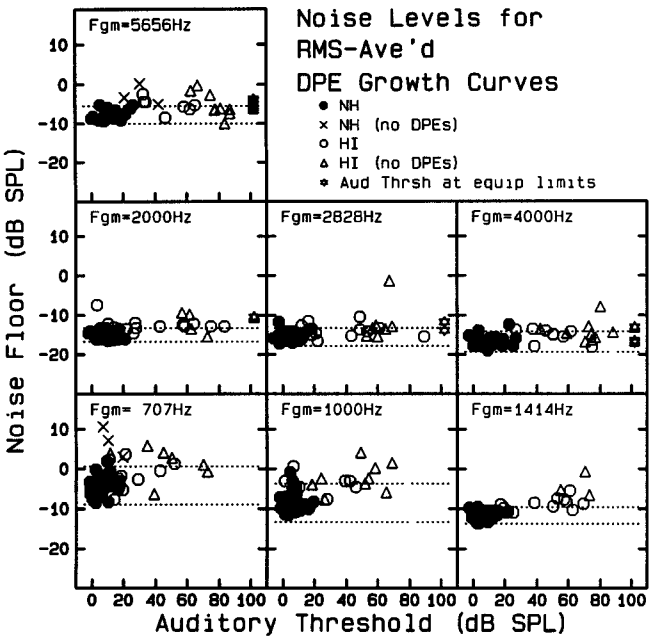

FIGURE 6. Scattergrams of noise levels and auditory thresholds from normal-hearing and hearing-impaired ears. Each panel shows results for different frequency regions. Dotted lines are plotted at 2 standard deviations above and below the mean noise floor for normal-hearing ears.

Discussion

Cochlear Mechanisms Underlying the DPE Growth Curve

The different types of growth curves observed here from individual ears are not unique. Examples of nonmonotonic and irregularly shaped growth curves have been reported from both animals and humans. Diphasic DPE growth curves, with evidence of at least two segments to the curves, have been obtained for equal-level primaries from normal ears of cats (Kim, Molnar, & Matthews, 1980, Fig. 14), from normal ears of chinchillas (Zurek, Clark, & Kim, 1982, e.g., Fig. 7), from normal ears of guinea pigs (Brown, McDowell, & Forge, 1989, Fig. 2), from normal ears of rabbits (Whitehead, Lonsbury-Martin, & Martin, 1990, Fig. 2), and from the normal adult ears of a gerbil (Norton, Bargones, & Rubel, 1991, Fig. 3). Saturating DPE growth curves for equal-level primaries have been seen in normal human ears (Gaskill & Brown, 1990, Fig. 4; Lonsbury-Martin & Martin, 1990, Fig. 2; Wier, Pasanen, & McFadden, 1988, Fig. 5). Individual DPE growth curves with *notches* or *bend over* have been reported from normal human ears by Lonsbury-Martin et al. (1990, Fig. 10), and nonmonotonic DPE growth curves have been reported by Wier et al. (1988, Fig. 6). Until now, it was not apparent that nonmonotonic and irregularly shaped growth curves were quite so prevalent in normal-hearing human ears (72%).

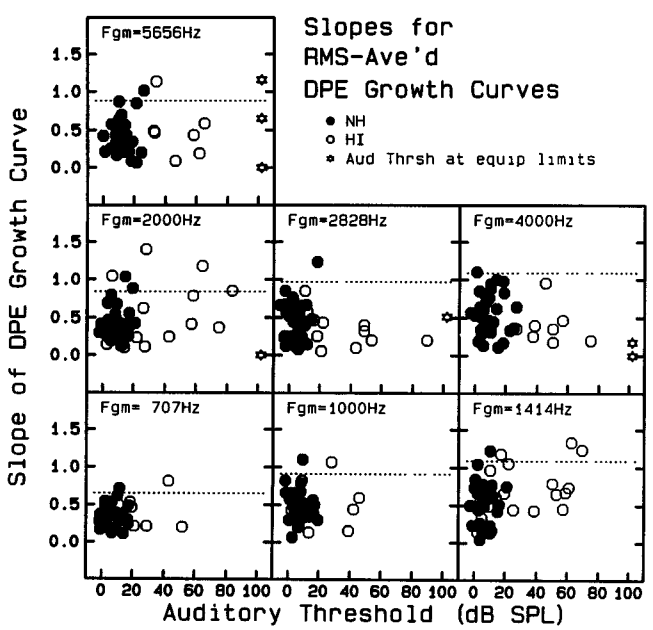

FIGURE 7. Scattergrams of slope values obtained from the DPE growth curves of normal-hearing and hearing-impaired ears. Dotted lines are drawn at 2 standard deviations above the mean noise floor for normal-hearing ears.

One possible explanation for some of the irregularly shaped curves seen here (diphasic, nonmonotonic, saturating) may be the existence of *spontaneous otoacoustic emissions* (SOAEs) at frequencies close to the DPE frequency. The nonmonotonic DPE growth curves from one ear reported by Wier et al. (1988) were only 20 Hz below a sizable SOAE (14 dB SPL), and the two irregularly shaped curves reported by Lonsbury-Martin et al. (1990) were from an ear with many recordable SOAEs. It is conceivable that DPEs may interact with SOAEs in such a way that at moderate levels the two partially cancel each other to produce a null or notch in the DPE growth curve. However, results from four individual ears reported by Wier et al. (1988) suggest that SOAEs tend to enhance the growth rate of DPEs only at low levels of the primaries. At moderate levels, DPEs appear to suppress the SOAE and either continue to grow with stimulus level or saturate. Unfortunately, the data-collection procedure used in this study did not include rigorous SOAE measurements on each ear. The graphic record of the response spectrum was monitored during data collection for obvious SOAEs, but no objective data were saved for later analysis. The exact effects of SOAEs on different growth patterns remain to be determined.

The irregularly shaped DPE growth curves seen here, specifically the diphasic curves shown in Figure 3(C) and the nonmonotonic curves shown in Figure 3(E), may also result from interactions between the cubic distortion product and other propogating distortion products at the $2f_1 - f_2$ place on the basilar membrane. Matthews and Molnar (1985), in their modeling of intracochlear and ear canal distortion products, have suggested that interactions between the $f_2 - f_1$ distortion component and the $2f_1 - f_2$ distortion component, at the $2f_1 - f_2$ place, might explain some of the notches seen in the DPE growth curve at higher levels. That interaction would require exact amplitude and phase relations between components for cancellation to occur, and it would also vary with f_2/f_1 ratio, so that the notching in the growth curve may or may not be seen in any specific ear or test frequency.

Another explanation for irregularly shaped DPE growth curves attributes those shapes to two underlying cochlear mechanisms (Manley, Koppl, & Johnstone, 1990; Norton & Rubel, 1990; Whitehead, Lonsbury-Martin, & Martin, 1990). One, vulnerable to ototoxicity, is associated with low-level stimuli and is thought to involve functional outer hair cells; the other, not as vulnerable, is associated with high-level stimuli and may not involve active participation by outer hair cells. A phenomenological model of the development of the DPE, $2f_1 - f_2$, in the gerbil (Norton et al., 1991), promises to provide some insight into the possible origin of the diverse types of DPE growth curves observed here. That model presumes a passive *nonlinearity* associated with the basilar membrane inner-hair-cell subsystem and at least one (maybe two) active *non-linearities* associated with the basilar membrane outer-hair-cell/tectorial-membrane subsystem. Output from the active nonlinearity begins to appear at low stimulus levels (less than 20 dB SPL). It then grows with a slope less than unity until it saturates at moderate stimulus levels (50–60 dB SPL). Output from the passive nonlinearity does not begin to appear until moderate levels of the primaries (40–50 dB SPL) are reached, and then it grows up to the highest stimulus levels with a slope near or exceeding unity. The model presumes that these two nonlinearities sum (vectorally) to produce a complex function similar to what has been referred to here as a diphasic DPE growth curve (see Norton et al, 1991, Fig. 11 for examples).

In support of this model, Norton et al. (1991) showed that the low-level saturating segment of the adult gerbil DPE growth curve disappeared quickly after death, leaving the high-level steeply sloped segment that presumably reflects the passive nonlinearities of the cochlear partition (see also Schmiedt & Adams, 1981). The higher level steeply sloped segment remained for at least an hour after death. They also found that the low-level active segment took longer to develop in the gerbil, as do outer hair cells and the tectorial membrane in other species (Pujol, Carlier, & Lenoir, 1980; Lenoir & Puel, 1987). Additional evidence supporting this model comes from the results of Brown, McDowell, and Forge (1989). In gentamycin treated guinea pigs with slight outer-hair-cell pathology, they found that the low-level saturating segment of the DPE growth curve disappeared, leaving the high-level steeply sloped segment. More recently Mills, Norton, and Rubel (1992) demonstrated in the gerbil that the low-level segment (DPEs for primaries < 55 dB SPL) covaried with the endolymphatic potential following furosemide injection. Both measures dropped quickly following the injection and recovered within 5 to 6 hours. By contrast, the high-level segment (DPEs for primaries > 70 dB SPL) remained constant after the injection. Whitehead, Lonsbury-Martin, and Martin (1992), using ethacrynic acid in a rabbit, reported similar reductions in the low-level segment of the DPE growth curve while the high-level segment remained relatively robust.

This general type of model was proposed in some detail by Zwicker (1979), and is entirely consistent with a model of basilar membrane motion described recently by Patuzzi, Yates, and Johnstone (1989). Their model, supported by basilar-membrane vibration measurements using the Mossbauer technique (Patuzzi, Johnstone, & Sellick, 1984; Robles, Ruggero, & Rich, 1986; Sellick, Patuzzi, & Johnstone, 1982), attributed the low-level saturating portion of the basilar membrane input-output curve to the active gain mechanism in the outer-hair-cell subsystem. The higher level portion of the basilar membrane input-output curve was attributed to the cochlear partition and inner-hair-cell system.

Most recently, Robles et al. (1991) recorded $2f_1 - f_2$ distortion directly from the basilar membrane of chinchillas using a linear laser-velocimetry technique. At very low levels of the equal-level primary tones, basilar membrane distortion was between 11 and 24 dB below the level of the primaries. For levels of the primaries between 30 and 50 dB SPL, basilar membrane distortion grew with slopes around 0.67. Saturation began between 50 and 70 dB SPL. Those growth curve characteristics for basilar membrane distortion are nearly identical to the growth curve characteristics seen here in humans for low-level segments of DPE growth curves, except that the largest DPE levels were about 40 dB below the level of each primary (for example, at Fgm = 1414 and 4000 Hz in Figure 1). Besides any species differences, this difference of 20–30 dB in relative distortion levels between basilar membrane measures (in chinchilla) and ear canal measures (in human) is not surprising considering the attenuation to the distortion components that must occur as they travel from the basilar membrane outward into the ear canal.

With this in mind, the different types of growth curves observed here might be interpreted as various outcomes of interactions between the active and passive nonlinearities along the cochlear partition. At low levels of the primary tones, the outputs from the active nonlinearities predominate, which then saturate at moderate levels, leaving the output from the steep-sloped passive nonlinearity. With different phases and magnitudes between the two segments, all five types of DPE growth curves seen here could easily result from vector summation within the cochlea or ear canal. Following this reasoning, evidence of a low-level segment in a DPE growth curve seems to indicate activity of the outer-hair-cell subsystem. Furthermore, the higher level steeply sloped segment of a DPE growth curve might indicate activity from the inner-hair-cell subsystem.

Table 2 indicates that a large number of DPE growth curves from the normal-hearing ears examined here demonstrated evidence of a low-level segment (72% of the normal curves were flat-steep, diphasic, saturating, or nonmonotonic). By contrast, a smaller number of the ears with significant hearing loss showed evidence of the low-level segments (38%). If only 4000 and 5656 Hz frequency regions are considered, where all of the hearing-impaired ears demonstrated large hearing losses, only 3 curves (7%) exhibited evidence of the flatter-sloped lower level segment. And in two of those curves a two-sloped (flat-steep) curve was evident, with the steep-sloped segment most prominent. In the majority of the hearing-impaired ears at 4000 and 5656 Hz (93%), either single-segment monotonic or two-point growth curves were obtained, or no DPEs could be measured. The single-segment and two-point growth curves were for higher levels of the primaries and had slopes that approached or exceeded unity.

This suggests that when DPE growth curves with slopes near 1.0 can be recorded from ears with large hearing losses,

those curves probably reflect the higher level passive nonlinearity of the inner-hair-cell subsystem. It also suggests that the presence of low-level DPEs exhibiting diphasic, saturating, or nonmonotonic shapes may indicate relatively normal functioning of the outer-hair-cell subsystem. These conjectures are, of course, only preliminary. Further research in both animals and humans is needed to test such suppositions. When they do, the shapes of DPE growth curves might differentiate micromechanical dysfunctions in human cochleas, which could prove invaluable in the clinical setting for preventing and treating cochlear hearing losses. On the other hand, the possible effects of SOAEs on DPE growth curves, and potential interactions between multiple propagating waves in the cochlea must not be ignored.

Correlations Between DPE Threshold and Auditory Threshold

With regard to predicting auditory threshold from measures of DPE threshold, the correlations given in Table 4 are disappointing. The strongest correlations were seen when both normal-hearing and hearing-impaired ears were combined. Those correlations ranged between 0.50 and 0.81, with an average of 0.73 across test frequency. When only the hearing-impaired ears were considered, correlations were lower, ranging between 0.41 and 0.85 at different frequencies, with an average of 0.61. Lonsbury-Martin and Martin (1990) reported correlation coefficients of 0.52, 0.69, 0.85, 0.70, 0.69, and 0.77 at test frequencies of 1, 2, 3, 4, 6, and 8 kHz, respectively. Their correlations for 16 hearing-impaired ears are somewhat higher than those observed in the present study, probably because they used a maximum limit for the primary tone levels of 85 dB SPL. The limit of 78 dB SPL used here essentially reduced the spread of the data on the x axis, possibly limiting the magnitude of the correlations. Furthermore, the data points at 78 dB SPL are not valid DPE thresholds; they are simply the intensity limits we set for our testing procedure. If we had tested with higher primary tone levels, say to 85 dB SPL, DPEs might have been observed and higher correlations may have resulted.

Also, the sample of 16 noise-induced ears investigated by Lonsbury-Martin and Martin (1990) was more restricted than ours in terms of etiology. In a later study, Ohlms, Lonsbury-Martin, and Martin (1991) found a correlation as high as 0.95 for a restricted group of ears with hereditary hearing loss. Thus, the selection of patients with etiologies likely to produce purely outer-hair-cell pathologies will probably result in stronger correlations between DPE threshold and hearing loss, and it is possible that the variety of etiologies included in the present study contributed to poorer correlations.

Nevertheless, judging from the scattergrams in Figure 5, there does not appear to be a sufficiently orderly relation between auditory threshold and DPE threshold to use these results to predict one from the other with precision.

The major aspect of the results that limits correlations is the spread of DPE thresholds seen among our normal-hearing ears. This is evident in the scatter of the filled circles in Figure 5. The range of DPE thresholds from normal-hearing ears was over 60 dB, from below 0 dB SPL to above 60 dB SPL. The scatter was less in the results reported by Leonard et al. (1990). Their DPE thresholds ranged between 30 and 60 dB SPL. Lonsbury-Martin and Martin (1990) reported the upper 95% confidence limit for DPE thresholds from their normal-hearing ears was 63 dB SPL. From this it appears that our range of DPE thresholds, broader than in previous studies, was due to the occurrence of lower DPE thresholds, most likely a direct result of using a more sensitive threshold estimation algorithm that relied partially upon the slope of the DPE growth curve. This type of algorithm could lead to artificially low estimates of DPE threshold, because as DPE levels approach the noise floor, the noise contributes more and more to the measured amplitude at the DPE frequency, and the slope of the growth curve will be reduced by the noise floor. With the present algorithm, more gradual slopes could lead to lower extrapolated DPE thresholds. This is illustrated in Figure 3(E), where the slope of 0.12 dB/dB leads to a DPE threshold of −2.0 dB SPL. Whether the more sensitive DPE thresholds seen here were artifacts of the slope-dependent threshold algorithm or were true indications of the lowest primary levels that would elicit a DPE is not clear. Further research may answer this question if noise levels can be reduced appreciably.

There appear to be several other factors that might contribute to the wide scatter of DPE thresholds seen in normal-hearing ears. One factor may be the potential effect SOAEs might have on low-level DPEs. It is well documented that SOAEs in the vicinity of DPE frequencies enhance DPE levels (Burns, Strickland, Tubis, & Jones, 1984; Kemp, 1979; Kemp & Brown, 1983; Wier et al., 1988; Wilson, 1980a, b). Although the detailed effects of SOAEs on DPE growth curves have not been studied extensively, the data from Wier et al. (1988), which were obtained at two levels of the primaries at multiple DPE frequencies around the SOAE frequency, show that DPE levels rose up to 25 dB higher when a recordable SOAE was 10–20 Hz higher in frequency than the DPE frequency. The presence of a SOAE near the DPE frequency could account for high DPE levels with low-level primaries and possibly lead to very low DPE thresholds. This might explain why DPE thresholds obtained in the present study were lower than those reported by Leonard et al. (1990). Careful examination of the graphs from Wier et al. (1988), indicates that SOAEs might also be associated with negative slopes in DPE growth curves, e.g., nonmonotonic curves. However, that observation is based upon comparisons of responses for only two levels of the primary tones. Clearly, more research is needed to determine how SOAEs influence the shapes of DPE growth curves.

Another factor that might contribute to the wide scatter of DPE thresholds seen in normal ears is the spatial origin along the basilar membrane of the two types of threshold measurements. In normal-hearing ears, the auditory threshold is elicited by a single tone with very low intensity (−10 to 20 dB SPL), which excites a very narrow region of the basilar membrane. In the present study, that tone was placed at the geometric mean frequency between the two primary tones used to elicit DPEs. By contrast the DPE threshold is elicited by two tones, each with intensities that are greater than 30 dB SPL. Those two tones excite a wider region of the basilar membrane than the tone used to obtain auditory threshold.

Therefore, the exact place of origin of the DPEs and the place of excitation for auditory threshold may be different.

Suppression and adaptation experiments indicate that the region of the basilar membrane most responsible for generation of DPEs is the region excited by the two primary tones (Brown & Kemp, 1984; Dolan & Abbas, 1985; Lonsbury-Martin, Martin, Probst, & Coats, 1987). However, at the levels of the primaries above 60 dB SPL that are required to elicit a DPE in some normal-hearing ears, the region of the basilar membrane excited by the two primaries may be quite broad. Differences in the microstructure of auditory sensitivity, and in the power of the DPE generators at different local places along the basilar membrane, might vary considerably, and thereby lead to some of the scatter we have observed in DPE thresholds.

In addition, the locale responsible for emission generation may vary with the level of the primaries. There is some indication in the results reported about rabbits by Lonsbury-Martin et al. (1987) that the frequency at which the most interference occurs from a third tone moves closer to the frequency of the f_2 primary as stimulus levels are reduced from 65 to 45 dB SPL. To obtain low DPE thresholds, we are interested primarily in emissions elicited by low-level stimuli, below 45 dB SPL if possible, which would presumably reflect output from the low-level segment of the growth curve related to the active nonlinearities of the outer-hair-cell subsystem. Further research might reveal that the place of generation for that low-level segment is not the same as it is for the higher level steeply sloped segment. In that case the measurement of auditory thresholds at a frequency region midway between the two primaries may not provide for the best correlations with DPE threshold.

Although the present recording procedure adjusted the input sound pressure to achieve a constant sound pressure at the probe microphone, this may not be the best procedure to ensure constant sound pressure across the ear drum itself. Changes in positioning of the probe inside the ear canal can lead to differences in microphone sound pressure, which might not be indicative of the effective sound pressure across the eardrum. Some of the variance among DPE thresholds in normal-hearing ears might be due to probe positioning differences among ears. Future procedures that involve determinations of the acoustic impedance of the middle ear might reduce some of the variance across ears.

Another interpretation of this wide scatter might attribute the higher level DPE thresholds among normal-hearing ears to micromechanical transduction differences within the cochlea, which may not be reflected in auditory threshold measurements. This might include differences among normal ears in the generating mechanisms for DPEs along the basilar membrane. It might also involve differences in the transmission process of acoustic energy from the inner ear to the outer ear.

For example, to obtain auditory thresholds, researchers measure the level of the input required to elicit an auditory sensation, and small differences among ears in the forward transmission of acoustic energy into the inner ear should be reflected in auditory thresholds. On the other hand, measurement of DPE thresholds involves transmission in both the forward and reverse directions. Small differences among ears encountered in forward transmission will be encountered again during reverse transmission of the DPE out of the inner ear. Consequently, one might expect that small differences in transmission efficiency among ears for forward transmission would be doubled for both forward and reverse transmission. Table 3 indicates that the average standard deviation across frequency for auditory thresholds in normal ears was about 6 dB, while the average standard deviation across frequency for DPE thresholds was about 13 dB. The variability among ears was about double for DPE thresholds over what it was for auditory thresholds. This increased variability among ears for DPE thresholds, compared with auditory thresholds, is consistent with the idea that DPE thresholds reflect both forward and reverse transmission.

The bidirectional transmission nature of the DPE threshold procedure should also affect the sensitivity of the DPE threshold measurement. Theoretically, if the DPE threshold represents the minimum acoustic input that elicits excitation along the basilar membrane, then the DPE threshold should be higher than the auditory threshold by at least the amount of the additional reverse transmission loss encountered by the DPE as it travels back out to the ear canal. Comparisons of relative DPE levels in humans and animals might provide some insight into the magnitude of that reverse transmission loss. The largest DPEs recorded from subjects in the present study was around 40 dB below the primaries. This was shown in Figure 1 for DPEs at 1414 and 4000 Hz for subject DJF(L). DPE levels were around − 10 dB SPL, with primaries at 30 dB SPL. By comparison, relative DPE levels on the basilar membrane from the chinchilla were reported by Robles et al. (1991) to be between 11 and 24 dB below the primaries. Ignoring other possible differences between species, this represents a difference of about 20–30 dB between relative DPE levels measured in the ear canal and those measured on the basilar membrane. From this one might speculate that reverse transmission loss might be on the order of 20–30 dB. If so, then one might expect that the DPE threshold, which is subject to reverse transmission, would be around 20–30 dB higher than auditory threshold, which is not subject to reverse transmission.

Examination of the group mean DPE thresholds and auditory thresholds in Table 3 for normal-hearing ears tends to support that expectation. For example, at 2000, 2828, and 4000 Hz, the frequency regions where the noise floors are the lowest and probably not limiting measurements of DPE levels, the average DPE threshold across frequency was about 34 dB SPL. By comparison, the average auditory threshold across those three frequencies was about 8 dB SPL. The DPE threshold was some 26 dB less sensitive, which is consistent with a 20–30 dB reverse transmission loss. This type of reasoning, although highly speculative, suggests that DPE thresholds from normal hearing ears should be around 30+ dB SPL and that they should also be dispersed by about twice the dispersion of auditory thresholds. Both these ideas are consistent with the results reported here.

Slopes of the DPE Growth Curves

The slopes of the DPE growth curves near the DPE threshold are shown in Figure 7. They varied over a large range, from near 0 to steeper than 1.0 for some growth

curves. Table 3 shows that the mean slope varied from 0.34 to 0.69 across the seven frequency regions tested. These slopes were flatter than those reported for humans and animals by most other investigators.

In human ears, Lonsbury-Martin et al. (1990) found slopes ranging from 0.40 at 1000 Hz (Fgm) up to near 1.0 at 6000 Hz, a clear increase in slope with frequency. Harris (1990) reported slopes ranging from 0.83 at 750 Hz up to 1.32 at 6000 Hz. Leonard et al. (1990) report an average slope around 0.50. Gaskill and Brown (1990) reported a mean slope of 0.91 across frequency for DPE growth curves obtained with equal-level primaries from two subjects who displayed large emissions. DPE growth curves from animals have largely been more robust than from humans, with large amplitudes and steeper slopes (Brown & Gaskill, 1990). Slopes of DPE growth curves for equal-level primaries in animals have been reported to be near 1.0 for low primary levels (Brown, 1987; Brown & Gaskill, 1990; Fahey & Allen, 1986; Kim, 1980; Lonsbury-Martin et al., 1987).

We suspect the reason the slopes of the present study are flatter than those obtained from human ears by other investigators is due to the sensitivity of our DPE threshold estimation procedure. The algorithm was designed to estimate the lowest possible DPE thresholds using the slope of the DPE growth curve near the noise floor to extrapolate to the average noise floor. However, as DPE levels approach the noise floor, the noise should influence the DPE measurement more and more, making the slope of the growth curve more gradual. Therefore, slopes determined as part of the threshold algorithm did not reflect the steepest sloped segments of the DPE growth curves. As mentioned earlier, when two segments existed, the higher level, steeply sloped segment often had a slope close to or steeper than 1.0. Other investigators used a much more straightforward threshold estimation procedure that reported DPE threshold as the lowest stimulus level that elicited a DPE that was 2 dB or 3 dB above the noise floor (Leonard et al., 1990; Lonsbury-Martin & Martin, 1990). Their slopes generally specified the steepest segment of the growth curve. Thus, for the higher level steeply sloped segments, the present results tend to agree with previous findings regarding slopes of DPE growth curves.

The gradual slopes of the DPE growth curves measured here suggest that, in certain cases, a large portion of the growth curve may have been below the noise floors obtainable with the present recording procedure. The noise floor may have prevented us from recording the lower level, steep-sloped segments of the growth curve. Norton et al.'s (1991) phenomenological model of an active low-level nonlinearity and a passive higher level nonlinearity served as a conceptual guide for our reasoning. From direct measures of basilar membrane distortion obtained by Robles et al. (1991), we expected to find DPE growth curves with slopes for the low-level segment around 0.67, which presumably reflects compressive nonlinearities from the outer-hair-cell subsystem. At higher levels, say above 70 dB SPL, we expected a slope near or exceeding 1.0, which would probably reflect the response of the cochlear partition and inner-hair-cell subsystem. In the region where the low-level active nonlinearity predominates and is in its saturation region, we expected very gradual slopes. Since that saturation region presumably occurs at moderate stimulus levels, we expected to find growth-curve slopes around 0.67 at very low stimulus levels, between 30 and 50 dB SPL. As indicated in Table 3 the slopes we obtained near DPE threshold from normal ears were well below 0.67. The mean slope across frequency was only 0.46 dB/dB, with some slopes as low as 0.12 dB/dB—Figure 3(E). This suggested to us that the threshold estimates obtained with gradually sloped DPE growth curves may have been from the saturating region of the active nonlinearity. This is illustrated in Figure 3(B) and 3(C) by the flat-steep and diphasic DPE growth curves. If lower noise floors could have been achieved, perhaps DPEs could have been recorded with stimulus levels well below 30 dB SPL. Furthermore, if the noise floor had limited our recording of the low-level segment that reflects the active nonlinearity, lowering the noise floor should have revealed DPE growth curves with slopes closer to 0.67.

This does not suggest that the procedures utilized here yielded higher noise floors than reported by other investigators. As shown in Table 3, average noise floors ranged from −4 dB SPL at 707 Hz down to −17 dB SPL at 4000 Hz, and then rose to −8 dB SPL at 5656 Hz. Both the absolute noise-floor levels and the frequency dependencies were similar to those reported by other investigators using similar instrumentation and procedures (Harris et al., 1989; Leonard et al., 1990; Lonsbury-Martin & Martin, 1990; Norton et al., 1991). The noise floor limits are comparable across studies and are due to the physics of the recording conditions. Unfortunately, with the procedures used here lower noise floors were not possible.

Another characteristic observed in many of the DPE growth curves from the present study was a high-level, steeply sloped segment with slopes near or exceeding 1.0. This was illustrated in Figure 3(B) and 3(C) by the flat-steep and diphasic growth curves. It was also illustrated in Figure 4 by the two-point growth curves seen at high stimulus levels. The occurrence of high-level, steeply sloped segments, together with more gradually sloped low-level segments, leads us to wonder if DPE growth curves with steep slopes (near or exceeding 1.0) at moderate and high stimulus levels might reflect only the passive nonlinearity of the human basilar membrane. If this is true, then many of the DPE thresholds obtained from hearing-impaired ears at high levels of the primaries, those that are derived from two-point growth curves, may reflect activity of the passive nonlinearity of the inner-hair-cell subsystem without any significant participation from inner hair cells.

From this discussion it appears that the relation between DPE threshold and auditory threshold might be much more complex than implied by a simple linear correlation between the two measures. The shape or pattern of the DPE growth curve may become more useful for differentiating underlying cochlear function than are DPE threshold measures for estimating auditory thresholds. It is clear that more research is required to resolve these issues.

DPE Thresholds for Screening Purposes

The lack of a strong linear correlation between DPE threshold and auditory threshold does not allow an accurate

TABLE 5. Contingency matrices for predicting abnormal auditory thresholds from DPE detection threshold measurements.

Study	DPE Threshold	
	Abnormal	**Normal**
Nelson & Kimberly (1992)		
Auditory threshold	(>*M* + 2*SD*)	(<*M* + 2*SD*)
Abnormal (>*M* + 2*SD*)	0.78 (95)	0.22 (27)
Normal (<*M* + 2*SD*)	0.16 (5)	0.84 (27)
Correct prediction	0.79 (122)	
Martin, Ohlms, Franklin, Harris, & Lonsbury-Martin (1990)		
Auditory threshold	(>63 dB SPL)	(<63 dB SPL)
Abnormal (>30 dB HL)	0.80 (40)	0.20 (10)
Normal (<30 dB HL)	0.09 (3)	0.91 (31)
Correct prediction	0.82 (71)	
Leonard, Smurzynski, Jung, & Kim (1990)		
Auditory threshold	(>60 dB SPL)	(<60 dB SPL)
Abnormal (>20 dB HL)	0.92 (12)	0.07 (1)
Normal (<20 dB HL)	0.03 (1)	0.97 (38)
Correct prediction	0.96 (50)	

Note: Entries are proportions with total numbers of cases in parentheses.

prediction of auditory thresholds from DPE thresholds. However, it does appear that a sufficient dichotomy exists between DPE thresholds from normal and impaired ears to predict the likelihood of an auditory threshold being abnormal at some frequency region from the determination of a DPE threshold at that frequency region. The accuracy of such a prediction is examined below in light of the results of the present study and two previous studies.

Table 5 contains contingency matrices for predicting auditory thresholds from DPE thresholds—the occurrence of abnormal and normal auditory thresholds is shown, contingent upon abnormal and normal DPE thresholds. For the results from the present study, abnormal DPE threshold was defined as a DPE threshold larger than two standard deviations above the mean (> *M* + 2 *SD*) for normal-hearing ears. Similarly, abnormal auditory threshold was defined as an auditory threshold larger than two standard deviations above the normal mean (> *M* + 2 *SD*). Given these dichotomies, one can then examine the likelihood of making a correct decision about auditory threshold being abnormal or not on the basis of whether the DPE threshold was abnormal or not.

In such a 2 × 2 contingency matrix there are two types of correct decisions and two types of incorrect decisions. The upper left cell of the matrix indicates the likelihood of correctly deciding that auditory threshold was abnormal, given an abnormal DPE threshold (hit). The frequency of occurrence is shown in parentheses. The lower right cell contains the likelihood of correctly deciding that auditory threshold was normal, given a normal DPE threshold (correct rejection). Together these yield the likelihood of making a correct prediction. The lower left cell of the matrix shows the likelihood of incorrectly deciding that the auditory threshold is abnormal, when indeed it is normal (false alarm). This is often called the "likelihood of a false positive for hearing loss." The upper right cell of the matrix shows the likelihood of incorrectly deciding that the auditory threshold is normal, when indeed it is abnormal (miss). This is often called the "likelihood of a false negative for hearing loss." Together, the false positives and false negatives yield an overall error rate.

The contingency matrix for the results from the present study includes DPE thresholds and auditory thresholds from 21 hearing-impaired ears at 7 frequency regions, for a total of 147 cases. As indicated in Table 5, the likelihood of correctly predicting abnormal auditory threshold from an abnormal DPE threshold was 0.78, and the likelihood of predicting a normal auditory threshold from a normal DPE threshold was 0.84. Since both of these are correct predictions, the likelihood of correct predictions with the specified decision criteria was 0.79. That is, 79% of the predictions were correct ones. The matrix also shows that the likelihood of a false positive was 0.16 and the likelihood of a false negative was 0.22. Together the likelihood of making an incorrect decision was 0.21. That is, 21% of the predictions about auditory threshold, made on the basis of DPE threshold measurements, would probably be made in error.

The contingency matrix for the results reported by Martin et al. (1990) is shown in the middle of Table 5. Their data were from 15 ears with noise-induced hearing losses. Abnormal DPE threshold was defined as a DPE threshold higher than 63 dB SPL, which was the upper value of their 95% confidence limit reported for normal ears. Abnormal auditory threshold was defined as an auditory threshold above 30 dB HL, which is slightly less stringent than the threshold used with the contingency matrix for the present study. In this case, 85% of the predictions were correct.

At the bottom of Table 5 is the contingency matrix for results reported by Leonard et al. (1990). Here the criteria for abnormal DPE thresholds and abnormal auditory thresholds were somewhat arbitrary. We set the criterion for abnormal DPE threshold at 60 dB SPL and the criterion for abnormal auditory threshold at 20 dB HL. As seen in Table 5, with these criteria the likelihood of making correct predictions was 0.96.

From the likelihoods in these types of contingency matrices, one can determine whether or not the predictions of hearing loss from DPE thresholds will be accurate enough to use DPE thresholds as a screening procedure for hearing loss. Of course, the relative amount of effort required for each type of screening procedure must also be considered before one can determine whether to implement one screening procedure or another.

At this stage of development, determining DPE thresholds seems promising for hearing screening purposes, but quantitatively predicting auditory threshold from DPE threshold (or DPE level) is, at this time, an inaccurate procedure. The results reported here indicate that DPE growth curves from normal-hearing and hearing-impaired ears may provide insight into the mechanisms of normal and abnormal cochlear function. The wide variety of shapes of DPE growth curves suggest that a complex set of cochlear subsystems underlie the curves. As we understand the outputs from those subsystems better, DPE growth curves may provide us with

procedures for evaluating them. We also need to find ways of recording DPEs elicited by lower level primaries so that the complete low-level segment of the active nonlinearity of the cochlea can be studied more rigorously.

Acknowledgments

This research was supported in part by grants from the NIDCD, the American Otologic Society, and the Lions 5M International Hearing Center. The authors wish to thank Brenda Lonsbury-Martin, Martin Whitehead, and Anna Schroder for generous comments on earlier versions of this manuscript. Special appreciation goes to John Van Essen, whose programming talents made this project possible, and to Nancy Markman and Anna Schroder for their assistance during data analysis.

References

American National Standards Institute. (1969). *Specifications for audiometers* (ANSI S3.6-1969). New York: ANSI.

Brown, A. M. (1987). Acoustic distortion from rodent ears: A comparison of responses from rats, guinea pigs and gerbils. *Hearing Research, 31,* 25–38.

Brown, A. M., & Gaskill, S. A. (1990). Measurement of acoustic distortion reveals underlying similarities between human and rodent mechanical responses. *Journal of the Acoustical Society of America, 88,* 840–849.

Brown, A. M., & Kemp, D. T. (1984). Suppressibility of the $2f_1 - f_2$ stimulated otoacoustic emissions in gerbil and man. *Hearing Research, 13,* 29–37.

Brown, A. M., McDowell, B., & Forge, A. (1989). Acoustic distortion products can be used to monitor the effects of chronic gentamicin treatment. *Hearing Research, 42,* 143–156.

Burns, E. M., Strickland, E. A., Tubis, A., & Jones, K. (1984). Interactions among spontaneous otoacoustic emissions. I. Distortion products and linked emissions. *Hearing Research, 19,* 191–198.

Dallos, P., Harris, D., Relkin, E., & Cheatham, M. (1980). Two-tone suppression and intermodulation distortion in the cochlea: Effect of outer hair-cell lesions. In van den Brink (Ed.), *Psychological and physiological studies in hearing.* Delft, Netherlands: Delft University Press.

Dolan, T. G., & Abbas, P. J. (1985). Changes in the $2f_1 - f_2$ acoustic emission and whole-nerve response following sound exposure: Long-term effects. *Journal of the Acoustical Society of America, 77,* 1614–1616.

Fahey, P., & Allen, J. (1986). Characterization of cubic intermodulation distortion products in the cat external auditory meatus. In J. Allen (Ed.), *Peripheral auditory mechanisms.* New York: Springer-Verlag.

Furst, M., Rabinowitz, W., & Zurek, P. M. (1988). Ear canal otoacoustic distortion at $2f_1 - f_2$ from human ears: Relation to other emissions and perceived combination tones. *Journal of the Acoustical Society of America, 84,* 215–222.

Gaskill, S. A., & Brown, A. M. (1990). The behavior of the acoustic distortion product, $2f_1 - f_2$, from the human ear and its relation to auditory sensitivity. *Journal of the Acoustical Society of America, 88,* 821–839.

Harris, F. P. (1988). *Distortion-product emissions and pure-tone behavioral thresholds.* Unpublished doctoral dissertation, University of Arizona, Tucson.

Harris, F. P. (1990). Distortion-product otoacoustic emissions in humans with high frequency sensorineural hearing loss. *Journal of Speech and Hearing Research, 33,* 594–600.

Harris, F. P., & Glattke, T. (1988). Distortion-product emissions in humans with high-frequency cochlear hearing loss. *Journal of the Acoustical Society of America, 84,* S74.

Harris, F. P., Lonsbury-Martin, B. L., Stagner, B. B., Coats, A. C., & Martin, G. K. (1989). Otoacoustic distortion products in humans: systematic changes in amplitude as a function of f2/f1 ratio. *Journal of the Acoustical Society of America, 85,* 220–229.

Horner, K., Lenoir, M., & Bock, G. (1985). Distortion product otoacoustic emissions in hearing-impaired mutant mice. *Journal of the Acoustical Society of America, 78,* 1603–1611.

Kemp, D. T. (1978). Stimulated otoacoustic emissions from within the human auditory system. *Journal of the Acoustical Society of America, 64,* 1386–1391.

Kemp, D. T. (1979). Evidence of mechanical nonlinearity and frequency selective wave amplification in the cochlea. *Archives of Otorhinolaryngology, 244,* 37–45.

Kemp, D. T., & Brown, A. M. (1983). An integrated view of cochlear mechanical nonlinearities observable from the ear canal. In E. de Boer & M. A. Viergever (Eds.), *Mechanics of hearing,* (pp. 75–82). Boston: Nijhoff.

Kim, D. O. (1980). Cochlear mechanics: Implications of electrophysiological and acoustic observations. *Hearing Research, 2,* 297–317.

Kim, D. O., Molnar, C. E., & Matthews, J. W. (1980). Cochlear mechanics: Nonlinear behavior in two-tone responses as reflected in cochlear-nerve-fiber responses and in ear-canal sound pressure. *Journal of the Acoustical Society of America, 67,* 1704–1721.

Kimberley, B. P., & Nelson, D. A. (1989a). Distortion product emissions as predictors of frequency-specific auditory thresholds. *Journal of the Acoustical Society of America, 86,* S44.

Kimberley, B. P., & Nelson, D. A. (1989b). Distortion product emissions and sensorineural hearing loss. *Journal of Otolaryngology, 18,* 365–369.

Kimberley, B. P., & Nelson, D. A. (1990). Time-averaged distortion product emissions. *Abstracts 13th ARO Midwinter Mtgs.,* 240.

Lenoir, M., & Puel, J. L. (1987). Development of $2f_1 - f_2$ otoacoustic emissions in the rat. *Hearing Research, 29,* 265–271.

Leonard, G., Smurzynski, J., Jung, M. D., & Kim, D. O. (1990). Evaluation of distortion product otoacoustic emissions as a basis for the objective clinical assessment of cochlear function. In G. F. Cianfrone & G. Kemp (Eds.), *Cochlear mechanisms and otoacoustic emissions, advances in audiology,* (pp. 139–148). Basel, Switzerland: Karger.

Lonsbury-Martin, B. L., Harris, F. P., Stagner, B. B., Hawkins, M. D., & Martin, G. (1990). Distortion product emissions in humans. I. Basic properties in normally hearing subjects. *Annals of Otology, Rhinology and Laryngology* (Suppl. 140), 3–14.

Lonsbury-Martin, B. L., & Martin, G. K. (1990). The clinical utility of distortion-product otoacoustic emissions. *Ear and Hearing, 11,* 144–154.

Lonsbury-Martin, B. L., Martin, G. K., Probst, R., & Coats, A. C. (1987). Acoustic distortion products in rabbit ear canal. I. Basic features and physiological vulnerability. *Hearing Research, 28,* 173–189.

Manley, G. A., Koppl, C., & Johnstone, B. M. (1990). Components of the $2f_1 - f_2$ distortion product in the ear canal of the Bobtail lizard. In P. Dallos, C. D. Geisler, J. W. Matthews, M. A. Ruggero, and C. R. Steele (Eds.), *The mechanics and biophysics of hearing* (pp. 210–218). Berlin: Springer-Verlag.

Martin, G. K., Ohlms, L. A., Franklin, D. J., Harris, F. P., & Lonsbury-Martin, B. L. (1990). Distortion product emissions in humans III. Influence of sensorineural hearing loss. *Annals of Otology, Rhinology and Laryngology* (Suppl. 140), 30–42.

Martin, G. K., Probst, R., & Lonsbury-Martin, B. L. (1990). Otoacoustic emissions in human ears: normative findings. *Ear and Hearing, 11,* 106–120.

Matthews, J. W., & Molnar, C. E. (1985). Modeling intracochlear and ear canal distortion product ($2f_1 - f_2$). In J. B. Allen, J. L. Hall, A. Hubbard, S. T. Neely & A. Tubis (Eds.), *Peripheral auditory mechanisms* (pp. 258–265). New York: Springer-Verlag.

Mills, D. M., Norton, S. J., & Rubel, E. W. (1992). Varying endocochlear potential by furosemide injection reveals different components in distortion product otoacoustic emissions. *Abstracts 15th ARO Midwinter Meetings,* 152.

Norton, S. J., Bargones, J. Y., & Rubel, E. W. (1991). Development of otoacoustic emissions in gerbil: Evidence for micromechanical changes underlying development of the place code.

Hearing Research, 51, 73–92.
Norton, S., & Mott, J. (1987). Effects of auditory fatigue on psychophysical estimates of cochlear nonlinearities. *Journal of the Acoustical Society of America, 82,* 80–87.
Norton, S. J., & Rubel, E. W. (1990). Active and passive ADP components in mammalian and avian ears. In P. Dallos, C. D. Geisler, J. W. Matthews, M. A. Ruggero, and C. R. Steele (Eds.), *The mechanics and biophysics of hearing* (pp. 219–226). Berlin: Springer-Verlag.
Ohlms, L., Lonsbury-Martin, B., & Martin, G. (1991). Acoustic-distortion products: Separation of sensory from neural dysfunction in sensorineural hearing loss in human beings and rabbits. *Otolaryngology Head and Neck Surgery, 104,* 159–174.
Patuzzi, R., Johnstone, B. M., & Sellick, P. M. (1984). The alteration of the vibration of the basilar membrane and inner hair cell receptor potential input-output functions in the guinea pig cochlea. *Journal of the Acoustical Society of America, 74,* 1734–1741.
Patuzzi, R. B., Yates, G. K., & Johnstone, B. M. (1989). Outer hair cell receptor current and sensorineural hearing loss. *Hearing Research, 42,* 47–72.
Pujol, R., Carlier, E., & Lenoir, M. (1980). Ontogenetic approach to inner and outer hair cell functions. *Hearing Research, 2,* 423–430.
Robles, L., Ruggero, M. A., & Rich, N. C. (1986). Basilar membrane mechanics at the base of the chinchilla cochlea. I. Input-output functions, tuning curves, and phase responses. *Journal of the Acoustical Society of America, 80,* 1364–1374.
Robles, L., Ruggero, M. A., & Rich, N. C. (1991). Two-tone distortion in the basilar membrane of the cochlea. *Nature, 349,* 413–414.
Ruggero, M. A., Rich, N. C., & Freyman, R. L. (1983). Spontaneous and impulsively evoked otoacoustic emissions: Indicators of cochlear pathology? *Hearing Research, 10,* 283–300.
Schmiedt, R. A., & Adams, J. C. (1981). Stimulated acoustic emissions in the ear canal of the gerbil. *Hearing Research, 5,* 295–305.
Sellick, P. M., Patuzzi, R. B., & Johnstone, B. M. (1982). Measurement of basilar membrane motion in the guinea pig using the Mossbauer technique. *Journal of the Acoustical Society of America, 72,* 131–141.
Siegel, J. H., & Kim, D. O. (1982). Cochlear biomechanics: Vulnerability to otoacoustic trauma and other alterations as seen in neural responses and ear canal sound pressure. In R. Hamernik, D. Henderson, and R. Salvi (Eds.), *New perspectives on noise-induced hearing loss* (pp. 137–151). New York: Raven Press.
Whitehead, M. L., Lonsbury-Martin, B. L., & Martin (1990). Actively and passively generated acoustic distortion at $2f_1 - f_2$ in rabbits. In P. Dallos, C. D. Geisler, J. W. Matthews, M. A. Ruggero, and C. R. Steele (Eds.), *The mechanics and biophysics of hearing* (pp. 243–250). Berlin: Springer-Verlag.
Whitehead, M. L., Lonsbury-Martin, B. L., & Martin, G. K. (1992). Effects of ethacrynic acid and of anoxia on acoustic distortion in the rabbit. *Abstracts 15th ARO Midwinter Meetings,* 152.
Wiederhold, M., Mahoney, J., & Kellog, D. (1986). Acoustic overstimulation reduces $2f_1 - f_2$ cochlear emissions at all levels in the cat. In J. Allen (Ed.), *Peripheral auditory mechanisms.* New York: Springer-Verlag.
Wier, C. C., Pasanen, E. G., & McFadden, D. (1988). Partial dissociation of spontaneous otoacoustic emissions and distortion products during aspirin use in humans. *Journal of the Acoustical Society of America, 84,* 230–237.
Wilson, J. (1980a). Evidence for a cochlear origin for otoacoustic re-emissions, threshold fine-structure and tonal tinnitus. *Hearing Research, 2,* 233–252.
Wilson, J. (1980b). The combination tone, $2f_1 - f_2$, in psychophysics and ear canal recording. In G. van den Brink (Ed.), *Pyschophysical, physiological studies in hearing* (pp. 43–50). Delft, Netherlands: Delft University Press.
Zurek, P. M. (1985). Otoacoustic emissions from the ear: A summary of results from humans and animals. *Journal of the Acoustical Society of America, 78,* 340–344.
Zurek, P. M., Clark, W. W., & Kim, D. O. (1982). The behavior of acoustic distortion products in the ear canals of chinchillas with normal and damaged ears. *Journal of the Acoustical Society of America, 72,* 774–780.
Zwicker, E. (1979). A model describing nonlinearities in hearing by active processes with saturation at 40 dB. *Biological Cybernetics, 35,* 243–250.

Received June 3, 1991
Accepted January 1, 1991

Contact author: David A. Nelson, PhD, Department of Otolaryngology, University of Minnesota, Box 396 UMHC, Minneapolis, MN 55455.

Appendix

An Algorithm for Estimating DPE Thresholds

Observations of the complex shapes of individual DPE growth curves indicated that a simple rule was not sufficient to estimate DPE thresholds. If the DPE threshold was taken as the level of the primaries at which the DPE was 2 or 3 dB above the individual noise-floor (NSF) level, some of the more complex low-level segments of the growth curve would be missed and only the high-level steeply sloped segment would be used to estimate DPE threshold. That would not have facilitated one of our goals, which was to estimate the lowest DPE thresholds possible. Furthermore, using only a single estimate of the noise-floor level might lead to spurious DPE thresholds. Consequently, a more complex algorithm was developed that would provide a sensitive estimate of DPE threshold. That algorithm examined pairs of DPE and NSF levels at the various stimulus levels employed, beginning at the lowest stimulus level (30 dB SPL per primary tone) and moving progressively toward higher stimulus levels. It looked for a set of consecutive DPE/NSF pairs that met specific selection criteria. For example, in Figure 3 the DPE/NSF pairs used to calculate DPE threshold are shown by filled circles. When a set of consecutive DPE/NSF pairs that met the selection criteria was found, the DPE levels in that set were fit with least-squares procedures, and the corresponding NSF levels in that set were averaged. Then DPE threshold was calculated as the stimulus level where the extrapolated DPE slope coincided with the average NSF level. The selection criteria and their values are listed opposite, along with a short description of the meaning of each criterion.

Selection Criteria

CONSEC = 3	*Number of consecutive DPE/NSF pairs required in a set. If 3, then 3 out of 3 points must meet selection criteria.*
SKIPMAX = 0	*Number of skipped points allowed in a consecutive set. If 1, then 3 out of 4 consecutive points must meet selection criteria.*
CECNIM = 0.25	*Minimum level difference required between current emission level and current noise level.*
CNPNMIN = −6	*Minimum level difference allowed between current noise level and previous noise level. To avoid noise spikes.*
CNPNMAX = n/a	*Maximum level difference allowed between current noise level and previous noise level. To avoid noise spikes.*
CEPEMIN = −1.5	*Minimum level difference allowed between current emission level and previous emission level. Negative slope detector.*
TETNMIN = 2.0	*Minimum level difference required between tail emission and tail noise level. Tail emission is last in set.*
TEHEMIN = 2.0	*Minimum level difference required between tail emission and head emission. Head emission is first in set.*
RERNPCT = 50	*Percentage of remaining emission levels in growth curve that are above the remaining noise levels. To insure valid curve.*

The values of the variables listed above were used to calculate threshold for the majority of the DPE growth curves measured. However, the complexity of some of the DPE growth curves did not allow rigorous adherence to those values for all growth curves obtained from normal and impaired ears. In the case of two-point DPE growth curves, the number of consecutive points in a set had to be reduced in order to estimate a DPE threshold. In some cases the slope for 3 consecutive points was so small that negative thresholds resulted. In those cases, 4 consecutive points usually produced a reasonable threshold estimate, e.g., Figure 3(E). There were 14 cases in which slight modifications to the above criteria were required to achieve what appeared graphically to be valid DPE threshold estimates.

The DPE threshold algorithm used here is only an initial attempt at finding an objective way to estimate DPE thresholds. This version was designed to utilize the slope of the DPE growth curve to increase the sensitivity of the estimation procedure, and compared to a simpler algorithm that uses a 2–3 dB signal-to-noise-ratio algorithm, it does estimate more sensitive DPE thresholds. Lower DPE thresholds were seen here than in previous studies. However, the increased sensitivity gained with this algorithm did not improve the correlation between DPE thresholds and auditory thresholds. It is possible that the inclusion of the slope of the growth curve in an estimation algorithm leads to artifactually low DPE thresholds. We cannot know that for certain until noise levels are reduced to reveal DPEs below the existing noise floors that are currently realizable with the RMS averaging technique used here.

Preliminary work with time-averaged DPE thresholds has shown that the noise floor can be lowered by another 20 dB with the existing instrumentation (Kimberley & Nelson, 1990). When that is done, lower DPE thresholds result, particularly at the lower frequencies where the noise floors are the largest. This suggests that the RMS averaging procedure used here limits the sensitivity of the DPE threshold estimates. It also suggests that the slope-dependency of the current algorithm makes up for some of the lack of sensitivity of the RMS averaging procedure. However, recent unpublished data from our laboratory indicates that the time-averaging procedure yields low-level DPEs with slopes that are much steeper than seen here at low levels. So, it is also apparent that the present algorithm underestimates the true slope of the DPE growth curve.

While the RMS averaging procedure yields noise floors that may limit DPE thresholds, the time-averaging procedure has its difficulties as well. It yields noise-level estimates that are much more variable than those obtained with an RMS averaging procedure. More variable noise levels make it more difficult for an objective algorithm to make reliable threshold estimates. Possible solutions may be found by averaging noise levels over a range of spectral components both above and below the DPE frequency. Other strategies might evaluate the variance of repeated noise measurements to increase the confidence of DPE threshold estimates. In any case, the DPE threshold estimating algorithm used here must only be considered an initial attempt. Its use here is not a recommendation for general clinical use. Clearly additional research is needed to find an appropriate algorithm for objectively estimating DPE thresholds.