Latent cochlear damage in personal stereo users: a study based on click-evoked otoacoustic emissions

Eric L LePage and Narelle M Murray

Abstract

Objective: To assess the effects of use of personal stereo systems (PS) on hearing by means of the objective measure of transient-evoked otoacoustic emissions.

Participants and setting: People aged between 10 and 59 years who had otoacoustic emissions recorded by the National Acoustic Laboratories between 1989 and 1997 were eligible for inclusion. Recordings from participants with hereditary disorders or any form of aural disease (eg, otitis media, otosclerosis, fluctuant hearing loss, Meniere's syndrome, or exposure to ototoxic substances) were excluded.

Methods: Transient-evoked otoacoustic emission (TEOAE) records were obtained with a standard 260 repetitions of an 80 dB train of clicks used for recording outer hair cell activity. The measure of otoacoustic emission strength was the Otodynamics ILO88 variable Waverepro%. For each participant, all the key factors relating to their hearing history were assessed from patient referral information or from demographic information obtained in writing at the time of recording either in the form of a detailed questionnaire or verbal assessment. Otoacoustic emission data were analysed according to age, industrial noise exposure and personal stereo use.

Results: Usable otoacoustic emission records were obtained from 1724 people (1066 males and 658 females). Otoacoustic emission strength declined with age, and was significantly lower in males than females, lower in people exposed to industrial noise than those not exposed, and significantly lower in users of personal

stereo systems than non-users. People with both kinds of noise exposure had values which were significantly lower again, indicating an additive effect.

Conclusions: As only 39 people with PS exposure admitted any hearing problems, decline in otoacoustic emission strength forewarns premature hearing loss in personal stereo users.

Introduction

The use of pure tone audiometry to assess the potentially harmful effects of amplified music on young people's hearing has failed to show any marked effect.[1-3] However, good grounds for concern remain as (i) the sound-pressure levels generated by live and recorded rock music are associated with premature hearing loss in industrial workers,[4,5] (ii) inexpensive stereo components have the capacity to generate high sound levels,[6] and (iii) the preferred listening levels of those listening to rock music through earphones in "Walkman"-style headsets is high -- an average of 95dB(A) for females and 97dB(A) for males, with an overall range of 75-110dB(A)[7-16] ("A" signifies the standard "A-weighting" correction to indicate approximately equal loudness across the audible frequency range). One research group felt that "further research may reveal more sensitive measures of cochlear damage than pure tone audiometry".[2] One such measure may be otoacoustic emissions, the principle of which is described in Box 1.

In this study, we examined the effects of personal stereo use on transient-evoked otoacoustic emissions (see Box 1) to investigate the possibility that this technique may indeed be a more sensitive method of early detection of ear damage resulting from sound amplification. To shed light on the significance of our findings, we compared people exposed to personal stereo use with those exposed to industrial noise -- a recognised high risk form of exposure.

Methods

The protocol we used for obtaining transient-evoked otoacoustic emission records is described in Box 1.

The data for this study were obtained from the records of

2

some 2500 people tested as part of the National Acoustic Laboratories research program between 1989 and 1997. Information about participants' hearing histories was obtained from details supplied by referring clinics or otologists (12%), standardised questionnaires filled out (60%) or verbal evaluation (18%) at the time of recording, or from Australian Hearing's NALCAM (National Acoustic Laboratories Computer Aided Management) database (10%). Subjects were asked about hereditary hearing loss, exposure to industrial and leisure noise, prescription drug use (particularly antibiotics, diuretics and cytotoxic drugs), head injury and any hearing-related symptoms they were currently experiencing. Sound-exposure histories were obtained by asking subjects to estimate their average number of hours per week and their years of exposure to any noisy activity, such as industrial noise, sporting activity, firearm use, the music industry, personal stereo use and other exposure to amplified music.

Participants' data were included in the analysis only if an acceptable pair of recordings were obtained for both left and right ears in the same recording session. Participants' records were excluded if the recording stability values (see Box 1) were less than 80%, if participants failed otoscopic inspection, or if they had a clear inherited factor or any form of aural disease (eg, otitis media, otosclerosis, fluctuant hearing loss, Menieres syndrome, or exposure to ototoxic substances). No form of noise or music exposure was considered grounds for exclusion.

For the 4.3% of participants who provided repeat records

over the nine years of recording, we included only the pair of records with the highest emission strength. Participants were aged 10 years to less than 60 years on the day of recording.

On the basis of the sound exposure histories recorded in the database, participants who reported personal stereo (PS) use were divided into three categories: PS = 0 (negligible, < 1 hour per week), PS = 1 (moderate, 1 hour to < 6 hours per week), and PS = 2 (heavy, >6 hours per week). Similarly, subjects were classified into two industrial noise (IND) categories according to whether they had ever worked in noisy industry: IND = 0 ("no"), and IND = 1 ("yes"). These classifications did not exclude other noise exposure factors.
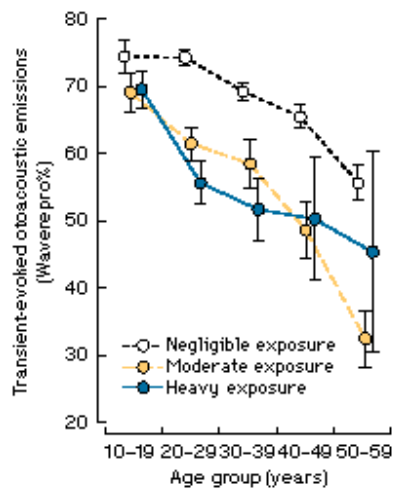
Statistical analysis

We applied analysis of variance with multiple linear regression in which the independent variables were sex, age (grouped by decade: 10-19, 20-29, 30-39, 40-49 and

50-59 years), PS use category, and industrial noise category. The dependent variable was the mean of values for both ears of otoacoustic emission strength (TEOAE Waverepro%; see Box 1). The multiple linear regression was performed using Statistica software.[22]

Results

Records from 1724 people (1066 males and 658 females) were included in the analysis.

Box 2 shows the relationship between age and otoacoustic emission strength for all subjects who reported being negligible, moderate or heavy users of PS systems (regardless of other noise exposure) and the corresponding sample sizes for these three groups. Considering each age range in turn, for the teenage range (10-19 years) there was no significant difference between any of the three PS use groups. For people aged 20-29 years and 30-39 years the PS = 0 (negligible exposure) group was significantly different from both the PS = 1 and the PS = 2 groups ($P <$ 0.01). For people in both the 40-49-years and 50-59-years ranges the PS = 0 group was significantly different from the PS = 1 group and also from the PS = 1 and PS = 2 groups combined ($P <$ 0.01). For all the adult age ranges, PS users had significantly lower values of the Waverepro% than non-users, with the lowest values in heavy users.

Box 3 compares, firstly, females and males with no industrial noise exposure and negligible PS use (the top two curves with open symbols) and shows that, for the three oldest age groups, the values of emission strength for males were significantly lower than those for females ($P <$

0.01). Secondly, the bottom three curves with filled symbols show the effect of PS use, industrial noise exposure and both forms of exposure for males only (we did not include females in this comparison as very few women [33] had had industrial noise exposure compared with men [286]). Thus, the four lowest curves in Box 3 compare four mutually exclusive noise exposure groups for males only. The corresponding sample sizes for males in these exposure categories are also shown in Box 3. Considering each age range in turn, there was no significant difference between four noise exposure groups at 10-19 years of age. However, for participants aged 20-29 years, all noise exposure groups were significantly different from each other ($P < 0.01$), with the exception of groups IND = 1 (industrial noise exposure only) and PS = 0, IND = 0 (negligible PS use and no industrial noise exposure). In the 30-39-years group the only significant difference was between the PS = 0, IND = 0 group and the group with both PS and industrial noise exposure ($P < 0.01$). For both the 40-49-years and the 50-59-years groups, the PS = 0, IND = 0 group was significantly different from the group with PS use only, and also the group with both PS and industrial noise exposure ($P < 0.01$). The mean values for the industrial noise exposure only group were the same as for the PS exposure only group but the sample size was small.

Box 4 shows the results of the multiple linear regression. For each of the selection conditions tested, the slopes (the "Effect" column) representing the rates of decline of Waverepro% are highly significant. This Table indicates that Waverepro% for males was about 5% lower than for

females. Waverepro% for moderate PS users was about 11% lower than for non-users, while for heavy PS users this value was 1.6% lower still; Waverepro% for respondents indicating industrial noise exposure was also 8% lower than for non-exposed people. The age dependence was a decline in Waverepro% of 0.49% per year, or 4.9% per decade.

Discussion

Our findings suggest that there is a strong trend for the strength of otoacoustic emissions to decline with protracted use of PS headsets, and that the size of this decline is proportional to the amount of exposure. Although the separation of PS users into moderate and heavy categories was based on self-report, the multiple regression showed an effect between the moderate and heavy users. However, the design of our study did not exclude the effects of other forms of noise to which people who tend to use PS systems may also be exposed. If PS exposure is associated with other lifestyle factors, our analysis would not have differentiated between them. Other factors, such as leisure and other non-occupational noise exposure (eg, power tools, car racing, concerts) and forms of injury to which males are more exposed (eg, head injury, barotrauma), may have accounted for the significant differences between the sexes in people with neither industrial nor PS exposure. We believe all such factors contribute to the high level of variance of the otoacoustic emission levels.[23]

Further, for males exposed to both PS use and industrial noise, the otoacoustic emission strength was significantly lower ($P < 0.001$) in all age ranges other than the 10-19-years and 40-49-years range. Remarkably, for the 30-39-years range there was no significant difference between the group exposed to industrial noise and the PS user group, both of which were significantly different from the non-exposed groups ($P < 0.001$). Yet the young adult PS users (20-29 years) had otoacoustic emission strengths significantly lower than non-users ($P < 0.001$), suggesting

that the decline occurs in the late-teenage and early-adult period -- a decade earlier than the expected industrial effect.

The multiple regression analysis showed that, in our sample, the apparent rate of decline in otoacoustic emission strength among young adults was greater for PS users who were also exposed to industrial noise. However, it is worth noting that this group included a subgroup of 26 deep coal miners whose mean values were a whole standard deviation lower than males with no industrial noise exposure.

Based on existing guidelines for occupational noise level limits in all Australian States and Territories of an eight-hour equivalent continuous A-weighted sound pressure level of 85 dB, it is surprising that PS use for less than six hours weekly at typical sound levels of 95 dB results in such a high level of damage accumulation. This leads to speculation that there may be other factors involved with sound delivered through earphones compared with free-field sound, such as more efficient delivery of high-frequency sound coupled with the high dynamic range of modern PS units. The popularity in recent years of units offering "additional bass boost" is consistent with the notion that users may be endeavouring to enhance the sense of sound envelopment which occurs at higher levels. Our findings strongly support the previous assertions by Waugh and Murray[24] of increased risk of ear damage from PS use, particularly if personal stereos are used in other environments in which users tend to raise the listening level

to mask out background noise (such as on public transport or while engaging in aerobic exercise), which may lead to generalised inner-ear problems.[25]

A 1996 study by Meyer-Bisch appears to be the only one to have succeeded in showing a significant difference between actual hearing levels of PS users and those of a control group.[26] Our findings illustrate why most previous studies of PS exposure have failed to observe any effect -- the preclinical phase of hearing loss[23] is extended, and PS units have not been around for long enough for critical levels of damage to be apparent in most users. Indeed, as only 39 PS users in our sample reported any hearing difficulties, the primary significance of our study is that transient-evoked (or click-evoked) otoacoustic emission measurement offers early warning for hearing loss.

Our findings on the effects of PS use and industrial noise exposure, both separately and together, illustrate an additive effect long held to be a basic property of noise-induced hearing loss.[23] In our study this effect may be partly the result of a small association between PS use and industrial exposure -- PS use was higher in the group who had industrial exposure.

The technique of measuring evoked otoacoustic emissions has direct application beyond the screening of neonates to programs for hearing loss prevention. It sheds light on the nature of presbycusis, or the normal hearing loss during ageing. Thus, by comparing the rates of decline shown in Box 4 with the relationship between Waverepro% and

audiometric hearing level illustrated in Box 1, it is possible to linearly estimate the remaining period of normal hearing. Beginning with Waverepro% values above 80%, as in normal neonates, and assuming a linear model, a decline in Waverepro% of up to 5% per decade should result in no hearing problems for life, while a decline of 7% per decade results in normal presbycusis (about seven decades of normal hearing). However, a decline of 20% per decade would give only 2.5 decades of normal hearing. The fact that many young people in our sample appear to have been subjected to such accelerated hearing loss suggests that it is not unreasonable to predict a rise in the number of young adults with premature hearing impairment.[27]

In summary, our findings highlight three important points related to hearing health:

- otoacoustic emissions may offer new precision in determining an individual's risk of hearing loss;
- the use of PS headsets, even in typical moderate use, is associated with rapid ageing of the cochlea comparable with industrial noise trauma;
- as personal stereos are here to stay, the essential message for preventing premature hearing loss in users is that listening times and volumes should be moderate, and that users should be aware of the potentiating effect of noisy background conditions which both add directly to the noise dose and encourage them raise the PS volume.

References

1. Carter NL, Waugh RL, Keen K, et al. Amplified music

10

and young people's hearing. *Med J Aust* 1982; 2: 125-128.

2. Carter NL, Murray NM, Khan A, Waugh RL. A longitudinal study of recreational noise and young people's hearing. *Aust J Audiol* 1984; 6: 45-53.

3. Carter NL, Murray NM, Bulteau VG. Amplified music, recreational noise and hearing in people aged 16-21 and 28-33 years. *Aust J Audiol* 1985; 7: 79-83.

4. Rintelmann WF, Bienvenue GR. Rock music and noise induced hearing loss: a review of research. *J Audio Eng Soc* 1977; 25: 134-145.

5. Cabot RC, Genter CR, Lucke T. Sound levels and spectra of rock music. *J Audio Eng Soc* 1979; 27: 267.

6. Wood WSI, Lipscomb DM. Maximum available sound-pressure levels from stereo components. *J Acoust Soc Am* 1972; 52: 484-487.

7. Kuras JE, Findlay, RC. Listening patterns of self-identified rock music listeners to rock music presented via earphones. *J Aud Res* 1974; 14: 51-56.

8. Clark WW. Noise exposure from leisure activities: a review. *J Acoust Soc Am* 1991; 90: 175-181.

9. Kirkwood DH. Washington starts waking up to hazards of recreational noise. *Hearing J* 1992; 45: 13-4, 16, 18, 20-23.

10. Bienvenue GR. Personal stereo systems: can they be used safely? New York: The New York State Speech/Language Hearing Association, 1984.

11. Catalano PJ, Levin SM. Noise-induced hearing loss and portable radios with headphones. *Int J Ped Otorhinolaryngol* 1985; 9: 59-67.

12. Clark WW. Amplified music from stereo headsets and its effect on hearing. *Hear Instrum* 1990; 41: 29-30.

13. Fearn RW, Hanson DR. Hearing level of young subjects exposed to amplified music. *J Sound Vibr* 1989; 3: 509-512.

14. Rice CG, Breslin M, Roper RG. Sound levels from personal cassette players. *Br J Audiol* 1987; 21: 273-278.

15. Royster JD, Royster LH. Amplified music and its effect on hearing. *Hear Instrum* 1990; 41: 28-29.

16. Turunen-Rise I, Flottorp G, Tvete O. A study of the possibility of acquiring noise-induced hearing loss by the use of personal cassette players (Walkman). *Scand Audiol Suppl* 1991; 34: 133-144.

17. Kemp DT. Stimulated acoustic emissions from within the human auditory system. *J Acoust Soc Am* 1978; 64: 1386-1391.

18. LePage EL, Murray NM, Tran K, Harrap MJ. The ear as

an acoustical generator: otoacoustic emissions and their diagnostic potential. *Acoustics Aust* 1993; 21: 86-90.

19. Murray NM, LePage EL. Age dependence of otoacoustic emissions and apparent rates of ageing of the inner ear in an Australian population. *Aust J Audiol* 1993; 15: 59-70.

20. LePage EL, Murray NM. Click-evoked otoacoustic emissions: comparing emission strengths with pure tone audiometric thresholds. *Aust J Audiol* 1993; 15: 9-22.

21. Davis AC. The prevalence of hearing impairment an reported hearing disability among adults in Great Britain. *Int J Epidemiology* 1989; 18: 911-917.

22. Statistica for Windows [computer program]. Version 5.1.2. Tulsa, Okla.: Stat Soft Inc., 1997.

23. LePage EL. Occupational Noise-Induced Hearing Loss: Its Origin, Characterisation and Prevention. *Acoust Aust* 1998; 26: 57-61.

24. Waugh D, Murray NM. The auditory hazards of using muff-like personal radio headsets in high ambient noise levels. *Aust J Audiol* 1989; 11: 107-114.

25. Weintraub MI. Vestibulopathy induced by high impact aerobics. A new syndrome discussion of 30 cases. *J Sports Med Phys Fitness* 1994; 34: 56-63.

26. Meyer-Bisch C. Epidemiological evaluation of hearing damage related to strongly amplified music (personal cassette players, discotheques, rock concerts): a high-definition audiometric survey on 1364 subjects. *Audiology* 1996; 35: 121-142.

27. LePage EL. A model forecasting the prevalence in hearing loss in the Australian population over the next 20 years based on trends in decline in otoacoustic emission strength. Proceedings of the Better Hearing Australia Conference, Adelaide, 7-11 August, 1994. Adelaide: Better Hearing Australia, 1994.

Authors' details

Hearing Loss Prevention Research, National Acoustic Laboratories, Sydney, NSW.
Eric L LePage, PhD, Senior Research Scientist;
Narelle M Murray, MA(Aud), Senior Audiologist.

Reprints will not be available from the authors.

Correspondence: Dr E L LePage, Hearing Loss Prevention Research, National Acoustic Laboratories, 126 Greville Street, Chatswood, NSW 2067.

Email: Eric.LePageATnal.gov.au