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Boundary conditions of otoacoustic emissions

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Document Version

Publisher's PDF, also known as Version of record

Publication date:

2001

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Citation for published version (APA):

Kleine, E. D. (2001). *Boundary conditions of otoacoustic emissions*. s.n.

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Chapter 5

Evoked otoacoustic emissions in patients with Menière's disease

Abstract

Click-evoked, as well as distortion product otoacoustic emissions (OAEs), were measured in 100 patients with Menière's disease. The incidence of the emissions in affected ears (56%) was lower than in unaffected — or contralateral — ears (85%). The mean emission amplitude in affected ears was also significantly lower (2.6 dB), and, in turn, the mean amplitude in unaffected ears was lower than in normal-hearing ears (5.3 dB). These differences are likely to be caused by the hearing loss involved. Further, ears with OAEs clearly showed smaller hearing losses than ears without OAEs (24 dB difference). The average hearing loss did show correlations with the emission amplitudes, although this was not very strong; when plotted against the smallest hearing loss, a certain upper boundary for the emission amplitude was present. Also, the amplitude of click-evoked OAEs showed a considerable correlation with the largest of the three distortion product OAEs. These observations confirm the view that OAEs are associated with normal or near-normal hearing.

5.1 Introduction

Patients with Menière's disease suffer from symptoms like hearing loss, tinnitus, and attacks of dizziness. Despite extensive research, the etiology of the disease has not yet been clarified. It is, however, hypothesized that an increased amount of endolymph (i.e., endolymphatic hydrops) may be the underlying mechanism causing the symptoms. Several models have been proposed by which an endolymphatic hydrops could be generated, and by which then the symptoms could be accounted for (for a review, see Kiang 1989 and Horner 1993).

Otoacoustic emissions (OAEs) are sounds generated in the inner ear which are measurable in the ear canal. They can either appear spontaneously or be evoked by a stimulus (for a review, see Probst et al. 1991). Since the prevalence of OAEs is connected to hearing loss, OAE measurements are nowadays widely used to probe cochlear functioning. In view of the fact that Menière's disease is probably related to inner-ear pathology, OAE measurements in patients might well provide more insight into the disease (e.g., Harris and Probst 1992; Van Huffelen et al. 1998; Cianfrone et al. 2000).

In our clinic, an ongoing comprehensive project is being carried out to study Menière's disease and its pathology in order to gain insight in the staging of the disease and to eventually develop a suitable therapy. Our patients are subjected to a set of otological, audiological, vestibular, radiological and laboratory examinations (see e.g., Horst and De Kleine 1999; Mateijssen et al. 2000). In this paper, we present the results of measurements of click-evoked and distortion product OAEs (CEOAEs and DPOAEs, respectively) in patients with Menière's disease. A comparison is made between the affected and unaffected ears of the patients, as well as with a group of ears from normal-hearing persons.

5.2 Materials and methods

Patients were diagnosed as suffering from Menière's disease when they (1) had a history of at least two vertigo attacks, (2) suffered or had suffered from tinnitus, and (3) had a cochlear hearing loss of at least 20 dB at one of the frequencies of the standard audiogram (aural fullness was omitted; see Mateijssen et al. 2000). Furthermore, other underlying pathologies were

excluded by a comprehensive set of diagnostic examinations. A total number of 111 patients with Menière's disease (54 males and 57 females) was examined. The complete set of otoacoustic emission measurements was — technically — successfully performed on 100 of these, yielding the results of 200 ears. This group of 100 patients consisted of 51 males and 49 females. The average age of the patients equaled 50 years [standard deviation (SD) 11]. Of these patients, 58 were unilaterally and 42 were bilaterally affected. Hence, 142 affected and 58 unaffected (i.e., contralateral) ears were included in our analyses.

Emission measurements were performed with the ILO equipment from Otodynamics Ltd. (for details, see Van Huffelen et al. 1998). Click-evoked otoacoustic emissions were measured using the nonlinear mode with 80- μ s rectangular pulses. The emissions discussed below were obtained with a stimulus of 90–95 dB. The criteria for a valid CEOAE were a successful probe fit, a reproducibility greater than 55%, and an amplitude of at least 0 dB SPL. Frequency-specific data on CEOAEs were not stored. Distortion product otoacoustic emissions at $2f_1 - f_2$ were measured with stimulus frequencies $f_2 = 1, 2$ and 4 kHz, and frequency ratio $f_2/f_1 = 1.2$ (they will be referred to as DPOAEs at these f_2 frequencies). The level of both stimulus tones was 70 dB. Emissions were regarded to be present when the signal exceeded the local noise level, and had a minimum amplitude of -10 dB SPL. Pure-tone audiograms were obtained for 6 frequencies: 0.25, 0.5, 1, 2, 4, and 8 kHz (with an accuracy of 5 dB; down to 10 dB HL, the lowest value being carried out in our audiometric practice). The average hearing loss was calculated as the average of these 6 threshold values; the smallest hearing loss was calculated as the minimum value of the 6 threshold values. For three ears, no reliable audiogram could be obtained. Data on the severity and duration of the distinct symptoms were gained by means of a questionnaire (see Mateijsen et al. 2000).

Statistical analyses were performed using SPSS software. The main methods we used were the Chi-squared test and the t test; further, Pearson's rank-correlation coefficient was used (several variables were not normally distributed). Throughout this paper, a significant result for the different tests implies $P < 0.001$ and a nonsignificant result implies $P > 0.05$, unless stated otherwise.

5.3 Results

Figure 5.1 shows the averaged pure-tone audiograms of our patient group. Left and right panel represent the affected and unaffected ears, respectively. From this picture, it is clear that affected ears show greater hearing losses than unaffected ears (44 versus 20 dB HL); moreover it demonstrates that the unaffected ears do not have normal hearing. Since the hearing losses were not normally distributed, we plotted the quartile values instead of the standard deviation to quantify the variability.

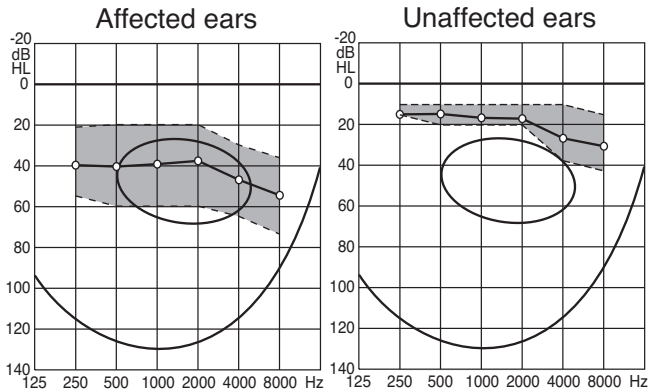


Figure 5.1: Averaged pure-tone audiograms of the affected ($N = 142$) and unaffected ($N = 58$) ears of patients with Menière's disease. The circles indicate the averaged value; the grey area represents the inter-quartile range (i.e., it covers the central 50% of the observed threshold values). The mean average hearing loss (for all frequencies) for affected and unaffected ears was 44 and 20 dB HL, respectively.

Figure 5.2 shows the incidence numbers of each type of otoacoustic emission (OAE). For all ears, each particular emission type was measurable (i.e., exceeded the noise) in approximately 65% of the ears. Overall, in 82 ears (41%) all four OAEs were measured, and 36 ears (18%) showed no OAEs at all. Comparing the affected and unaffected (i.e., contralateral) ears, an average of 56% (range: 50–58) of the affected ears showed OAEs and 44% did not, whereas for the unaffected ears an average of 85% (range: 71–93) showed OAEs and 15% did not. The differences between these percentages were evaluated by performing a Chi-squared test. This gave strong evidence

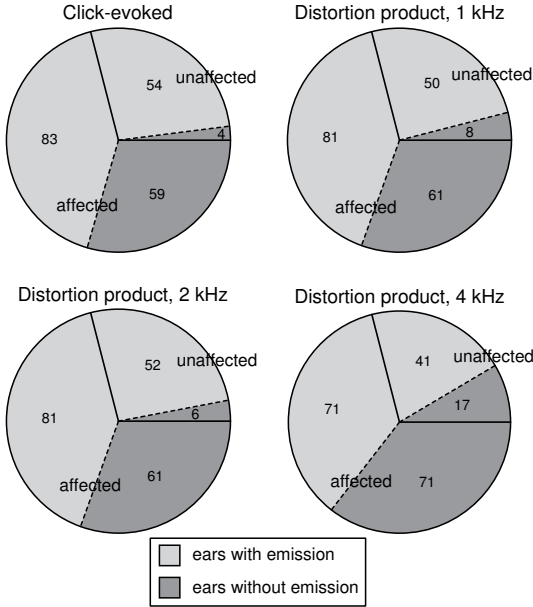


Figure 5.2: Incidence of otoacoustic emissions in patients with Menière’s disease. The circles indicate the incidence of click-evoked and the three distortion product OAEs in all 200 ears: 142 affected and 58 unaffected. Light and dark grey parts, respectively, indicate ears with and without emission. Absolute numbers are indicated in each part. Incidence in affected ears ranged from 50–58%, in unaffected ears from 71–93%. On the whole, in 82 of all ears (41%) all four OAE types could be measured, and in 36 ears (18%) no OAEs could be measured at all. According to a Chi-squared test, a relation between OAE presence and affectedness was present.

in support of an association between affectedness and the presence of each OAE type ($P < 0.01$ for DPOAEs at 4 kHz). The strength of these relations, as expressed by Cramer’s V (ranging from 0 to 1, for no relation and a perfect relation, respectively) ranged from 0.19 for DPOAEs at 4 kHz, to 0.34 for CEOAEs. Thus, a relation between affectedness and presence of OAEs existed but was not very strong. The Chi-squared test further indicated that the differences in OAE incidence between the affected ears of uni- and bilaterally affected patients were not significant.

The association between otoacoustic emissions and hearing loss was ex-

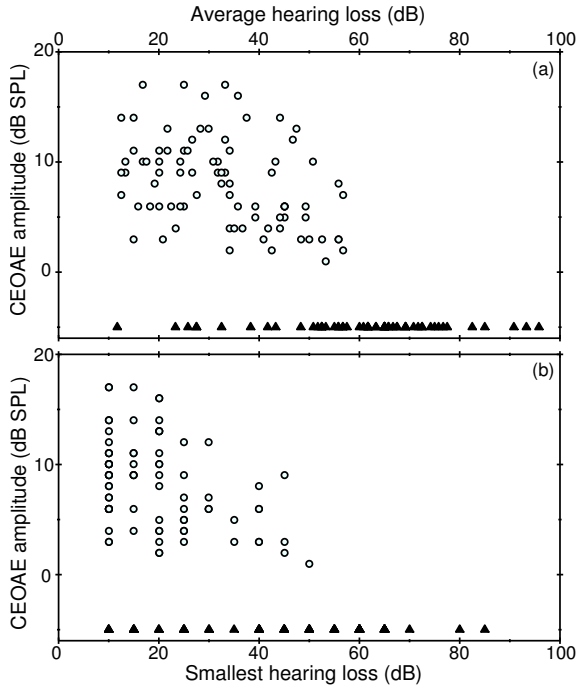


Figure 5.3: Amplitudes of click-evoked otoacoustic emissions as a function of hearing loss, for all affected ears. The open circles indicate ears from which an emission was measured ($N_{\text{CEOAE}+} = 82$); the filled triangles indicate ears from which no emission could be measured ($N_{\text{CEOAE}-} = 58$), the latter points' amplitude is consequently meaningless. (a) Emission amplitude versus average hearing loss. The mean average hearing loss for the “CEOAE+” ears was 33 dB (SD 13), for the “CEOAE-” ears 61 dB (SD 17). The mean OAE amplitude equaled 8.2 dB SPL (SD 4.0). For the 82 measurements, Spearman's correlation coefficient $r_s = -0.39$. (b) Emission amplitude versus the smallest hearing loss of the audiogram. Same as (a); note the fact that many points coincide.

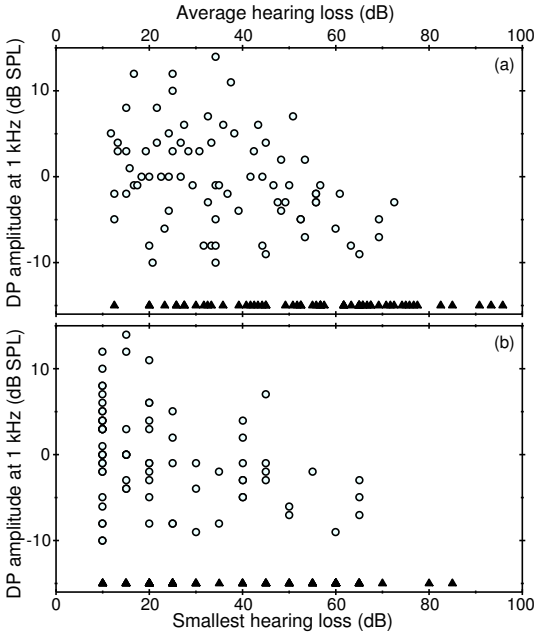


Figure 5.4: Amplitudes of distortion product otoacoustic emissions at 1 kHz as a function of (a) the average hearing loss, and (b) the smallest hearing loss (as Fig. 5.3). The mean average hearing loss for the “DP1+” ears was 36 dB (SD 16), for the “DP1-” ears 55 dB (SD 20). The mean OAE amplitude equaled -0.2 dB SPL (SD 5.6). For the open circles of panel (a), Spearman’s correlation coefficient $r_s = -0.36$. $N_{\text{DP1+}} = 79$, $N_{\text{DP1-}} = 61$.

amined with the scatter plots in Figs. 5.3–5.7. Figure 5.3(a) shows the average hearing loss versus the amplitude of the CEOAEs, for the affected ears. Ears with (“CEOAE+” ears; open circles) and without measurable emissions (“CEOAE-” ears; filled triangles) were included in the graph, the amplitudes of the “CEOAE-” ears consequently being meaningless. As can be observed from this figure, the association between CEOAE amplitude and average hearing loss was weak; Spearman’s rank correlation coefficient $r_s = -0.39$ (for the “CEOAE+” ears). According to a t test, the mean values of the average hearing loss for the “CEOAE+” and the “CEOAE-” ears differed significantly, by 28 dB. Panel (b) shows the smallest hearing loss versus the amplitude of the CEOAEs, for the same ears. Thus, all points

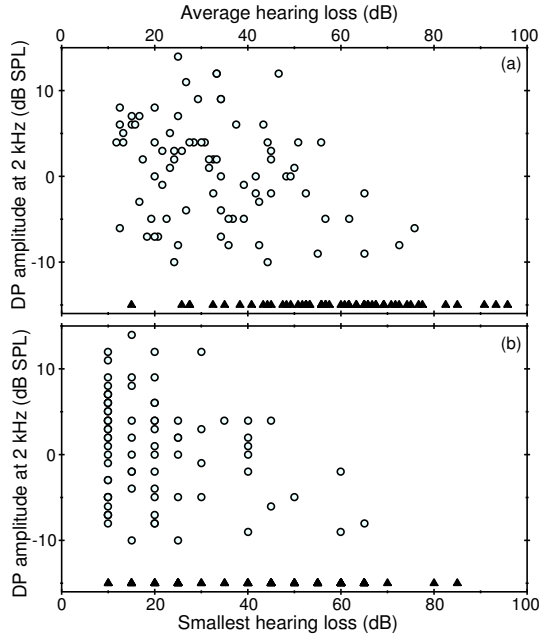


Figure 5.5: Amplitudes of distortion product otoacoustic emissions at 2 kHz as a function of (a) the average hearing loss, and (b) the smallest hearing loss (as Fig. 5.3). The mean average hearing loss for the “DP2+” ears was 33 dB (SD 15), for the “DP2−” ears 59 dB (SD 16). The mean OAE amplitude equaled 0.8 dB SPL (SD 5.9). For the open circles of panel (a), Spearman’s correlation coefficient $r_s = -0.29$. $N_{\text{DP}2+} = 80$, $N_{\text{DP}2-} = 60$.

from panel (a) were, in fact, shifted to the left (by minimally 0 dB, in the case of a flat audiogram), especially in audiograms with one or more better points. Plotted this way, the spread of the points was clearly smaller, showing a maximum value for the emission amplitude for a certain hearing loss. The same procedures were followed for DPOAEs and the outcomes yielded similar results (Figs. 5.4–5.6). For DPOAEs at 1 kHz, Spearman’s rank correlation coefficient $r_s = -0.36$; the mean values of the average hearing loss for the “DP1+” and the “DP1−” ears differed significantly by 19 dB. For DPOAEs at 2 kHz, $r_s = -0.29$, the mean hearing loss differed significantly by 26 dB. For DPOAEs at 4 kHz, there was no correlation; the mean hearing loss differed significantly by 24 dB.

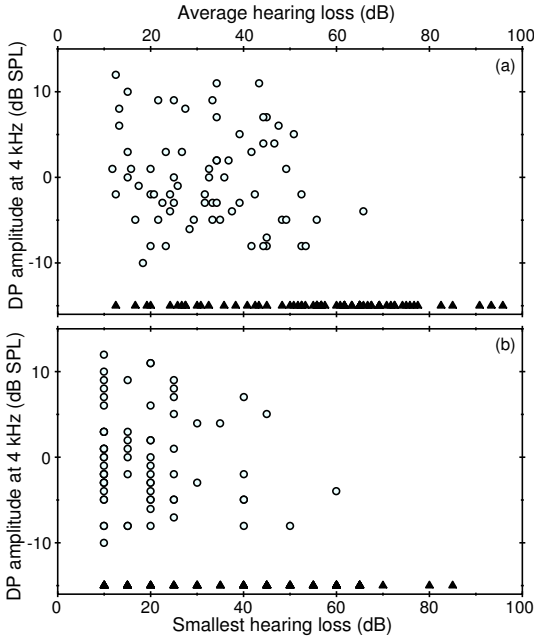


Figure 5.6: Amplitudes of distortion product otoacoustic emissions at 4 kHz as a function of (a) the average hearing loss, and (b) the smallest hearing loss (as Fig. 5.3). The mean average hearing loss for the “DP4+” ears was 32 dB (SD 13), for the “DP4-” ears 56 dB (SD 19). The mean OAE amplitude equaled -0.1 dB SPL (SD 5.6). The open circles of panel (a) were not correlated. $N_{\text{DP4+}} = 70$, $N_{\text{DP4-}} = 70$.

Distinct points of the audiometric threshold also showed some correlations with OAEs (for affected ears). Considering the value of the hearing threshold at 1, 2, or 4 kHz, this variable correlated most strongly with the DPOAE of the same frequency (with $r_s = -0.45$, -0.29 , and -0.34 , respectively); the opposite, that is, starting from the DPOAE value, was not the case. CEOAEs correlated most strongly with the threshold at 1 kHz ($r_s = -0.43$). Therefore, the improvement of correlation with respect to the average hearing loss was small. Furthermore, the outcomes of speech audiometry showed certain correlations with OAE amplitudes: for the maximum speech discrimination and the CEOAE amplitude $r_s = 0.42$, this being the greatest value.

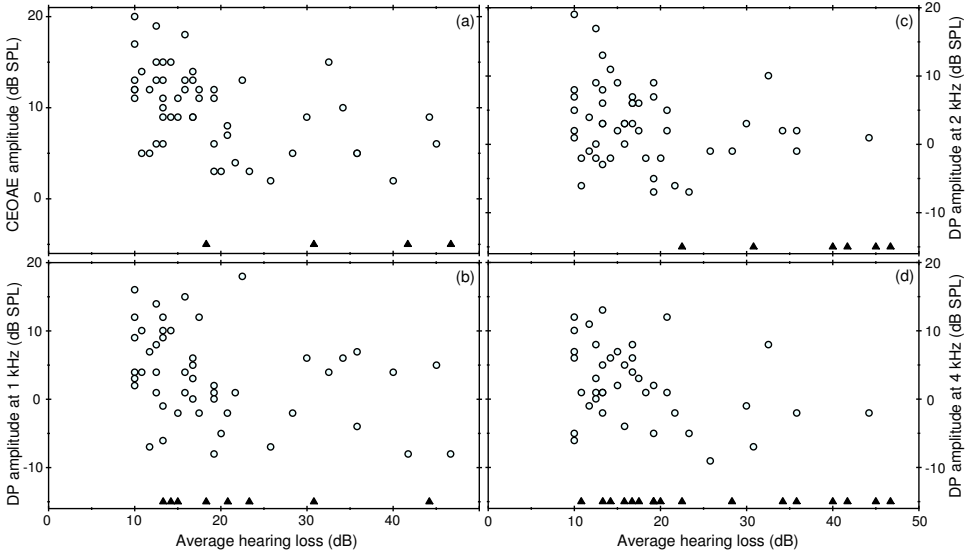


Figure 5.7: Emission amplitude versus the average hearing loss, for the unaffected ears (cf. Figs. 5.3–5.6, note the scale differences). (a) Click-evoked OAEs: mean amplitude = 10.1 dB SPL (SD 4.4); Spearman’s correlation coefficient $r_s = -0.50$; $N_{\text{CEOAE}+} = 53$, $N_{\text{CEOAE}-} = 4$. (b) Distortion product OAEs at 1 kHz: mean amplitude = 3.6 dB SPL (SD 6.6); $r_s = -0.35$ ($P = 0.013$); $N_{\text{DP1}+} = 49$, $N_{\text{DP1}-} = 8$. (c) Distortion product OAEs at 2 kHz: mean amplitude = 3.0 dB SPL (SD 5.6); no correlation; $N_{\text{DP2}+} = 51$, $N_{\text{DP2}-} = 6$. (d) Distortion product OAEs at 4 kHz: mean amplitude = 2.3 dB SPL (SD 5.6); no correlation; $N_{\text{DP4}+} = 40$, $N_{\text{DP4}-} = 17$. Smallest hearing losses were not plotted since 83% of the unaffected ears had a smallest hearing loss of 10 dB.

For the unaffected ears, the correlation of the CEOAE amplitude with average hearing loss was higher in comparison with the affected ears ($r_s = -0.50$). For all DPOAEs, correlations were smaller when present at all. The latter fact was probably due in part to the smaller spread in the hearing losses. Figure 5.7 shows data of the unaffected ears. The mean average hearing losses for ears with and without OAEs were not compared, due to the small number of ears without OAEs. Since most unaffected ears (83%) had a smallest hearing loss of 10 dB, OAE data were not plotted against

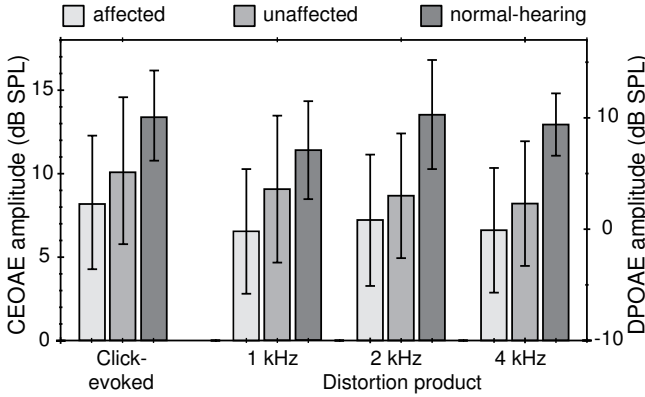


Figure 5.8: Mean amplitudes of different otoacoustic emission types. The mean amplitudes (and SDs) of click-evoked and distortion product OAEs as measured in (1) the affected and (2) unaffected ears of patients with Menière’s disease (cf. Figs. 5.3–5.7), and (3) normal-hearing ears. The left and right ordinate denote the amplitudes of the click-evoked and distortion product OAEs, respectively. Significant differences in mean existed between affected, unaffected, and normal-hearing ears for all emission types ($P < 0.05$). The mean differences between the affected ears of uni- and bilaterally affected patients were not significant for all emission types. Normal-hearing data were taken from Van Huffelen et al. (1998), with $N = 26$.

smallest hearing loss. The mean amplitudes of the OAEs for unaffected ears were larger than for the affected ones (see also Fig. 5.8).

Considering the ears in which an OAE was actually measured, the mean amplitudes of the OAEs were calculated and compared (Fig. 5.8). In addition to the data of the affected and unaffected ears of our patient group, data of normal-hearing ears was included (taken from Van Huffelen et al. 1998). These data were also obtained in our clinic, with the same equipment and experimental setup; therefore, a good comparison could be made. The mean amplitudes of the unaffected ears were larger than those of the unaffected ears for all OAE types ($P < 0.01$, and $P < 0.05$ for DPOAEs at 4 kHz). In turn, the mean amplitudes of the normal-hearing ears were larger than those of the unaffected ears, also for all four OAE types ($P < 0.05$). No significant difference between affected ears of uni- and bilaterally affected patients was present. The mean average hearing loss and the mean smallest hearing loss

(cf. Fig. 5.1) for affected ears were 44 resp. 20 dB HL; for unaffected ears, 20 resp. 11 dB HL; and for normal-hearing ears both values equaled 10 dB HL (strongly influenced by our audiometric limit of 10 dB HL).

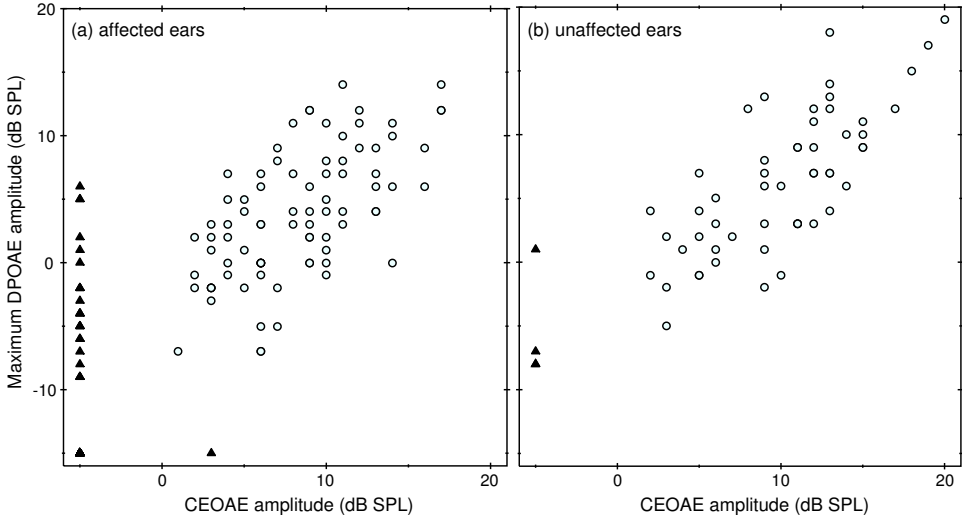


Figure 5.9: The maximum value of the 3 distortion product amplitudes versus the amplitude of the click-evoked otoacoustic emissions, for (a) the affected, and (b) the unaffected ears. The open circles indicate ears from which an emission was measured (OAE+); the filled triangles indicate ears from which no emission could be measured (OAE-). The latter points' amplitude is consequently meaningless [(a): $N_{\text{OAE}+} = 82$, $N_{\text{OAE}-} = 60$; (b): $N_{\text{OAE}+} = 54$, $N_{\text{OAE}-} = 4$]. For the affected and unaffected ears, Spearman's correlation coefficients equaled $r_s = 0.63$ and $r_s = 0.74$, respectively.

Relating the different OAEs, Fig. 5.9 shows the amplitude of the click-evoked otoacoustic emissions versus the maximum value of the 3 distortion product amplitudes (at 1, 2, and 4 kHz), for (a) the affected, and (b) the unaffected ears. In this way, Spearman's rank correlation coefficient r_s was larger than for any of the separate distortion products. For the affected ears, $r_s = 0.63$ and for the unaffected ears, $r_s = 0.74$. As was noted above, the mean OAE amplitudes for unaffected ears were larger than for affected ears.

For affected ears, the duration of the affection differed significantly between the ears with and without DPOAEs at 2 kHz: 5.9 versus 8.8 years

($P < 0.05$). Furthermore, the duration of the hearing loss showed an association with the presence of three OAEs (click-evoked, and distortion products at 2 and 4 kHz); the mean duration for ears with these OAEs was 4.7–5.0 years, and for ears without OAEs 7.9–8.5 years ($P < 0.01$). Other variables, such as age, gender, and severity of the different aspects of the disease, did not show any correlation or association with OAE incidence or amplitude.

5.4 Discussion

The characteristics of click-evoked and distortion product otoacoustic emissions in ears of patients with Menière's disease were investigated. We compared the properties of the affected and unaffected (i.e., contralateral) ears of 100 patients. The incidence of OAEs in affected ears (56%) was lower than in unaffected ears (85%; see Fig. 5.2). The mean emission amplitude in affected ears was also significantly lower (2.6 dB). The affected ears of uni- and bilaterally affected patients showed no differences. Further, ears with OAEs clearly showed smaller hearing losses than ears without OAEs (24 dB difference). Correlations between average hearing loss and the various emission amplitudes were present, although these were not very strong; when plotted against the smallest hearing loss, a certain upper boundary for the emission amplitude was present (see Figs. 5.3–5.6).

Menière's disease is usually defined by the diagnostic triad of episodic vertigo, fluctuant sensorineural hearing loss, and tinnitus, although sometimes a feeling of fullness in the ear is also included (for an overview see Kiang 1989 and Horner 1993). The hearing loss involved in Menière's disease is described as increasing; its nature is thought to be related to hair-cell pathology. Although still uncertain, the pathophysiology of these symptoms is thought to be an endolymphatic hydrops. Various causes have been hypothesized for this increased amount of endolymph, mostly tracing back to an imbalance between the secretion and reabsorption of the cochlear fluids (e.g., Kimura 1982; Horner 1993; Dunnebier et al. 2000). Mechanisms yielding a possible endolymphatic hydrops via a pressure-buildup in the inner ear have also been studied by many authors through manipulation of the endo- and perilymphatic pressures (e.g., Takeuchi et al. 1991; Wit et al. 2000). In addition, histological studies in the guinea pig point out that induced endolymphatic hydrops is accompanied by hair-cell damage (Dunnebier et al. 2000).

Since the publication of Kemp (1978), many authors have performed measurements of otoacoustic emissions. At present, OAEs are commonly regarded as originating from the inner ear, presumably through the electromotile responses of outer hair cells (for a review see Probst et al. 1991). From the very first measurements (Kemp 1978), it was noted that OAEs were not present in ears with cochlear deafness; nowadays, OAEs are widely used as an objective screening tool for cochlear hearing loss. Numerous reports have shown that OAEs are highly affected by cochlear pathology, although some disagreement exists on the amount of hearing loss at which OAEs are absent. Generally, otoacoustic emissions are detectable when average hearing (from 0.25 to 8 kHz) is better than 20 dB HL, and are undetectable if the average hearing loss exceeds approximately 45 dB HL (e.g., Probst et al. 1987; Bonfils et al. 1988; Prieve et al. 1993; Wagner and Plinkert 1999). The observations are frequency dependent; that is, OAEs are not found in frequency regions where hearing is below approximately 30 dB HL, while OAE components may be present in adjacent frequency regions, in the same ear, where hearing is relatively normal (e.g., Collet et al. 1993; Gorga et al. 1993; Prieve et al. 1996; Tognola et al. 1999). Nevertheless, a strict relation with which the audiogram could be predicted from OAE measurements could not be established (e.g., Mauermann et al. 1999; Gorga et al. 2000). Tognola et al. (1999) conclude that the presence of a CEOAE component is always associated with thresholds ≤ 25 dB HL, while, on the other hand, an absence is equally associated with normal or abnormal hearing. We studied ears with various types and degrees of hearing loss and observed all four OAEs correlating weakly with the — averaged — thresholds. Considering the fact that partly-normal hearing possibly yields OAEs, we plotted the OAE amplitudes against the smallest hearing loss (for the affected ears). This indeed showed a more determinate upper boundary for the OAE amplitudes at a certain value of the smallest hearing loss (Figs. 5.3–5.6). It could be argued that not all threshold frequencies equally contribute to the OAEs (Probst et al. 1987). However, since this may be due to external factors and it is not clear to what extent the dependence holds, we included all threshold values. Furthermore, we note that, regarding CEOAEs, measures like signal-to-noise ratio and reproducibility could have yielded beneficial information. However, Prieve et al. (1993) report these measures to identify hearing loss equally well. Altogether, our observations confirm the view that OAEs are associated with normal or near-normal hearing, whereas, on

the other hand, absent or weak OAEs are either associated with normal or abnormal hearing.

Otoacoustic emissions in patients with Menière's disease have previously been investigated. Firstly, otoacoustic emissions of affected and unaffected ears were found to differ in level (Harris and Probst 1992; Pérez et al. 1997; Van Huffelen et al. 1998) and spectral composition (Harris and Probst 1992); however, these were strongly influenced by the audiometric configuration. Moreover, OAEs from unaffected and normal-hearing ears differed, although hearing was not always completely normal in these ears. These findings were confirmed by our observations. The OAE incidence, as well as the mean amplitude, were lower in affected than in unaffected ears; and, in turn, the mean amplitude in unaffected ears was lower than in normal-hearing ears. Our audiometric limit of 10 dB HL caused the comparison between the smallest hearing losses of the various groups to be restricted. Secondly, the duration of the disease was of no influence in the study of Harris and Probst (1992), whereas Cianfrone et al. (2000) found a difference in duration between the emitting and non-emitting ears (2.7 versus 5.9 years). Furthermore, Pérez et al. (1997) report a correlation with a certain staging, however, not being the duration. We found a difference in duration of the disease only between the ears emitting and non-emitting a DPOAE at 2 kHz (5.9 versus 8.8 years; $P < 0.05$). The duration of the hearing loss was found to differ between the emitting and non-emitting ears for three OAE types ($P < 0.01$). These findings could offer possibilities for a certain staging. In the third place, most authors mention the — surprising — presence of OAEs in ears with large hearing loss (e.g., Harris and Probst 1992; Van Huffelen et al. 1998; Cianfrone et al. 2000). Our observations also include ears with considerable loss and still OAEs, but they fit into the complete picture (see Fig. 5.3b). In summary, OAEs in affected and unaffected ears of patients with Menière's disease clearly show properties differing from each other and from OAEs in normal-hearing ears. There is no strong reason not to ascribe these differences to the hearing loss involved. A more precise comparison can hardly be realized because OAEs show a large spread in ears with various degrees of hearing loss.

Considering the fact that the various OAE types correlate with hearing loss, it is likely that OAEs correlate with each other. Spectral relationships between OAEs have been demonstrated before (e.g., Zwicker and Schloth 1984). Since we measured one broadband OAE (click-evoked) and three

narrowband OAEs (the distortion products), we plotted the CEOAE amplitude against the maximum value of the three DPOAEs (Fig. 5.9). In this way, a cochlea partially generating emissions yields high values for both variables (see previous paragraph). The fact that the unaffected ears yielded a stronger correlation (0.74) than the affected ears (0.63), cannot yet be explained. Thus, the correlations we observed between the OAEs confirm the view that OAEs are interrelated, although a precise spectral examination could not be made.

In conclusion, we observed the incidence as well as the mean amplitude of click-evoked and three distortion product OAEs being lower in affected than in unaffected ears; and, in turn, the mean amplitude in unaffected ears to be lower than in normal-hearing ears. These differences are likely to be caused by the hearing loss involved, although a more accurate information of the audiogram could yield additional insight. Information concerning the hearing threshold at more frequencies (e.g., by Békésy-tracking; see Horst and De Kleine 1999) and a precise determination of the level (above and below 0 dB HL) could eventually yield a more definite relation with otoacoustic emissions.