Evidence for the distortion product frequency place as a source of distortion product otoacoustic emission (DPOAE) fine structure in humans. II. Fine structure for different shapes of cochlear hearing loss[a)]

Manfred Mauermann[b)] and Stefan Uppenkamp[c)]
AG Medizinische Physik, Universität Oldenburg, D-26111 Oldenburg, Germany

Peter W. J. van Hengel
BCN, Rijksuniversiteit Groningen, 9747 AG Groningen, The Netherlands

Birger Kollmeier
AG Medizinische Physik, Universität Oldenburg, D-26111 Oldenburg, Germany

(Received 10 January 1999; accepted for publication 24 August 1999)

Distortion product otoacoustic emissions (DPOAE) were recorded from eight human subjects with mild to moderate cochlear hearing loss, using a frequency spacing of 48 primary pairs per octave and at a level $L_1 = L_2 = 60$ dBSPL and with a fixed ratio f_2/f_1. Subjects with different shapes of hearing thresholds were selected. They included subjects with near-normal hearing within only a limited frequency range, subjects with a notch in the audiogram, and subjects with a mild to moderate high-frequency loss. If the primaries were located in a region of normal or near-normal hearing, but DP frequencies were located in a region of raised thresholds, the distortion product $2f_1 - f_2$ was still observable, but the DP fine structure disappeared. If the DP frequencies fell into a region of normal thresholds, fine structure was preserved as long as DPOAE were generated, even in cases of mild hearing loss in the region of the primaries. These experimental results give further strong evidence that, in addition to the initial source in the primary region, there is a second source at the characteristic place of f_{DP}. Simulations in a nonlinear and active computer model for DPOAE generation indicate different generation mechanisms for the two components. The disappearance of DPOAE fine structure might serve as a more sensitive indicator of hearing impairment than the consideration of DP level alone. © *1999 Acoustical Society of America.* [S0001-4966(99)02912-4]

PACS numbers: 43.64.Jb, 43.64.Kc [BLM]

INTRODUCTION

The recording of distortion product otoacoustic emissions (DPOAE) is claimed to be useful as an objective audiometric test with a high-frequency selectivity by various clinical studies. In many papers, the reported correlation of audiometric thresholds and DPOAE levels is mainly based on large databases of many subjects (e.g., Nelson and Kimberley, 1992; Gorga *et al.*, 1993; Moulin *et al.*, 1994; Suckfüll *et al.*, 1996). The prediction of individual thresholds based on DPOAE requires a detailed and comprehensive dataset from each individual subject, including growth functions, at multiple stimulus frequencies (Kummer *et al.*, 1998). However, for extensive use as a diagnostic tool, a more detailed understanding of the DPOAE generation mechanisms is still required.

In agreement with theoretical and experimental work reported by other groups (Brown *et al.*, 1996; Gaskill and Brown, 1996; Heitmann *et al.*, 1998; Talmadge *et al.*, 1998, 1999), the experimental results from normal-hearing subjects in the accompanying paper (Mauermann *et al.*, 1999) showed that DPOAE should be interpreted as the vector sum of two sources, one at the initial generation site due to nonlinear distortion close to the f_2 place, the other at the characteristic site of the particular DP frequency of interest. The results from simulations using a nonlinear and active model of the cochlea presented in Mauermann *et al.* (1999) showed that the component from the f_{DP} site is sensitive to the existence of statistical fluctuations in the mechanical properties along the cochlea partition, i.e., roughness, while the initial generation component is not. From the model point of view, this indicates different underlying mechanisms for the generation of the two DPOAE components. However, removing the roughness from certain areas along the cochlear partition—as shown in the computer simulations in Mauermann *et al.* (1999)—cannot directly be transformed into a controlled experiment with human subjects.

Other studies on modeling OAE fine structure (Zweig and Shera, 1995; Talmadge *et al.*, 1999) showed that—in addition to the roughness—the model needs another feature to produce DPOAE fine structure: broad and tall excitation patterns have to be generated to allow coherent reflections. The generation of a broad and tall excitation pattern requires an active feedback mechanism in the model. In the real co-

[a)]Parts of this study were presented at the 21st Midwinter Research Meeting of the Association for Research in Otolaryngology, 1998 in St. Petersburg Beach, FL [Mauermann *et al.*, Abstract No. 595, p. 149].

[b)]Author to whom correspondence should be addressed. Electronic mail: mani@medi.physik.uni-oldenburg.de

[c)]Current address: CNBH, Department of Physiology, University of Cambridge, Downing Street, Cambridge CB2 3EG, U.K.

chlea, this mechanism is most probably related to the motility of outer hair cells (OHC), as found by Brownell *et al.* (1985) and Zenner *et al.* (1985). If the activity of OHC in the cochlea is reduced because of damage to certain areas, most probably no broad and tall excitation pattern can build up there. As a consequence, there would be no coherent reflection from the re-emission site and no DPOAE fine structure would be observable.

This assumption is mainly based on the model results obtained so far (Mauermann *et al.*, 1999), but can be tested in carefully selected hearing-impaired human subjects. In the present paper, results on DPOAE fine structure from subjects with different audiogram shapes will be presented to investigate the effects of damage in different regions of the cochlea in more detail. Our subjects included persons with near-normal hearing only within a limited frequency range (''bandpass listeners''), a notch in the audiogram (''bandstop listeners''), or a hearing loss at high frequencies only (''low-pass listeners''). This allowed measurement of DPOAE while restricting either f_{DP} or the primaries to ''normal'' or ''near-normal'' BM regions. It was expected that only the component generated in a region of cochlear damage would be reduced. The experiments were designed to obtain further evidence for the two-source model as discussed in the accompanying paper (Mauermann *et al.*, 1999). To support the arguments, a ''hearing-impaired'' version of the computer model was also tested, simulating the bandstop listener situation mentioned above.

I. METHODS

A. Subjects

Eight subjects with different types of hearing loss participated in the experiments. They were selected because of the shapes of their audiograms. Subjects HE and HA (63 and 59 years old, respectively) showed hearing loss with a bandpass characteristic, i.e., near-normal threshold within only a small frequency band at 1.5 kHz with raised thresholds for frequencies above and below. The second group of subjects (DI, FM, MM, 29–50 years old) showed a notch in the hearing threshold of about 40 dB centered at 4 kHz. The third group (RH, HL, JK, 59–63 years old) showed a moderate high-frequency hearing loss. All subjects except MM had a stable audiogram for at least 6 months. The notch in the audiogram of MM was caused by a mild sudden hearing loss. The threshold recovered almost completely over a period of 6 months.

B. Instrumentation and experimental procedures

All instrumentation and the experimental methods for recording DPOAE fine structure are described in detail in the accompanying paper (Mauermann *et al.*, 1999). In short, an insert ear probe, type ER-10C, in combination with a signal-processing board Ariel DSP-32C, was used to record DPOAE. All stimuli were generated digitally at a sampling rate of 22.05 kHz and as harmonics of the inverse of the frame length (4096 samples, i.e., harmonics of 5.38 Hz). They were played continuously to the subjects via 16-bit D/A converters, a computer-controlled audiometer, and low-pass filters at a presentation level of $L_1 = L_2 = 60$ dBSPL. An automatic in-the-ear calibration was performed before each run to adjust the primaries to the desired sound-pressure levels. In most subjects, a frequency ratio of $f_2/f_1 = 1.2$ was used. This was increased to $f_2/f_1 = 1.25$ in some cases to achieve a larger separation of f_{DP} and the primary frequencies.

DPOAE were recorded at a high-frequency resolution of 32 frequencies per octave. The microphone output was amplified, A/D converted and averaging in the time domain of at least 16, and if necessary up to 256 repeated frames were performed for each pair of primaries to increase the signal-to-noise ratio. Again, all results were plotted as a function of f_2 and as a function of f_{DP}. This permits us to relate the data both to the initial generation site near f_2 and to the presumed second source located around the characteristic site of f_{DP}.

In addition to the clinical audiogram, absolute thresholds were measured for five of the eight subjects with an adaptive three-alternative forced-choice (3AFC) two-step method to get a more accurate estimate of the shape of the audiogram. The absolute threshold was measured at a resolution of eight frequencies per octave in the transition regions between near-normal hearing and increased threshold. Sinusoids of 375-ms duration including 45 ms Hanning-shaped ramps at the start and end were used as stimuli. They were played through one of the speakers of the ER10C probe. After threshold detection, the sound pressure in the ear canal was measured with the ER10C probe microphone for a fixed attenuation of the audiometer. The sound-pressure level in dB SPL at threshold was computed from the difference of this attenuator value and the attenuation at hearing threshold.

II. RESULTS

Figures 1–4 show the DPOAE fine-structure patterns and absolute thresholds for eight subjects. In each figure, the left column gives the clinical audiogram. The middle column shows DPOAE results (black line) and absolute threshold (gray line) as a function of f_{DP} to permit a direct comparison around the assumed re-emission site. The right column shows the same data as a function of f_2 to allow a direct comparison of absolute threshold and DPOAE fine structure at the initial generation site close to f_2. For the middle and right columns, the left ordinate holds for the DPOAE level in dB SPL, while the right ordinate is for the absolute threshold. This threshold was measured in dB SPL for the subjects who did the adaptive procedure (HE, HA, DI, HL, JK), while for the other subjects (MM, FM, RH) the threshold in dB HL from the clinical audiogram (left column) is given. In addition to the threshold data (or audiogram data), the black bars on the top of the middle and right column panels give a sketch of the area with thresholds of 20 dB or better, i.e., frequency ranges with normal or near-normal hearing (as taken from the audiograms).

Figure 1 shows results for two subjects with a near-normal threshold in only a limited frequency band. A reduced DPOAE fine structure with level fluctuations smaller than 5 dB (areas indicated by the gray bar on the top of the

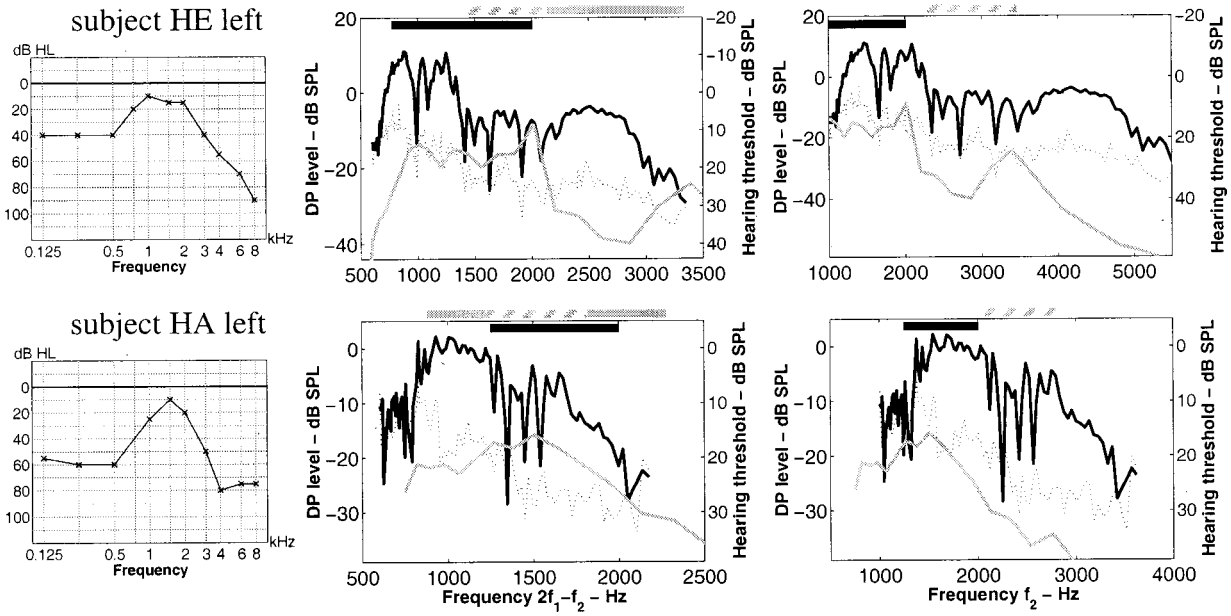

FIG. 1. DPOAE fine structure from two ears with near-normal threshold within a bandlimited frequency region only. Left: clinical audiogram. Middle: absolute threshold (gray line, right ordinate) and DP level as a function of $2f_1-f_2$ (black line, left ordinate). Right: as middle column, but DP level as a function of f_2. The dotted line represents the noise floor during the DPOAE recording. Frequency regions with near-normal hearing (20 dB HL or better) are indicated by black bars on the top of the plots. Regions with reduced fine structure (less than 5-dB level fluctuations) are indicated by gray bars. Hatched gray bars mark areas with reduced DPOAE level but still pronounced fine structure. Top row: subject HE, left ear. Bottom row: subject HA, left ear. Frequency ratio of the primaries: $f_2/f_1=1.25$; levels of the primaries: $L_1=L_2=60$ dB SPL.

middle column plots) can be observed when f_{DP} falls into a region of raised threshold. On the other hand, when the distortion products are at frequencies with near-normal hearing and the primaries at frequencies of a moderate hearing loss, the DPOAE level is reduced, but a preserved fine structure with level fluctuations of 5 dB or more can still be observed in a certain area (indicated by the hatched gray bars on the top of the plots).

Figure 2 shows the results from four ears of three subjects with notches in their audiograms. As was the case for the data shown in Fig. 1, when the distortion product frequencies fall into the area of hearing loss, the fine structure disappears but nearly no reduction in DPOAE level occurs. If the initial DPOAE generation site, i.e., the area around f_2, falls into the region of hearing loss while the related f_{DP} frequency covers a region of near-normal hearing, the DPOAE level is reduced but a pronounced fine structure is still observed. If both f_2 and f_{DP} fall into a region of mild to moderate hearing loss, DPOAE level and DPOAE fine structure are reduced. Subject FM (bottom row in Fig. 2) showed a reduced fine structure in a limited frequency band above 4 kHz only. This might be due to a narrow-band notch in the absolute threshold in this frequency region which was not resolved using the clinical audiogram.

Figure 3 shows DPOAE fine-structure patterns for two subjects with a moderate hearing loss only at high frequencies. When plotted as a function of f_2 (right column), a DPOAE level similar to the level in regions with near-normal hearing could still be observed in the region of raised threshold, while the fine structure disappeared as soon as the distortion product frequencies fell into the region of hearing loss (cf. middle column for plot as a function of $2f_1-f_2$). These cases suggest that the DPOAE fine structure might provide a more sensitive indicator of cochlear damage than the DPOAE level, which is mainly related to the initial generation site close to f_2.

Figure 4 gives one example of a different effect of hearing loss on DPOAE. This subject also had a high-frequency hearing loss, but the level of DPOAE decreased with the high-frequency hearing loss, while the fine structure was preserved as long as DPOAE are recordable, i.e., below $f_2=4$ kHz. As can be seen in the audiogram, thresholds were normal in the region below 2.5 kHz, i.e., in the region of the related f_{DP} frequencies. Therefore, an effect on DPOAE fine structure is not expected in this particular case of a steep high-frequency hearing loss.

Figure 5 shows again the DPOAE fine structure for subject MM (cf. Fig. 2), this time at three different stages of recovery from the mild sudden hearing loss. After 4 months (dark gray line), the clinical audiogram was almost normal and the DPOAE level had returned to normal (notice the recovery from the notch in the middle trace between 2000 and 3000 Hz for $2f_1-f_2$, corresponding to a f_2 range of 3000–4000 Hz, cf. Fig. 2). A reappearance of fine structure over the whole range could only be observed after 6 months. It is possible that a slight cochlear disorder still affected the DPOAE fine structure after 4 months, while the DPOAE level and the audiogram had almost completely recovered. Again, the fine structure appears to be a more sensitive indicator for local cochlear damage than the consideration of overall DPOAE level alone, revealing even slight disorders not detectable in the clinical audiogram.

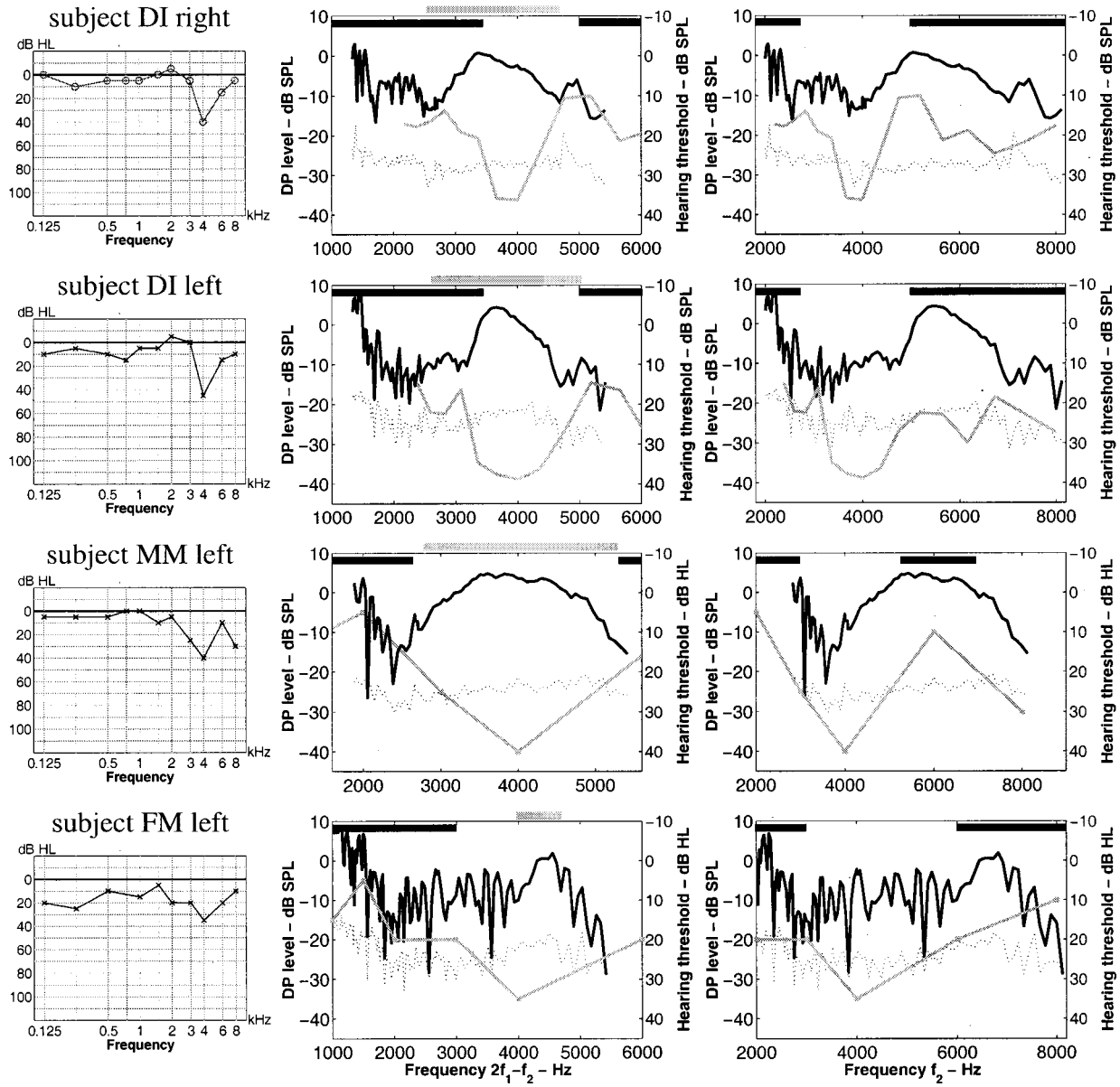

FIG. 2. As Fig. 1, but showing results from four ears with a notch in the audiogram. Top row: subject DI, right ear. Second row: subject DI, left ear. Third row: subject MM, left ear. Fourth row: subject FM, left ear. The notch in the audiogram of subject MM was caused by a mild sudden hearing loss. MM and FM did not perform the adaptive procedure for evaluation of absolute thresholds. Therefore, the threshold curves are taken from the clinical audiograms in these cases. Frequency ratio of the primaries: $f_2/f_1=1.2$; levels of the primaries: $L_1=L_2=60$ dB SPL.

III. SIMULATIONS IN A NONLINEAR AND ACTIVE MODEL OF THE COCHLEA

As shown in the accompanying paper (Mauermann *et al.*, 1999), the behavior of DPOAE fine-structure patterns in different experimental paradigms can be well simulated with a nonlinear and active transmission line model of the cochlea. Here, a ''hearing-impaired'' version of this computer model is tested to investigate the effects of local changes of the damping function, as an analog of hearing loss in a restricted frequency region. This can be achieved by looking at a partly ''passive'' cochlea. The biggest hearing loss that can be modeled by making the damping independent of velocity and fixing it to the maximum value that occurs in the ''normal-hearing'' model is about 40 dB. The value of 40 dB corresponds to the assumed gain of cochlea activity probably due to motility of the OHC (discussed in Pickles, 1988; Hoth and Lenarz, 1993). Therefore, thresholds distinctively higher than 40 dB probably have to be related to damage of the inner hair cells (IHC), which cannot be simulated in this kind of macromechanical model.

Figure 6 shows one example of a fine-structure pattern, calculated using a model cochlea with raised threshold around 3.7 kHz. The hearing loss in this case was introduced by taking a relatively high positive damping, independent of velocity, for the segments representing the frequencies from 3.6 to 3.8 kHz in the model cochlea (segments 210 to 219 of a total of 600 segments) with smooth transitions (over 3.3 to 3.6 kHz and 3.8 to 4.5 kHz) (see the Appendix). The ''delayed feedback stiffness'' is smoothly decreased to 0 in this region in proportion to the increased damping (for a more detailed description of the model, see Mauermann *et al.*, 1999). As found in the experiments, the fine structure in the

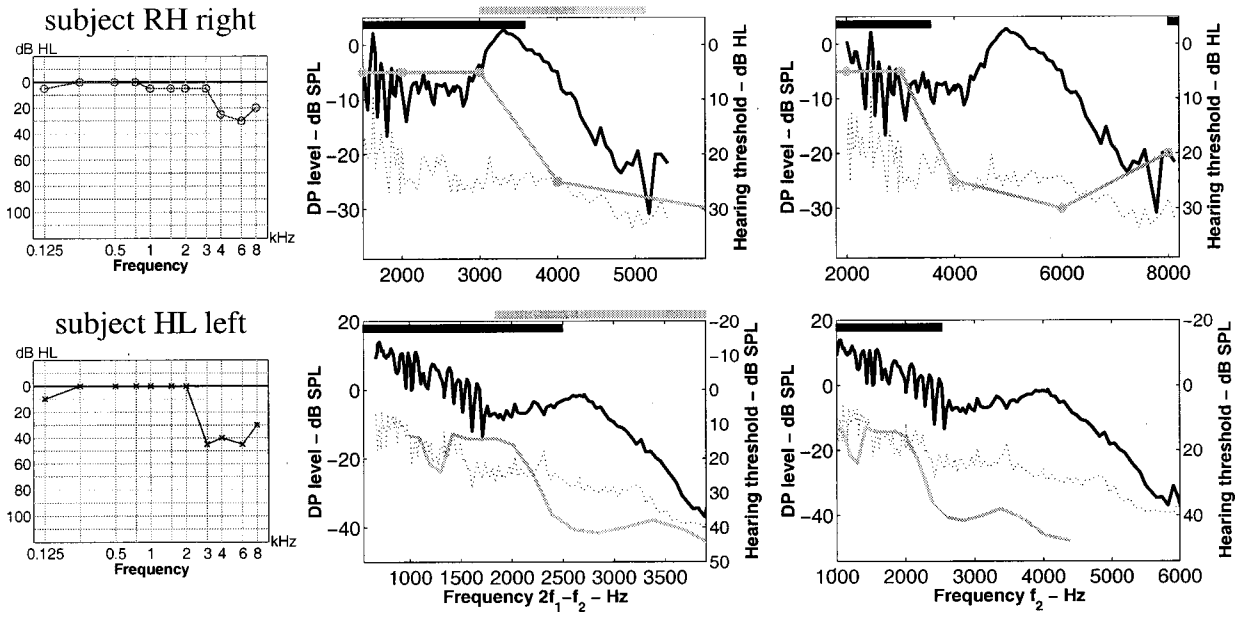

FIG. 3. As Fig. 2, but showing results from two subjects with high-frequency hearing loss. Top row: subject RH, right ear. Bottom row: subject HL, left ear. Absolute threshold was taken from the clinical audiogram for subject RH.

computer simulation disappears as soon as f_{DP} falls into the region of increased damping, i.e., increased threshold, while a reduction of DPOAE level is observable when the primaries cover the region of ''hearing loss.'' The hearing loss was quantified by finding the stimulus level required to give the same excitation as a stimulus level of 0 dB SPL in the normal-hearing version of the model.

IV. DISCUSSION

Most previous studies on DPOAE from hearing-impaired subjects related DPOAE levels to hearing thresholds based only on a comparatively low number of frequencies. In the only study dealing with DPOAE from hearing-impaired subjects with a high-frequency resolution (He and Schmiedt, 1996), it was concluded that a fine structure would always be observable as long as DPOAE can be recorded. Their study included 14 hearing-impaired subjects, all of whom had a more or less steep high-frequency hearing loss. This conclusion of unaffected fine structure holds only for one subject (JK, Fig. 4) from this investigation, while the others showed a substantial decrease of DPOAE fine structure when $2f_1-f_2$ fell into a region of hearing loss. The subjects presented here were selected because of the particular shape of their audiograms with either raised thresholds or normal hearing in a limited frequency band only. This sample might not be representative for the clinical population, but it permitted measurements with either the primary frequencies or the distortion product frequencies covering a region of cochlear damage, motivated by the results and computer simulations reported in the accompanying paper (Mauermann *et al.*, 1999). Consequently, our data will be discussed in respect to the two-source model of DPOAE generation. The experiments were not intended as a representative clinical study. Nevertheless, the results might still improve the value of DPOAE as a diagnostic tool.

The initial generation of DPOAE is due to nonlinear distortion at the primary site close to f_2. The cases reported in Figs. 1–3 exhibit a coincidence of the disappearance of DPOAE fine structure with damage at the characteristic place of f_{DP}. This strongly supports the interpretation of the data in terms of a two-source model of DPOAE generation, as discussed in the accompanying paper for normal-hearing subjects (Mauermann *et al.*, 1999). If only the component generated in the primary region contributes to the emission measured in the ear canal, no fine structure can be observed. When there is also a contribution from the re-emission site at

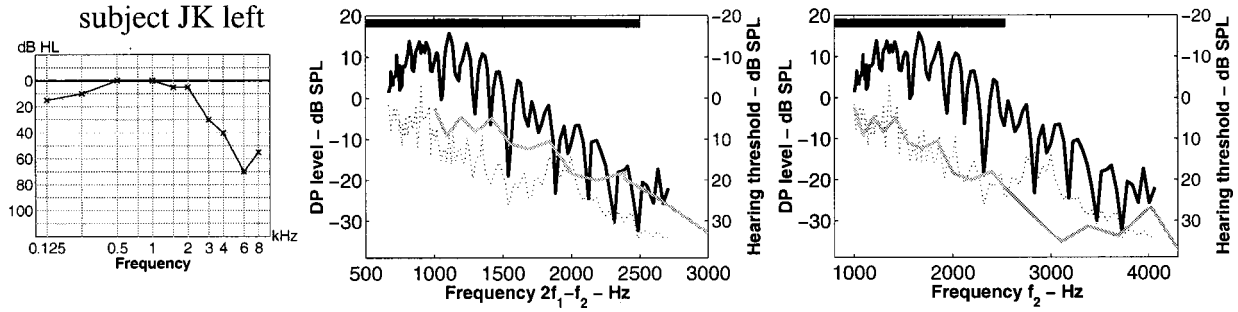

FIG. 4. As Fig. 1, but for subject JK, left ear. This subject also shows a high-frequency hearing loss. In contrast to the data shown in Fig. 3, the DPOAE level decreases with increasing threshold but the fine structure remains unaffected, as long as DPOAE are recordable at all.

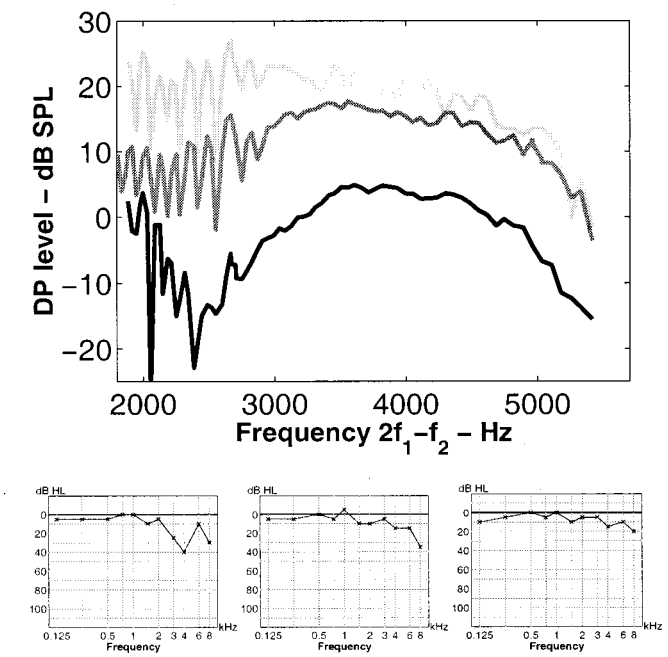

FIG. 5. Recovery of DPOAE fine structure after a mild sudden hearing loss, subject MM, left ear (cf. Fig. 2). Top panel: DPOAE level is plotted as a function of $2f_1-f_2$ on the day of sudden hearing loss (bottom trace), 4 months later (middle trace), and 6 months later (top trace, complete recovery of absolute threshold). The ordinate holds for the bottom trace only, the other traces are shifted by +10 and +20 dB, respectively. Bottom row, from left to right: the clinical audiograms at the day of sudden hearing loss, 4 months later, and 6 months later.

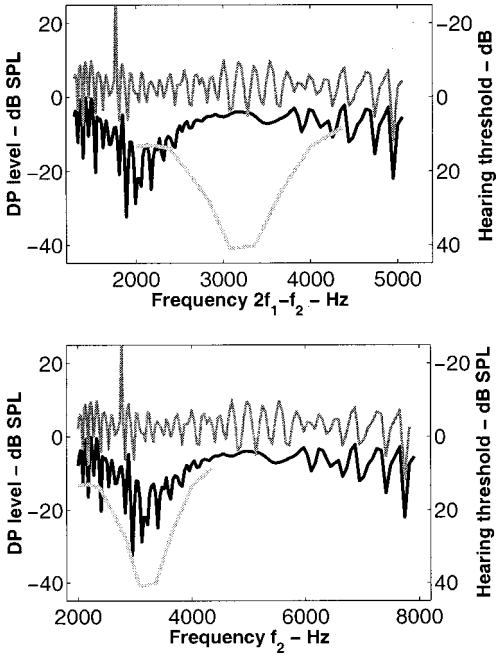

FIG. 6. Simulation of DPOAE fine structure for a frequency ratio $f_2/f_1 = 1.22$ in a version of the transmission-line model with a notch in the audiogram in the region between 3.3 and 4.5 kHz vs the simulated DPOAE fine structure of the normal-hearing model. Black line: DPOAE fine structure of the hearing-impaired model at a frequency resolution of 64 steps per octave. Gray line: DPOAE fine structure of the normal-hearing model (shifted 10 dB up). Light gray line: simulated hearing threshold of the hearing impaired model at eight frequencies per octave.

f_{DP}, a quasiperiodic fine structure is observable, i.e., DPOAE can be treated as the vector sum of two different components, as suggested by Brown *et al.* (1996).

Heitmann *et al.* (1998) showed that the presentation of an additional suppressor tone close to f_{DP} (25 Hz above f_{DP}) causes a disappearance of fine structure due to suppression of the component from the f_{DP} place. As demonstrated here, damage in the DP frequency region has a similar effect to the suppressor. While the addition of a third tone could cause unwanted side effects when investigating the DP generation mechanisms, such as additional distortion products (Harris *et al.*, 1992), the experiments presented here take advantage of the ''naturally'' reduced cochlear activity.

As already reported in previous studies (e.g., Schlögel *et al.*, 1995), it is not uncommon for subjects with high-frequency hearing loss to have DPOAE not substantially different in level from those from normal-hearing subjects, even when the primaries fall into the region of a hearing loss of 30 dB or more. Two more examples for this are the subjects RH and HL in this study (cf. Fig. 3). However, DPOAE fine structure disappeared in both subjects as soon as f_{DP} fell into a region of even slightly raised thresholds (i.e., above 2.5 kHz for subject HL, above 4 kHz for subject RH). This suggests that moderate cochlear damage can already influence the re-emission component of the DPOAE. A similar interpretation holds for the DPOAE fine structure during recovery from a mild sudden hearing loss shown in Fig. 5. The reduced fine structure after 4 months still might indicate some slight damage, although the threshold—as measured in the clinical audiogram with its accuracy of about ±5 dB and its limited frequency resolution—had already recovered. This high vulnerability to cochlear damage of the DPOAE fine structure has also been noticed in other studies. Engdahl and Kemp (1996) showed that noise exposure causing a temporary threshold shift results in a temporary disappearance of DPOAE fine structure. Furthermore, the fine structure gets reduced during aspirin consumption before an overall DPOAE level reduction can be observed (Rao *et al.*, 1996; Long, 1999). Overall, it appears as if the consideration of fine structure can serve as a more sensitive tool for the detection of slight cochlear damage in certain cases than the DPOAE level alone. However, a prospective clinical study involving more subjects would be required to evaluate this possible application.

In contrast to the subjects discussed so far, the DPOAE from subject JK (Fig. 4) appears to behave differently: DPOAE level decreases with increasing hearing loss while the fine structure is unaffected. This behavior is more in line with the data reported by He and Schmiedt (1996). The seeming contradiction to our other data can be interpreted as follows: When the primary at f_2 falls into a region of a distinct hearing loss, i.e., above 4 kHz for this subject, no measurable initial DPOAE component is generated. Consequently, no reflection component from the re-emission site close to f_{DP} can be recorded. However, when f_2 is below 4 kHz, the corresponding f_{DP} frequency still falls into a region of normal or near-normal hearing for this subject (i.e., threshold of 20 dB HL or better), which is sufficient to create the re-emission component due to coherent reflection. The interaction of the two components generates the fine struc-

ture. A similar explanation would also hold for most of the subjects described by He and Schmiedt (1996), who had steep high-frequency hearing losses.

The effects of frequency-specific hearing loss on DPOAE fine structure can be simulated in a realistic way using the hearing-impaired version of the computer model of the cochlea (cf. Figs. 2 and 6). The very good correspondence between data and simulations even in the case of hearing impairment gives further support for a whole class of similar cochlea models recently described in Talmadge *et al.* (1999). In the model, the generation mechanisms for the two sources are different. The re-emission component can be interpreted as a coherent reflection sensitive (1) to the presence of ''roughness,'' and (2) to the presence of broad and tall excitation patterns (e.g., Talmadge *et al.*, 1998), which are generated by an active feedback mechanism. The initial generation—which is not sensitive to the presence of roughness (see Mauermann *et al.*, 1999; Talmadge *et al.*, 1998)—is not connected to coherent reflection but should be interpreted as a consequence of nonlinear distortion only.

This view of two different mechanisms is in agreement with the conclusions drawn by Shera and Guinan (1999). They also distinguish two DPOAE mechanisms: coherent reflection from the f_{DP} site [similar to stimulus frequency otoacoustic emissions (SFOAE)] and nonlinear distortion from the generation site near f_2. The different effects on DPOAE fine structure and overall level caused by cochlear damage, as reported in our experiments, might reflect an experimental confirmation of two different generation mechanisms.

V. CONCLUSIONS

(i) Distortion product emissions measured in the human ear canal are produced by two sources, one at the characteristic place of the primaries and the second at the characteristic place of f_{DP}.

(ii) The DPOAE fine structure is mainly influenced by the local state of the cochlea at the characteristic place of f_{DP} and appears to be a more sensitive indicator of cochlear damage than DPOAE level alone.

(iii) At least from the model point of view, the initial generation at the site close to f_2 is caused by nonlinear interaction of the primaries while the re-emission from the characteristic site of f_{DP} can be treated as a coherent reflection.

(iv) The evaluation of fine structure could considerably improve the clinical use of DPOAE, e.g., for early identification of hearing loss or to monitor the recovery from a sudden hearing loss more accurately.

ACKNOWLEDGMENTS

This study was supported by Deutsche Forschungsgemeinschaft, DFG Ko 942/11-2. Helpful comments by Brian Moore and Glenis Long on an earlier version of the manuscript are gratefully acknowledged.

APPENDIX

The nonlinearity in the model is introduced by a nonlinear damping $d(x,v)$ and a stabilizing delayed feedback stiffness $c(v)$ (see Mauermann *et al.*, 1999).

$$d(x,v)=\left[d_l+\frac{\beta(d_h-d_l)|v|}{1+\beta|v|}\right]\sqrt{ms(x)}, \tag{A1a}$$

$$c(v)=c_l+\frac{-\beta d_l|v|}{1+\beta|v|}, \tag{A1b}$$

where v is the velocity of basilar membrane (BM) section; m is the mass (0.375 kg/m^2); x is the distance to the base of the cochlea; s is the stiffness of BM section; d_l is the damping parameter, determines the damping at low BM velocities (d_l is -0.12 in the normal-hearing model); d_h is the damping parameter, determines the damping at high BM velocities (d_h is 0.5 in the normal-hearing model); c_l is the parameter of delayed feedback stiffness (c_l is 0.1416 in the normal-hearing model); and β is the parameter to determine the shape of the nonlinear damping function.

The hearing loss was introduced by taking $d(x,v)=d_h$ in the region from 3.6 to 3.8 kHz with smooth transitions (over 3.3 to 3.6 kHz and 3.8 to 4.5 kHz). This could be done by letting $\beta\rightarrow\infty$. Because this is impossible in practice, we let $\beta(d_h-d_l)$ go to 0 and d_l go to d_h simultaneously. In correspondence to the increased damping, the delayed feedback stiffness $c(v)$ is smoothly decreased to 0 in this region.

Brown, A. M., Harris, F. P., and Beveridge, H. (**1996**). ''Two sources of acoustic distortion products from the human cochlea,'' J. Acoust. Soc. Am. **100**, 3260–3267.

Brownell, W., Bader, C. R., Bertrand, D., and Ribaupierre, Y. (**1985**). ''Evoked mechanical responses of isolated cochlear outer hair cells,'' Science **227**, 194–196.

Engdahl, B., and Kemp, D. T. (**1996**). ''The effect of noise exposure on the details of distortion product otoacoustic emissions in humans,'' J. Acoust. Soc. Am. **99**, 1573–1587.

Gaskill, S., and Brown, A. (**1996**). ''Suppression of human acoustic distortion product: Dual origin of 2 $f1-f2$, '' J. Acoust. Soc. Am. **100**, 3268–3274.

Gorga, M. P., Neely, S. T., Bergman, B., Beauchaine, K. L., Kaminski, J. R., Peters, J., and Jestaedt, W. (**1993**). ''Otoacoustic emissions from normal-hearing and hearing-impaired subjects: Distortion product responses,'' J. Acoust. Soc. Am. **93**, 2050–2060.

Harris, F., Probst, R., and Xu, L. (**1992**). ''Suppression of the 2 $f1-f2$ otoacoustic emission in humans,'' Hear. Res. **64**, 133–141.

He, N., and Schmiedt, R. A. (**1996**). ''Effects of aging on the fine structure of the 2 $f1-f2$ acoustic distortion product,'' J. Acoust. Soc. Am. **99**, 1002–1015.

Heitmann, J., Waldmann, B., Schnitzler, H. U., Plinkert, P. K., and Zenner, H. P. (**1998**). ''Suppression of distortion product otoacoustic emissions (DPOAE) near 2 $f1-f2$ removes DP-gram fine structure—Evidence for a secondary generator,'' J. Acoust. Soc. Am. **103**, 1527–1531.

Hoth, S., and Lenarz, T. (**1993**). *Otoakustische Emissionen—Grundlagen und Anwendung* (Thieme, Stuttgart), p. 12.

Kummer, P., Janssen, T., and Arnold, W. (**1998**). ''The level and growth behavior of the 2 $f1-f2$ distortion product otoacoustic emission and its relationship to auditory sensitivity in normal hearing and cochlear hearing loss,'' J. Acoust. Soc. Am. **103**, 3431–3444.

Long, G. R. (**1999**). Personal communication.

Mauermann, M., Uppenkamp, S., van Hengel, P. W. J., and Kollmeier, B. (**1999**). ''Evidence for the distortion product frequency place as a source of distortion product otoacoustic emission (DPOAE) fine structure. I. Fine structure and higher order DPOAE as a function of the frequency ratio $f2/f1$,'' J. Acoust. Soc. Am. **106**, 3473–3483.

Moulin, A., Bera, J. C., and Collet, L. (**1994**). ''Distortion product otoacoustic emissions and sensorineural hearing loss,'' Audiology **33**, 305–326.
Nelson, D. A., and Kimberley, B. P. (**1992**). ''Distortion product emissions and auditory sensitivity in human ears with normal hearing and cochlear hearing loss,'' J. Speech Hear. Res. **35**, 1142–1159.
Pickles, J. (**1988**). *An Introduction to the Physiology of Hearing*, 2nd ed. (Academic, London), p. 149.
Rao, A., Long, G. R., Narayan, S. S., and Dhar, S. (**1996**). ''Changes in the temporal characteristics of TEOAE and the fine structure of DPOAEs with aspirin consumption,'' in Abstracts of the 19th ARO Midwinter Research Meeting, Abstract, p. 27.
Shera, C. A., and Guinan, J. J. (**1999**). ''Evoked otoacoustic emissions arise by two fundamentally different mechanisms: A taxonomy for mammalian OAEs,'' J. Acoust. Soc. Am. **105**, 782–798.
Schlögel, H., Stephan, K., Böheim, K., and Welzl-Müller, K. (**1995**). ''Distorsionsprodukt otoakustische Emissionen bei normalem Hörvermögen und bei sensorineuraler Schwerhörigkeit,'' HNO **43**, 19–24.
Suckfüll, M., Schneeweiss, S., Dreher, A., and Schorn, K. (**1996**). ''Evaluation of TEOAE and DPOAE measurements for the assessment of auditory thresholds in sensorineural hearing loss,'' Acta Oto-Laryngol. **116**, 528–533.
Talmadge, C. L., Tubis, A., Long, G. R., and Piskorski, P. (**1998**). ''Modeling otoacoustic emission and hearing threshold fine structure,'' J. Acoust. Soc. Am. **104**, 1517–1543.
Talmadge, C. L., Long, G. R., Tubis, A., and Dhar, S. (**1999**). ''Experimental confirmation of the two-source interference model for the fine structure of distortion product otoacoustic emissions,'' J. Acoust. Soc. Am. **105**, 275–292.
Zenner, H., Zimmermann, U., and Schmidt, U. (**1985**). ''Reversible contraction of isolated mammalian cochlear hair cells,'' Hear. Res. **18**, 127–133.
Zweig, G., and Shera, C. A. (**1995**). ''The origin of periodicity in the spectrum of evoked otoacoustic emissions,'' J. Acoust. Soc. Am. **98**, 2018–2047.