Methodological paper

Otoacoustic detection of risk of early hearing loss in ears with normal audiograms: A 3-year follow-up study

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A 3-year follow-up study was carried out on a population of pilots aged 20–40 years (n = 521). Data collection consisted of tonal audiograms, DPOAEs measurements with a calculation of an index of abnormality (the IaDPOAE). Of the 521 pilots enrolled, 350 (67%) had follow-up data 3 years later. In pilots with normal audiograms (n = 219, all frequencies = 10 dB HL), we observed the occurrence of hearing threshold shifts after 3 years depending on whether the IaDPOAE was initially high (group 1) or low (group 2). We used this index to test the hypothesis that reduced DPOAE levels are potential ear vulnerability biomarkers in apparent normal hearing ears.

Results: After a 3-year follow-up, the initial IaDPOAE in normal hearing subjects was correlated with final noise-induced hearing threshold shifts at high frequencies (p < 0.01). The occurrence of abnormal audiograms was significantly higher in group 1 compared to group 2 (p = 0.003). In group 1, 13% of audiograms were found with at least one frequency > 25 dB HL compared to 3% of audiograms in group 2. In both groups, impairments occurred at high frequencies and hearing in the 4 kHz frequency range was significantly more impaired in group 1 (p = 0.035).

Group 1 was associated with a relative risk of 2.29 (95% CI 1.26–4.16, p = 0.005) of sustaining early hearing loss. There was no significant differences between groups for age and noise exposure.

Discussion: In adults with a normal audiogram, ear vulnerability to noise could be elicited by the use of objective DPOAE measurements. A high IaDPOAE that corresponded to reduced DPOAE levels constitutes a risk for early hearing loss. This study emphasised the interest of DPOAE measurements in public health and occupational noise prevention policies. The IaDPOAE calculation may also be interesting for clinicians because no DPOAE index of abnormality is currently available.

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1. Introduction

For many years now occupational physicians and military physicians have been interested in using an objective index that could help them to decide if an individual is more at risk of early hearing loss, especially if exposed to noise.

Studies carried out by Kemp (Kemp et al., 1990), Gorga and colleagues (Dorn et al., 1999; Dorn et al., 2001; Gorga et al., 1997; Gorga et al., 1999; Gorga et al., 2000; Gorga et al., 2002; Gorga et al., 2003; Neely et al., 2003), on the development of research on distortion product otoacoustic emissions (DPOAEs) stimulated the hope of finding such an index, but there are almost no longitudinal studies in normal hearing adults. DPOAEs rely on the contractile properties of the outer hair cells. In response to tonal sound stimuli, these cells can generate retrograde wave sounds, the intensity of which can be captured and recorded by a very sensitive probe microphone in the external auditory canal. DPOAEs are essentially used to study cochlear oxidative stress with regard to deleterious agents in numerous studies on animals because they can provide an objective measurement of outer hair cell selective impairments at various corresponding frequency range of the cochlea (Avan et al., 1990; Lataye et al., 2003; Lukashkin et al., 2002; Mom et al., 1999; van Dijk et al., 2003; Whitehead et al., 1992). In neonates, absence of DPOAEs is a sign of severe deafness (Janssen et al., 2006; Swanepoel de et al., 2006; Tsui et al., 2008;
Wagner et al., 2008; Xu and Li, 2005). In adults, the presence of hearing loss on audiograms is usually associated with low or absent DPOAEs (Cianfrone et al., 2000; Gorga et al., 1999; Harris, 1990; Johnson et al., 2007; Nagy et al., 2002; Sisto et al., 2007; Stover et al., 1996; Wagner et al., 2005). In some studies, normal hearing subjects have been demonstrated to have reduced DPOAEs (Job and Nottet, 2002; Lutman and Deeks, 1999; Shiomi et al., 1997) compared to others, and the physician did not have an appropriate scientific method of deciding if their DPOAE results could reveal a vulnerability that could lead to a more rapid decrease of hearing capabilities. Some factors had been reported in cross-sectional studies in normal hearing subjects to cause DPOAEs reductions. Age in normal hearing subjects has been shown to reduce DPOAEs (Uchida et al., 2008). Also, reduced levels of DPOAEs were shown by Shiomi (Shiomi et al., 1997) and Job (Job et al., 2007) to be associated with susceptibility to tinnitus. Because of the lack of longitudinal studies, the use of DPOAEs is still limited to the use of deafness detection in neonates and to the use of objective confirmations of hearing damage when audiograms are abnormal in adults.

In this study, we use a longitudinal study to demonstrate that DPOAEs could be useful to elicit a vulnerability at noise exposure. This vulnerability led to a more rapid impairment of hearing.

2. Materials and methods

2.1. Subjects

We chose a population of pilots because noise exposure was intense, regular, continuous and without acute acoustic trauma due to impulsive noise. Regular noise exposure may be calibrated by the number of hours of flight. Acoustic pressure ranges from 90 to 110 dB A (idling speed) and from 110 to 140 dB A (flat-out speed). Protection is provided by headsets.

In all, data from 521 French pilots aged 20–40 years were collected by an ear–nose–throat (ENT) specialist, between October 2003 and October 2004 at the aircrew medical centre, near Paris. Data were collected during pilots’ annual medical check-up and far from periods of noise exposure (i.e., 24 h, at least). Written, informed consent was obtained from each subject.

Between October 2006 and October 2007, pilots audiometric data were recollected with the same device, the same clinician and the same experimental protocol. Data from the two date points were compared.

2.2. Questionnaire

Pilots were interviewed by the ENT specialist with a standardized questionnaire. Age and gender were recorded. For aircraft noise exposure, hours of flight, years of flight, type of aircraft (i.e., fighter, transport and helicopter) flown and type of headsets were recorded.

Risk-factors or symptoms associated with noise that could affect hearing include: history of acute acoustic trauma, noisy leisure exposure, presence of chronic tinnitus, history of otitis media, otitis due to air pressure and head injury.

2.3. Audiological examinations

Prior to tonal audiograms, the pilots underwent an otoscopy to check that there were no obstructions in the ear canal.

2.4. Tonal audiograms

Tonal audiograms were collected in a sound proof simple-walled booth (Elstar, Paris) with an average noise attenuation of 35 dB SPL. Hearing levels (HL) were measured by trained clinician at seven pure tone frequencies (0.250, 0.500, 1, 2, 3, 4 and 8 kHz) in each ear. The sound level was systematically increased and decreased by steps of 5 dB to find the critical value (i.e. the threshold) that separated the audible from the inaudible range, using a clinical audiometer (Interacoustic; model AC40; Assens, Denmark). The audiometer was calibrated according to the standards of the International Organization for Standardization, ISO 389 and checked every year. During the test, if sounds intensity was audible at 10 dB HL lower intensities were not tested.

2.5. DP-grams

Following an ear examination and audiograms, DPOAEs were measured using commercial equipment (Graison-Stadler GSI 60 system). The stimuli consisted of two pure tones (f1 and f2; f2/ f1 = 1.2) presented simultaneously, with the lower frequency stimulus at 65 dB SPL and the higher frequency primary tone at 55 dB SPL. Intensity levels in dB of the 2f1–f2 DPOAEs and corresponding noise floor were registered and displayed as a function of f2. DPgrams at eight points/octave over a four-octave-frequency range, were recorded (a total of 32 measurements). The total duration of measurements per ear was 3 min. During measurements, noise floors were typically –10 ± 5 dB, over the 1- and 8-kHz regions and –20 ± 5 dB over the 2–4-kHz frequency ranges. DPOAE responses were valid when signal/noise ratio was at least ≥2 dB.

2.6. The DPOAE index of abnormality, IaDPOAE

We used an index of abnormality to test our hypothesis that reduced DPOAEs in apparent normal hearing subjects might reveal a vulnerability to noise.

2.7. Study providing DPOAE reference curves

We extracted reference curve values from a previous study where the DP-gram protocol and the DPOAE system was the same as in the present study (Job and Nottet, 2002). The study took place in the medical department of the army recruitment centre. Subjects were aged 18–24 years. As a pre-selection criterion, only those having normal bilateral audiograms (i.e., <15 dB HL on 0.5, 1, 2, 4 and 8 kHz, with a 5-dB step method) were examined and interviewed.

Otoscopies were carried out with an otomicroscope by an otolaryngologist and by other physicians trained by the specialist. Type A tympanograms were required (i.e., values comprised from 0.5 to 1.6 ml for middle ear mobility and ±50 daPa for the pressure peak). Impedance tympanometric measurements were conducted using standard clinical instrumentation, a 226 Hz probe tone (Graison-Stadler GSI 75). In addition, young subjects were administered a short questionnaire about their possible histories of otitis media (i.e., repeated episodes or myringotomy). This information was checked in the subjects’ personal health records. Subjects with eardrum abnormalities of any kind, including dull, cicatricial, bulging, retracted or tympanosclerotic eardrum were not in the reference group. Lastly, we checked that they had no particular intense exposure to noise. Finally, DPOAEs data from 69 subjects (119 ears) were considered as a reference population for this particular Dpgram protocol. No audiogram frequencies exceeded 10 dB HL in this ear group.

In Fig. 1, DPOAEs means and standard deviations (−1 SD and −2 SD) for each DPOAE values could be implemented in the software of the GSI system or any open system and could be displayed for each ear tested. Thus, it was possible for the clinician to calculate a statistical index of abnormal DPOAEs (the IaDPOAE) for each ear at the end of the test. Finally, The IaDPOAE calculation was standardized using a computer program.
2.8. IaDPOAE calculation

Each new DP-gram had its IaDPOAE calculated. Every point (intensity measure) of each DP-gram corresponded to a weighted score based on standard deviations (SD) of mean of the reference population (Fig. 1).

Each intensity (8 measures/octave = 8 × 4 octaves = 32 measures) were either above the 1 SD curve (Score = 0), between the 1 SD and 2 SD curves (score = 1), or under the 2 SD curve (score = 2). The smallest score was given to the intensities of DPOAEs exactly equal to the standard deviations. The index is the sum of the weights of all the measures (i.e., 32) divided by the sum of the highest weighted score (i.e., 2 × 32), therefore, a score of 64 represented 100% abnormality. This was the case if all intensity measures were under the 2 SD curve. This index could be generalized to DP-grams with different numbers of points.

All hearing frequency ranges are involved in the IaDPOAE calculation. This is particularly essential in the making of a general vulnerability index, to avoid giving too much weight to accidental deleterious events that could occur to hearing. In theory, acoustic accidents are relatively independent of vulnerability, are mostly frequency specific and extremely variable. For this reason and for the purpose of the study, we chose a population with regular continuous noise exposure.

2.9. Sensitivity and specificity of IaDPOAE

Continuous noise exposure led to progressive hearing loss, consequently hearing loss is a continuous variable. In Fig. 2A, the ears of the 521 pilots show that, as expected, losses on audiograms are associated with higher IaDPOAEs (Pearson cor. coef., r = 0.46, p < 0.001). As in previous studies (Job and Nottet, 2002; Shiomi et al., 1997) some reduced DPOAEs or high IaDPOAEs were also associated with normal audiograms (75th percentile + outliers).

To aid the process of medical prognosis, we try to obtain an IaDPOAE cut-off value to separate impaired hearing ears from normal hearing ears. For this, we used data from initial measurements (n = 521) and the ROC-curve method (Fig. 2B).

Despite the fact that there is no strict gold standard to define what is normal hearing, clinical practitioners and several publications dealing with normative hearing thresholds, established an empirically-based dividing line between impaired and normal audiograms at around 20–30 dB HL (Avan and Bonfils, 2005; Dorn et al., 1999; Gorga et al., 1997).

The best IaDPOAE cut-off point should be the value that gives the best compromise between sensitivity and specificity. The best IaDPOAE cut-off value calculated by ROC analysis was found to be around 15% when the entire population was split in two between ears with at least one frequency ≥ 25 dB HL and the others. The sensitivity and specificity were 72% and 64%, respectively (area = 0.750, 95% CI 0.71–0.78, p < 0.001). When we applied a more stringent definition of normal audiograms (i.e., all frequencies = 10 dB HL). The best cut-off value was also found at around 15%. The sensitivity and specificity were 71% and 74%, respectively (area = 0.803, 95% CI 0.76–0.84, p < 0.001). Thereby in ears with all frequencies = 10 dB HL, we compared two groups: group 1 associated with an IaDPOAE >15% and group 2 associated with an IaDPOAE ≤ 15%.

In addition, we performed a quantitative analysis, excluding the notion of cut-off, in order to test our hypothesis that normal hearing thresholds but high IaDPOAE may reflect a vulnerability that could lead to an early subsequent hearing loss. For this, we observed correlations between initial IaDPOAE and final hearing thresholds in normal hearing pilots of the beginning of the study (i.e., all frequencies = 10 dB HL).

2.10. Statistical analysis

We used SPSS V.13.0 software (Chicago, IL) for all the analyses (i.e., ROC curves, Chi-square tests, risk estimates for hearing loss...
and comparisons between groups). Intra-individual difference in hearing thresholds or DPOAE values between the year 2004 and 2007 were tested by paired difference \( t \)-tests. Comparisons of quantitative values between groups were tested by independent \( t \)-tests. \( p \) values <0.05 were considered significant. Pearson correlation coefficients were used to test correlations between initial value of IaDPOAE and final hearing thresholds at each frequency of audiogram.

3. Results

3.1. General follow-up

350 pilots were re-examined under the same conditions 3 years later, by the same clinician. The missing 171 pilots had their annual check-up with another ENT clinician and no DPOAE measurements were taken. Pilots flew for about 700 h (±400 h) during the period of the study. Significant changes on audiograms appeared beginning at 3 kHz (Fig. 3) with a maximum mean loss difference of 1.7 ± 10 dB HL at 8 kHz for left ears. DP-grams also showed as well significant changes with a reduction of DPOAEs levels from 0.6 to 6 kHz. The maximum reduction occurred in the 3–5 kHz range with 1.8 ± 3 dB SPL and 1.6 ± 3 dB SPL for right and left ears respectively (\( p < 0.001 \)). Concerning the evolution of the IaDPOAE, the index was increased (\( p < 0.001 \)) for right and left ears, by +3.5 ± 8% and +3.7 ± 9%, respectively.

3.2. Ears with all frequencies equal to 10 dB HL

Among the 350 pilots, 160 pilots had, at least one ear with all frequencies equal to 10 dB HL, some had both, at the beginning of the study. Consequently we found a total of 219 ears with these initial hearing thresholds. Among those ears, 63 had an IaDPOAE > 15% (group 1); the IaDPOAE mean in this ear group was 27 ± 12%. In contrast, 156 had an IaDPOAE ≤ 15% (group 2); the IaDPOAE mean in this group was 5 ± 4%. IaDPOAE being an index of DPOAE abnormality, the difference between groups meant that group 1 had reduced DPOAE intensities compared to group 2. There was no difference in age between these groups (31 ± 5 years vs 30 ± 5 years; \( p = 0.058 \)) neither hours of flight during the 3-year period (677 ± 405 h vs 705 ± 400 h; \( p = 0.631 \)). We also compared prevalence of head trauma, acute acoustic trauma, history of otitis media, otitis due to air pressure, presence of tinnitus and noisy leisure activities; there were no differences between groups despite (close to the significant limit) a higher prevalence for a history of otitis media in group 1 than in group 2 (i.e., 17% vs 10%, respectively).

3.3. Occurrence of hearing thresholds shifts

In Table 1A, the occurrence of hearing threshold shifts on audiograms in group 1 were significantly higher at the end of the study (\( \chi^2 = 11.44, p = 0.003 \)). Indeed, as we can see in Table 1A, 13% initial normal audiograms of group 1 were found with at least one fre-
Frequency ≥ 25 dB HL instead of 3% in group 2. Only 44% normal audiograms remained normal after 3 years in group 1 as opposed to 65% normal audiograms in group 2. In Table 1B, when ears with threshold shifts were pooled (i.e., 56% in group 1 and 35% in group 2, respectively); the association between occurrence of impairments and initial IaDPOAE groups was still significant ($\chi^2 = 7.63$, $p = 0.005$). Group 1 was associated with a relative risk of 2.29 (95% CI 1.26–4.16, $p = 0.005$) of having early hearing loss. This result means that the risk of rapidly developing a hearing impairment which is visible on an audiogram at exposure to noise is twice as important in normal hearing subjects with the IaDPOAE > 15%.

In order to demonstrate more objectively than splitting the subjects into artificial IaDPOAE categories, that initial IaDPOAE could predict hearing loss in normal ears; we performed in parallel a quantitative correlation analysis in the 219 ears (Table 2). The results confirmed that final hearing losses (beginning from 2 to 8 kHz) was significantly correlated with the initial IaDPOAE.

Hearing shifts were focused essentially at high frequencies as shown in Fig. 4. Shifts showed the biggest shift for both groups at 8 kHz: group 2 showed a difference of about 4.5 dB HL whereas group 1 showed a difference of about 5.5 dB HL, nevertheless 8 kHz-shifts were not significant between groups except at 4 kHz ($p = 0.035$). Group 1 was more impaired than group 2 at this frequency range. This hearing frequency range is particularly sensitive to noise.

In contrast on final DP-grams (Fig. 4), DPOAE reductions occurred at almost all frequencies but were not significantly different between groups. Group 1 initial DPOAEs were lower than group 2 and at all frequency range ($p < 0.001$). Differences were nevertheless smaller at the lowest and highest frequencies.

4. Discussion

In this study, we sought to determine whether the measurements of DPOAEs added value to commonly used tonal audiograms. We also aimed to assess the use of this measurements to predict vulnerability of ears even when the audiogram is clinically normal.

The result of this large prospective study showed that DPOAEs could be biomarkers of vulnerability to noise. Our quantitative analysis clearly showed that in normal ears initial DPOAEs could predict final hearing loss because they are well correlated. The IaDPOAE > 15% also predicted whether or not more rapid increases of hearing loss would occur at noise exposure. Reduced DPOAEs at almost all frequencies corresponded to a high IaDPOAE and revealed a susceptibility of being more easily and severely impaired at high frequency by a harmful noisy event. The question of which mechanism would be involved in such a susceptibility is now open.

### Table 1

Assessment of the risk of early hearing loss in subjects with normal audiograms (all frequencies = 10 dB HL). (A) Cross-table showing the association between the occurrence of hearing thresholds shifts and the prevalence of an initial high IaDPOAE. (B) 2 x 2 Cross-table showing total occurrence of hearing threshold shifts after 3 years of noise exposure and risk estimates of hearing impairments when initial IaDPOAE > 15%.

#### A

**AUDIOMGRAMS 2007**

<table>
<thead>
<tr>
<th></th>
<th>At least one &gt;=25 dB HL</th>
<th>At least one = 15-20 dBHL</th>
<th>All = 10 dBHL</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>AUDIOMGRAMS 2004 All = 10 dB HL</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>IaDPOAE &gt; 15%</td>
<td>n 8</td>
<td>27</td>
<td>28</td>
<td>63</td>
</tr>
<tr>
<td>IaDPOAE &lt;= 15%</td>
<td>n 12.7%</td>
<td>42.9%</td>
<td>44.4%</td>
<td>100%</td>
</tr>
<tr>
<td>Total</td>
<td>n 13</td>
<td>77</td>
<td>129</td>
<td>219</td>
</tr>
<tr>
<td></td>
<td>5.9%</td>
<td>35.2%</td>
<td>58.9%</td>
<td>100%</td>
</tr>
</tbody>
</table>

#### B

**Audiograms 2007**

<table>
<thead>
<tr>
<th></th>
<th>At least one &gt;10dBHL</th>
<th>All = 10 dBHL</th>
<th>Total</th>
<th>Risk Estimate</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Audiograms 2004 All = 10 dBHL</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>IaDPOAE &gt;15%</td>
<td>n 35</td>
<td>28</td>
<td>63</td>
<td></td>
</tr>
<tr>
<td>IaDPOAE &lt;=15%</td>
<td>n 55</td>
<td>101</td>
<td>156</td>
<td></td>
</tr>
<tr>
<td>Total</td>
<td>n 90</td>
<td>129</td>
<td>219</td>
<td>Odds ratio for laDPOAE &gt;15% 2.295 1.265 4.165</td>
</tr>
<tr>
<td></td>
<td>41.1%</td>
<td>58.9%</td>
<td>100.0%</td>
<td>N of Valid Cases 219</td>
</tr>
</tbody>
</table>
The vulnerability could originate either in middle ear or in outer hair cells or both; it is not currently possible to identify the role played by each part at a subclinical state and because they are intrinsically linked. Indeed, we have already demonstrated that in subjects with antecedent of otitis media and normal audiograms (up to 20 dB HL), DPOAEs were reduced (Job and Nottet, 2002); this antecedent had been shown to potentiate hearing loss and to increase the risk of acute acoustic trauma and tinnitus in cases of exposure to noise (Job et al., 1999). Middle ear muscles reflex has often been shown to be missing in adults with this antecedent (Greisen and Neergaard, 1975; Kazanas and Maw, 1994; Renvall and Holmqvist, 1976). This effect could be due to partial ossicular chain fixation. But before total ossicular chain fixation, which would normally reduce acoustic energy into the cochlea, abnormal ossicular movements (due to inflammation-induced necrosis of middle ear and Eustachian muscles) could promote impairments due to the missing protective reflex and the possibility of high acoustic pressure entering the cochlea. Also the possibility of labyrinthitis following otitis media cannot be excluded. Impairments could therefore be the results of inflammatory processes in the middle ear by disruption of the blood-labyrinth barrier and contacts with pathogenic germs via the round windows. In this study, we did not find a significant difference in the prevalence of history of otitis media despite a higher prevalence in group 1 than in group 2. What ever the aetiology, our results suggest that any subclinical dysfunction of the middle ear or cochlea outer hair cells could slightly reduce DPOAEs and could influence the issue of hearing performance in individuals exposed to noise.

To our knowledge, this is the first prospective study that has proposed a statistical indicator of abnormality of DPOAEs to grade DPOAEs abnormalities and detect a risk of early hearing loss in normal hearing subjects exposed to noise.

DPOAEs are objective measures and are more sensitive to detect sub-clinical impairments than behaviournal conventional tonal audiometry. The question of whether a finer tonal audiometry such as Bekesy tracking would have separated the two groups of normal hearing subjects had been raised. However it seems that Bekesy audiometry is able to detect abnormal microstructures but there was no correlation with reduced DPOAEs (Avan and Bonfils, 2005; Lutman and Deeks, 1999). Should we have screened under 10dB HL with conventional audiometry to be able to assess the risk of early hearing loss and to detect subclinical impairments in a normal hearing population? It appears that DPOAEs could be a sensitive tool for this type of detection.

In this study, the follow-up is limited to 3 years, but the differences between groups might have been higher with a 5-year follow-up or more.

The index of abnormality presented here is only usable with the DPOAE parameters define in the materials and methods. The cut-off value of 15% that separated impaired from normal hearing ears was used for the purpose of the study with a population exposed to continuous noise, this value might be used as well for any regular continuous noise exposure (i.e., industrial, regular noisy activities, etc.). In case of impulse noise exposure, the vulnerability index might not predict temporary threshold shifts (TTS), but possibly the prognosis of recovery after an acute acoustic trauma. In that case, the DPOAE might be used as a covariate to take into account in the progression of hearing recovery or in assessments of therapeutical agents.

We encourage the use of DPOAE measurements in addition to tonal audiograms, more specifically when audiograms are defined as normal. They could be useful in prevention policies directed to any population exposed or potentially exposed to noise (e.g., teenagers, industrial workers, military personnel, etc.).

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References


Lutman, M.E., Deeks, J. 1999. Correspondence amongst microstructure patterns observed in otoacoustic emissions and Bekesy audiometry. Audiology 38, 263–266.


