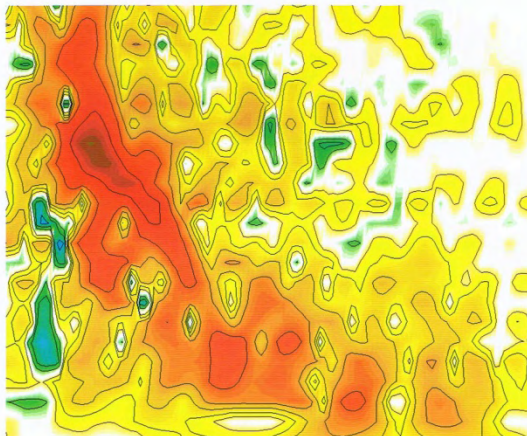
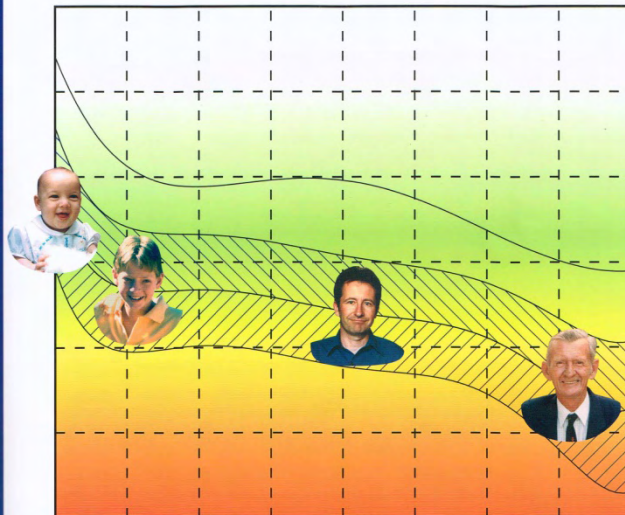




**Research & Development
Annual Report 1998/99**



**National Acoustic Laboratories
Annual Report 2001/2002**



**EXTRACTS FROM NAL ANNUAL REPORTS
CONCERNING HEARING LOSS PREVENTION
1992-2002**

Year	Page	
1992	4	Normative click-evoked otoacoustic emission data and their relationship to pure tone audiometry E. LePage, Narelle Murray, K. Tran
	5	Measures of susceptibility to hearing loss: otoacoustic emissions and noise exposure E. LePage, Narelle Murray
	8	Cochlear Motor Cells E. LePage, H.P. Zenner, G. Reuter, (University of Tübingen)
	9	Screening Criteria for Newborns E. LePage, N. Murray, R. Lovegrove, D. Starte (RNS hospital)
	9	Advanced Signal Processing E. LePage, M.J. Harrap (ADFA), K. Tran
1993	12	Normative click-evoked otoacoustic emission data, their relationship to pure tone audiometry and their age dependence Eric LePage, Narelle Murray, Khanh Tran
	14	Measures of susceptibility to hearing loss: otoacoustic emissions and noise exposure Eric LePage, Narelle Murray with Ken Mikl (WorkCover Authority, NSW)
	16	Cochlear Motor Cells E. LePage, H.P. Zenner, G. Reuter, (University of Tübingen)
	17	Selective Attention E. LePage, P. Michie, N. Solowij (school behavioural sciences, Macquarie University)

1994	17	Screening Criteria for Newborns	Eric LePage, Narelle Murray, R. Lovegrove, D. Starte (RNS hospital)
	19	Predictions of a rise in hearing loss in young people	Eric LePage
	21	Tracking the growth of cochlear damage in a cohort study	Narelle Murray, Eric LePage
	23	Defining "dynamic" susceptibility to hearing loss in relation to the problem of impact noise	Eric LePage, K. Tran, M. Harrap (ADFA)
1995	26	Ear damage in orchestral musicians	Eric LePage, Narelle Murray, Ken Mikl
	27	Evaluation of the Aural Reflex Earmuff	NL Carter, Dr. Han Tin French (DSTO)
	28	A new approach to hearing loss prevention	Eric LePage, Narelle Murray
	34	Longitudinal study of permanent shifts in otoacoustic emissions	Narelle Murray, Eric LePage
1996	34	Longitudinal study of hearing of orchestral musicians	Narelle Murray, Eric LePage, Ken Mikl
	36	Role of efferent control loops: Selective attention in hearing. An otoacoustic analog and its potential role in hearing loss prevention	Patricia Mitchie, Eric LePage
	37	Temporary shifts in otoacoustic emissions	Eric LePage, Narelle Murray, Khanh Tran, Michael Harrap
	39	Prevention of hearing loss in children	John Macrae
1997	39	The role of audiometry in occupational noise management	John Macrae
	41	Editorial	
	42	Hearing conservation - from before the beginning	Narelle Murray and Eric LePage
	45	Survey: Awareness of hearing issues among the Australian population	Eric LePage, Sheralyn Shacknoffsky, Narelle Murray
1998	48	Accelerated ear damage in listeners to headset exposure	
	51	Noise and Hearing Loss Prevention Research - editorial	
	52	Workers compensation for industrial deafness	John Macrae
	54	An evaluation of the Australia National Standard for Occupational Noise	John Macrae
1999	57	Longitudinal study of inner ear damage in an orchestra	Narelle Murray, Eric LePage, Ken Mikl
	59	Watching the ear age - an examination of otoacoustic emission cohort data	Eric LePage, Narelle Murray
	63	Face page Time Frequency response	
	64	Editorial hearing loss prevention and noise research	
2000	65	Longitudinal study of a non-noise exposed cohort comparing click-evoked otoacoustic emission techniques and pure tone audiometry	Narelle Murray and Eric LePage
	68	Hearing Status of aboriginal prisoners	Eric LePage, Narelle Murray and Tony Butler
	69	Modelling transient otoacoustic emissions in noise-induced hearing loss	Eric LePage and Ake Olofsson
	74	NAL-OAE1: NAL transient evoked otoacoustic emission analysis software	Eric LePage, Dan Zhou, Narelle Murray John Seymour
2000	76	To fit or not to fit: preliminary otoacoustic emission assessment of children with severe mental and physical disabilities for considering of hearing aid fitting	Narelle Murray, Eric LePage, Greg Birtles, Donna Smith, Annette Smith, Lindsay Hamilton
	78	Transforming hearing conservation into hearing loss prevention: testing measures of early warning for cochlear hearing loss	Eric LePage, Narelle Murray, John Seymour and Dan Zhou

2001	81	The scope of non-noise factors influencing research aimed at the effects of noise: one example - blood cholesterol level	Eric LePage, Narelle Murray, Tony Butler
	84	Nine years' progress report of study of inner ear damage in an orchestra	Narelle Murray and Eric LePage
	88	Effective training methods for workplace noise reduction	Suzanne Purdy and Warwick Williams
	89	Face page Aging curve Australian population	
	90	New NAL direction funding from hearing aid company	
	91	NAL-OAE analysis software - philosophy and a potted history	Eric LePage
	93	Effect of static ear pressure on click-evoked otoacoustic emissions	Johannes Lantz, Eric LePage
	95	Cochlear model for emissions	Eric LePage, Ake Olofsson
2002	98	Causes and prevention of hearing loss: global trends in industrial and leisure noise, interactions, definitions and strategies - a one day colloquium	Henrik Dahl, Donald Henderson, Ramesh Rajan, Eric LePage, Ross Dineen and Narelle Murray
	102	New Research Director's overview - returning prevention to behavioural research	Harvey Dillon
	105	An examination of the apparent poor performance of some hearing protectors	Warwick Williams
	109	Increasing the effectiveness of OHS education for noise-exposed individuals	W. Williams, S. Purdy, N. Murray, E. LePage, K. Challinor
	113	Evaluation of the effects of high noise exposure: coal miners	Eric LePage, Narelle Murray, Adrian O'Malley
	116	Personal Stereo Noise Exposure	W. Williams

HEARING CONSERVATION

This research program is directed at developing a better understanding of the causes of hearing loss through biological research plus objective measurements designed to characterise the state and rates of aging of human ears. An important object is to identify individuals who possess prematurely aged ears and forewarn them that they are at greater risk for acquiring a hearing loss or tinnitus (ringing in the ears) so they can modify their lifestyles and risk factors accordingly. It is seen as being complementary to the need for blanket reduction of noise levels in industry since those workers who are most at risk for noise-induced hearing loss (NIHL) may be the ones most likely to succumb due to excessive leisure music/noise exposure (which cannot be bound by legislation), or due to the ototoxic effects of many common drugs.

The studies listed below carried out by physiologist Dr. Eric LePage, and audiologist Narelle Murray concern cochlear mechanics and its aging, and applying the information gained to prevention services and screening programs to be offered in AHS centres. The basic research into cellular mechanisms is being carried out in collaboration with Professor H.P. Zenner and his group at the University of Tübingen, Germany; behavioural research into the role of selection in listening is in collaboration with Dr. P.T. Michie and her group at Macquarie University. The damage assessment approach uses the recent technique termed Evoked Otoacoustic Emissions (EOAE), or sounds emitted by the ear. The characterisation of these sounds forms the basis of several projects with the WorkCover authority and the Coal Industry; neonatal screening with Dr. D. Starte at the Royal North Shore hospital, and advanced data collection and processing methods in collaboration with Dr. M.J. Harrap of the Australian Defence Force Academy in Canberra.

Normative click-evoked otoacoustic emission data and their relationship to pure tone audiometry

Investigators: E. LePage, Narelle Murray, K. Tran

Background: The most significant byproduct of basic research into the mechanisms of hearing and hearing loss in the past fifteen years is otoacoustic emissions, sounds which are emitted from the ear. The characteristics of these emissions can provide a large amount of information about the state of damage to the inner ear. High emission strengths indicate little damage to the outer hair cells which control hearing sensitivity, low emission strengths indicate a great deal of damage.

Research Questions: There is no theoretical reason why hearing levels should indicate the extent of cochlear damage, but until now it has been the widespread assumption that no hearing loss means no permanent damage. A key object of the normative study is to compare pure tone audiometric thresholds with measures of emission strength for the same ears and to compare how each depends on aging and noise-exposure. Secondly, susceptibility to hearing loss varies greatly amongst people with similar audiometric pictures for reasons yet to be revealed.

Research Procedures: The project applies an Otoacoustic Emission (OAE) analyser to the Australian population, investigating OAE characteristics. Data were collected from individuals in all age ranges in screening mode after checking for external or middle ear complications. Pure tone audiograms and aural histories were also sought from as many subjects as possible within this group.

Findings: Data were analysed and a preliminary definition of Coherent Emission Strength (CES) derived. It has been found that emission strength values may decrease over most of the normal range

before there is any shift in audiometric thresholds (Fig. 1). Emission strength decreases with age in a way which correlates well with the onset of hearing loss as determined in the recent British epidemiological study (Davis, 1989). Evidently CES is a good candidate for measure of individual susceptibility to hearing loss.

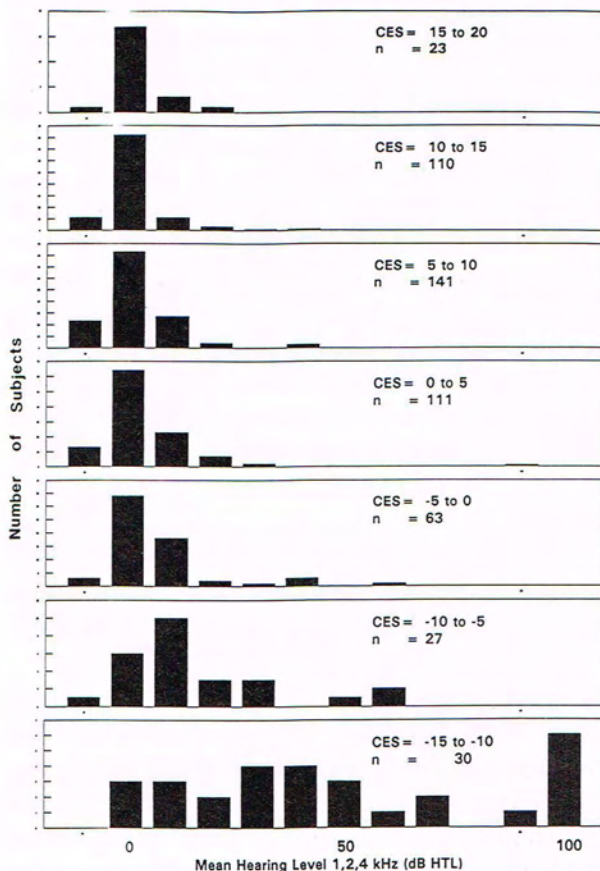


Fig. 1. Interrelationship of hearing level and Coherent Emission Strength (CES dB SPL). CES decreases by 75 percent of its operating range before there is a significant deviation in hearing levels from normal.

Significance: All measures to prevent hearing loss are currently levelled at the population as a whole. To be able to determine susceptibility in individuals is to be able to provide early warning of increased risk for hearing loss and the individual will thus be able to take precautionary measures.

References:

Davis, A.C. 1989. The prevalence of hearing impairment and reported hearing disability among adults in Great Britain. *Int. J. of Epidemiology*, 18, 911-917.

LePage, E.L., Murray, N.M. 1993. Click-evoked otoacoustic emissions: comparing emission strengths with pure tone audiometric thresholds. *Aust. J. of Audiol.*, 15, 9-22.

Murray, N.M and LePage, E.L. 1993. Age dependence of otoacoustic emissions and apparent rates of aging of the inner ear in an Australian population. *Aust. J. Audiol.* (in press).

Measures of susceptibility to hearing loss: otoacoustic emissions and noise exposure.

Investigators: E. LePage, Narelle Murray

Two projects have been undertaken applying otoacoustic emission measurement techniques with a view to quantifying the extent of cochlear damage accumulated (1) in a high noise level environment by a pilot study of 24 deep coal mine workers, and (2) in a relatively low level of noise exposure through a study of orchestral musicians.

(1) Otoacoustic emission assessment of ear damage in coal mine workers

This investigation was commissioned by the Australian Coal Industry Research Laboratories Limited, with support provided from the National Energy Research, Development and Demonstration Program.

Background: A pilot study was invited to investigate if the apparent predictive ability of the otoacoustic emission test has practical value to individuals exposed to high levels of noise for long periods.

Research Questions: 1. To compare emission strengths of mine workers and those of an Australian population obtained over the past three years. 2. To test whether the otoacoustic emission test is a useful measure of noise dose for mine workers. 3. To test whether the definition of emission strength for permanent damage is adequate for temporary disturbance caused by noise exposure.

Research Procedures: Otoacoustic emission screening tests were carried out on 24 mine workers before and after shifts over a period of ten days, with

follow up carried out three months later. Their pure tone audiograms were obtained from their employer and estimations made of their noise dose and wearing of hearing protectors by an accompanying engineer.

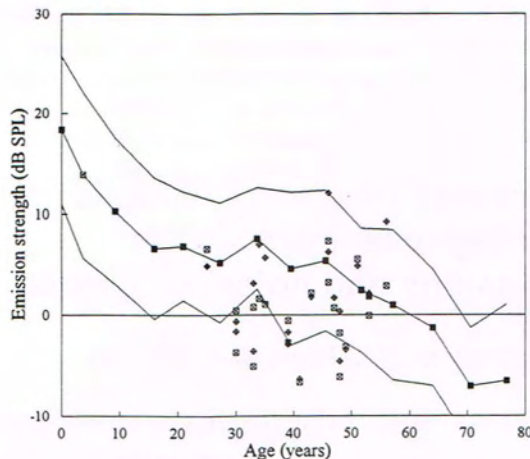


Fig.1. Mean emission strength of miners compared with mean (\pm 1 S.D.) emission strength of a normal Australian population.

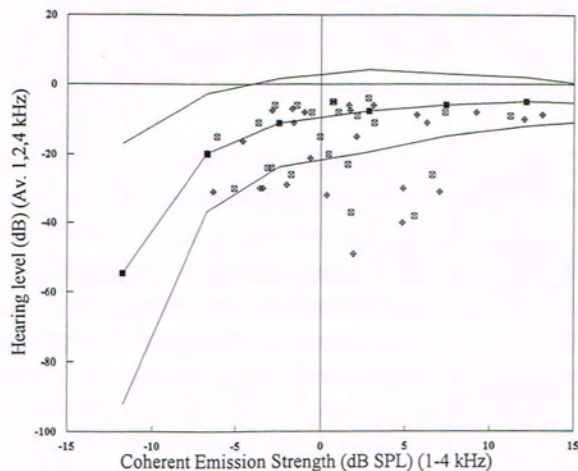


Fig.2. Relationship of mean emission strength of miners and their pure tone audiometric hearing level (mean 1, 2, 4 kHz) compared with mean (\pm S.D.) relationship between emission strength and pure tone audiometry of the normal population.

Findings: The pilot study revealed that it is possible to obtain a sensitive, single-parameter measure (*Coherent Emission Strength*, (CES)) of the extent of cochlear damage and that, on average, this is significantly higher in miners than in the Australian population of the same age range (Fig. 1).

A comparison of the mean emission strength values with the mean of pure tone audiogram values for 1, 2 and 4 kHz was set against the mean plus and minus one standard deviation for normal Australian

population (Fig. 2). Those individuals with lower emission strengths and no obvious hearing loss may be those at higher risk for acquiring a hearing disability, while those with emission strength below zero are more likely to be already displaying signs of a mild hearing loss. Those with emission strengths approaching -10 dB may have a hearing impairment in the moderate to severe range. Some 10 ears showed a reasonably high emission strength (0 dB SPL) and a pure tone loss (20 dB HL average for 1,2,4 kHz). Pure tone audiometry in these cases should be reassessed.

Significance: Coherent Emission Strength appears to be a useful parameter for assessing permanent damage and over a period of time could be useful for assessing the effectiveness of wearing hearing protection devices. A potentially very important application of transiently-evoked otoacoustic emissions for the Coal Industry is as an objective cross-check on the validity of pure tone audiometry.

References:

LePage, E.L., Murray, N.M. and Macrae, J.H. 1993. Otoacoustic emission assessment of ear damage in coal mine workers: Pilot study May-October 1992. National Acoustic Laboratories Commissioned Report No. 75.

(2) Otoacoustic emission assessment of ear damage in orchestral musicians

Investigators: E. LePage, Narelle Murray, with K. Mikl (WorkCover Authority, NSW)

Background: The WorkCover Authority was requested by the Sydney Opera House Trust to make an assessment of occupational noise problems and to identify acoustic problems with the use of the orchestra pit in the Opera Hall of the Sydney Opera House. The Hearing Conservation Research Unit of NAL was invited to carry out otoacoustic emission testing on members of the Australian Opera and Ballet Orchestra as part of this investigation.

Research Questions: 1. How do emission strength measures and pure tone audiometry of a relatively low noise-exposed population compare with the same measures of a high noise-exposed (mine workers) and a non-noise-exposed (office workers) population and a normative Australian population? 2. Is the derived parameter of Coherent Emission

Research Procedures: Otoacoustic emission screening and pure tone audiometry were carried out on 60 orchestra members who rehearse or perform daily over a season extending for the greater part of the year.

Findings: From the first assessment of ear damage it is notable that younger members of the orchestra have emission strengths which show less damage than the overall population, with values significantly above the means of the population (up to one standard deviation). However, they also appear to age at a faster rate. Older members of the orchestra tend to have emission strengths which lie below the means of the population (Fig. 1).

From a comparison of emission strengths with pure tone audiometric hearing levels (mean 1,2,4 kHz) it was noted that there were 25 ears with negative emission strengths and pure tone audiometric thresholds within normal limits. It is suggested that these individuals may be more susceptible to a hearing loss (Fig. 2). Eight ears were found to have positive emission strength values and pure tone audiometric thresholds 20 dB. Pure tone audiometry was reassessed in these cases, with the majority having improved audiometric thresholds.

Significance: Since this initial study was carried out changes have been made both to the orchestra pit and to seating arrangements within the orchestra. Repeat measurements will be taken in 1993 to see if these alterations have helped maintain the stability of hearing of the orchestra members.

Both the study undertaken with the orchestra and that undertaken with the coal miners, together with an earlier study of non-noise exposed workers have highlighted the value of otoacoustic emission screening for hearing conservation purposes. It has proved to be an easily applied, rapid and, most importantly an objective method of screening for cochlea damage.

References:

WorkCover Authority, Acoustic Services. 1992. Noise Hazard Assessment - Australian Opera and Ballet Orchestra.

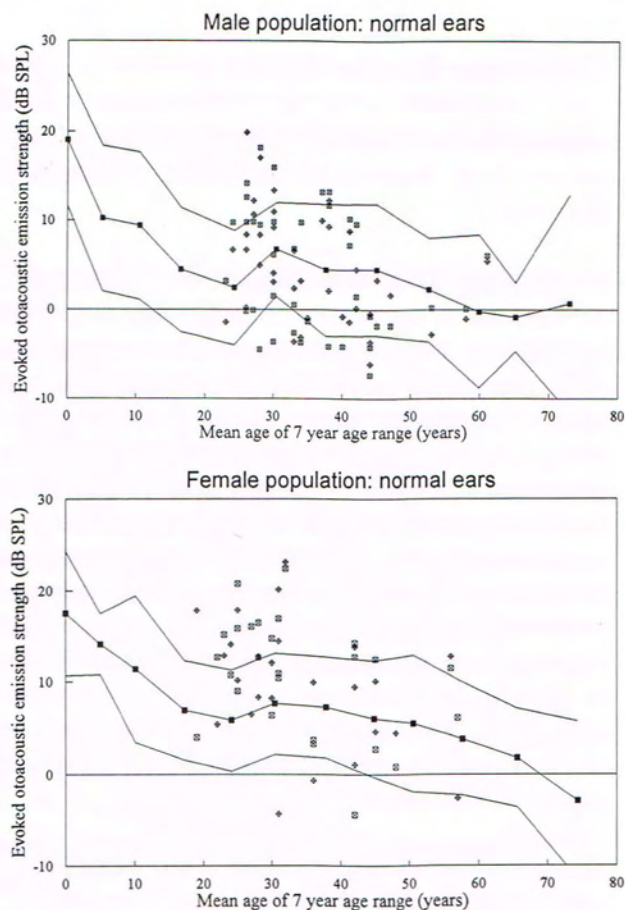


Fig.1. Mean emission strength of male (top panel) and female (lower panel) orchestra members compared with (± 1 S.D.) emission strength of a normal Australian population (males and females separately).

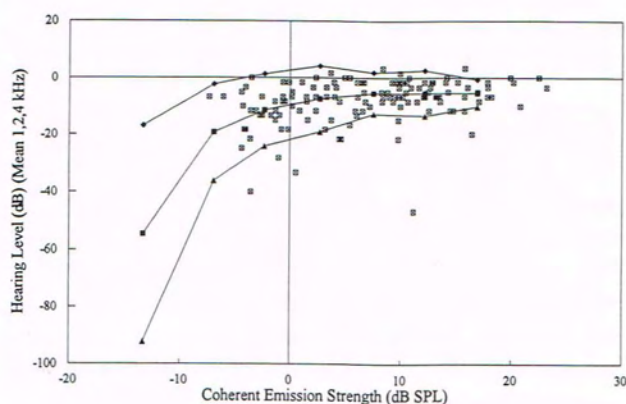


Fig.2. Relationship of mean emission strength of male and female orchestra members and their pure tone audiometric hearing level (mean 1,2,4 kHz) compared with mean (± 1 S.D.) relationship between emission strength and pure tone audiometry of the normal population.

Cochlear Motor Cells

Investigators: E. LePage with Professor H.P. Zenner and Dr G. Reuter of the University of Tübingen, Germany.

Background: Loss or damage of outer hair cells of the cochlea is the primary cause of sensorineural hearing loss. The manifestation of their motor activity to provide for normal hearing sensitivity is the topic of intense research activity. Models of cochlear function, used extensively throughout hearing science, e.g. in speech processing, are based upon the Nobel prize-winning work of Georg von Békésy which predates the discovery of the motor action of these cells. This collaboration has brought together two investigators who have carried out experimental work on the extracellular (LePage, 1987) and cellular (Zenner, 1986) mechanical measurements of hair cell activity. The outcome is yielding a new class of cochlear models which stands to have a profound impact on the field of audiology.

Research Questions: 1. What is the mode of action of the outer hair cells in living cochlear tissue? 2. What is the basis of the so-called cochlear amplifier?

Research Procedures: These questions are being approached by classifying the various motor characteristics by speed and mode of action in living cochlear tissue. All experiments have been carried out at, and funded by the University of Tübingen, subject to the stringent requirements of animal research.

Findings: The outer hair cells display a mode of activity beyond that required of current models of the cochlear amplifier. The changes of length of the outer hair cells (LePage, 1989; Zenner 1993) constitute a major mode of action responsible for how the ear adjusts to changes in stimulus level over a 120 dB dynamic range, accounting for the loss of dynamic range in hearing loss and the effects of loud sound (LePage, Reuter and Zenner, 1993). A significant outcome is a first viable model for tinnitus (LePage, 1993) - unwanted sounds which are generated in the cochlea due to damage.

Significance: A major update to the cochlear amplifier theory with biproducts for explaining the ori-

gins of sensorineural hearing loss, variability of individuals in response to loud noise which will have application to prevention activities, and a model for tinnitus which will be immediately useful in explaining the condition to tinnitus sufferers.

References:

LePage, E. L. 1987. Frequency-dependent self-induced bias of the basilar membrane and its potential for controlling sensitivity and tuning in the mammalian cochlea. *J. Acoust. Soc. Am.*, 82, 139-154.

LePage, E.L. 1989. Functional role of the olivo-cochlear bundle: A motor unit control system in the mammalian cochlea. *Hear. Res.*, 38, 177-198.

LePage, E.L. 1993. A model for cochlear origin of subjective tinnitus: excitatory drift in operating point of inner hair cells, In, *Tinnitus Mechanisms*, ed. by J. Vernon and A. Møller, Allyn and Bacon, U.S.A. (in press)

LePage, E.L., Reuter, G., and Zenner, H.P. 1993. Summating baseline shifts and mechanical adaptation in a guinea pig cochlear explant shown with two displacement measuring techniques. In, *Biophysics of hair cell sensory systems*, edited by H. Duifhuis, J.W. Horst, P. van Dijk and S. van Netten. World Scientific Publishing, (in press).

Zenner, H.P. 1986. Motile responses in outer hair cells. *Hear. Res.* 22, 83-90.

Zenner, H.P. 1993. Possible roles of outer hair cell d.c. movements in the cochlea. *rit. J. Audiol.* 27, 73-77.

Selective Attention

Investigators: E. LePage, with P.T. Michie and N. Solowij of the School of Behavioural Science, Macquarie University.

Background: Pure tone audiometry is an exercise in selective attention. It is a measure of how well any person can sort out a pure tone from background noise. The first sign of hearing loss is that this selection process is diminished in conditions of speech in raised background noise. There are several reasons to think that the selection process normally involves activity of the outer hair cells which are connected to the brain and to the responses of

the opposite ear (LePage, 1989). Speech discrimination and spatial selection may therefore be fundamentally dependent on normal outer hair cell function which is why noise damage leads to difficulties in discrimination.

Research Questions: Otoacoustic emissions are good measures of the activity of outer hair cells. Therefore, are emissions strongly affected by voluntarily selecting one tone in a pair of tones so that the brain regards the non-attended tone as background noise?

Research Procedures: Normally hearing subjects are tested for otoacoustic emission strength to determine suitability for the experimental protocol. Subjects then listen to an exacting task where they must identify target sounds and respond with a button push to confirm identification of the various tone bursts while the stimulus conditions remain unaltered. The amplitudes of the resulting otoacoustic emissions are compared to determine whether the sounds are being attended to or not.

Findings: A small attentional effect has been seen by a number of investigators. However, thus far our experiments have not confirmed this result, despite considerable theoretical justification for thinking a significant effect exists.

Significance: The most common complaint of the hearing impaired is the interference to their hearing of background noise. Background noise constitutes the most significant technical problem to be overcome in new hearing aid designs. A positive result should provide more understanding of the background noise problem and reveal why other investigators have failed to demonstrate it and provide considerable support for the revised theory of outer hair cell action.

References:

LePage, E.L. 1989. Functional role of the olivo-cochlear bundle: A motor unit control system in the mammalian cochlea. *Hear. Res.*, 38, 177-198.

Giard, M-H., Bouchet, P., Collet, L., and Pernier, J. 1992. Auditory selective attention at the cochlear level. Presented at the 5th International Conference on Cognitive Neurosciences (ICON V) Jerusalem, Israel, June 14-19, 1992.

Puel, J-L., Rebillard, G., Bonfils, P. and Pujol, R. 1989. Effect of visual selective attention on otoacoustic emissions, in *Cochlear Mechanisms*, ed. by J.P. Wilson and D.T. Kemp, Plenum Publishing Corp.

Screening Criteria for Newborns

Investigators: E.LePage, N. Murray, R. Lovegrove, with Dr. D. Starte (Royal North Shore Hospital), AHS staff.

This project is looking at special cases of the Age study, i.e. neonates and infants, for which data is being collected from a variety of sources, with values for specificity and sensitivity being determined. Those who fail the criteria would be referred on for ABR testing or Electrocochleography.

Advanced Signal Processing

Investigators: E.LePage, M.J. Harrap (ADFA), K. Tran.

Otoacoustic emission methods provide much more information than pure tone audiometry. However, even more information is contained in otoacoustic emission records than is currently being used. At present the spectra of the click-evoked response is an average of all the activity of the record in time. By contrast, using the short-time Fourier transform it is possible to display dynamic variations in the spectra giving a time picture of how the ear responds to a click (LePage, et al., 1993). Since impulse noise, e.g. gunfire, is the most damaging form of noise and represents a major problem for Defence Forces, the project is aimed at determining susceptibility to hearing loss in terms of how well the cochlea can protect itself by dynamically adapting to the fast pressure wave front. There is a significant commercial potential for the project by virtue of updating current recording methods.

Reference:

LePage, E.L., Murray, N.M., Tran, K. and Harrap, M.J. 1993. The ear as an acoustical generator: otoacoustic emissions and their diagnostic potential. *Acoustics Australia*, December 1993.

EFFECTS OF NOISE ON PEOPLE

The research of of this group is concerned with the effects of noise on people. This includes the identification of hitherto unknown or only suspected effects of noise, as well as increasing knowledge of relatively well-known effects such as those on the ear and hearing. The work may include research into methods for utilising the beneficial effects of noise as well as the amelioration of its harmful effects.

By its nature much of the research is interdisciplinary. Dr. Carter has set up collaborative arrangements for research with the Royal North Shore Hospital, the University of Newcastle and the Defence Science and Technology Organisation. Dr. Carter is also chairman of an interdisciplinary committee developing research programs into the effects of noise from the Third Runway at Sydney Kingsford Smith Airport.

Traffic Noise and Sleep Disturbance

Funded by the Roads and Traffic Authority (RTA)

Investigator: Norman Carter

Background. Community reaction to night-time traffic noise, particularly that due to trucks, has been severe. The RTA and similar authorities require information as to the best metrics to use in the measurement of traffic noise.

Research Questions. Is sleep disturbance due to traffic noise predictable from its long term LAeq (equivalent A-weighted noise level)? Is prediction improved by including the number of particularly noisy events such as that due to trucks?

Procedures. Volunteer (paid) subjects are asked to sleep in a laboratory for five nights at intervals of one week. Their sleep is monitored continuously overnight. Noise exposures combine one of two levels of continuous traffic noise with different numbers of truck noises per night.

Findings. Study continuing.

Laboratory Study of Sleep. Noise Induced Arousal, Cardiac Arrhythmia and Urinary Catecholamines

Investigator: Norman Carter in collaboration with Royal North Shore Hospital and University of Newcastle.

Background. Sleep is characterised by less frequent cardiac arrhythmia and lower sympathetic nervous system activity than the waking state. Sympathetic nervous activity is probably related to cardiac arrhythmia and is associated with increased output of catecholamines. Chronically raised catecholamine levels could cause adverse health effects.

Research Questions. Does environmental (truck and aircraft noise) during sleep increase the frequency or severity of cardiac arrhythmia and overnight excretion of urinary catecholamines? Is this related to sleep stage at noise onset?

Procedures. Outpatients of the Royal North Shore Hospital with a history of cardiac arrhythmia slept in a laboratory at the hospital. Recorded truck and aircraft noise was played during the night, and subjects' sleep and cardiac arrhythmia were monitored. Overnight urine was collected and catecholamines assayed by staff of the renal laboratory at Royal North Shore hospital.

Findings. Noise events caused five times as many arousals as occurred spontaneously. Cardiac arrhythmia was related to sleep stage but not to noise events. There appeared to be no noise effect on urinary catecholamines. These findings are significant because of their basic questions of the effects of noise on health.

Development of Research Proposals for the Federal Airports Corporation into the Effects of Noise From the Third Runway on Health

Background. A committee headed by Dr. N. Carter was formed to assess the feasibility of research into the possible effects of the changes in aircraft noise exposure due to the Third Runway on health. The committee comprises Mr. P. Peplow of Noise Prevention Services at NAL, Associate Professor R. Taylor, Department of Public Health, University of Sydney, and Dr. S. Job from the Department of Psychology at Sydney University. A report of this committee setting out comprehensive proposals for research in this area was produced in October 1992. These proposals proved too costly for the FAC to support and a revised program was developed and presented to the FAC. This document has now gone to academic reviewers in Australia and overseas before submission to the FAC for funding.

Procedures. The study proposals incorporate advanced epidemiological and social survey designs, including a cohort study of blood pressure in children and a survey of mental health in adults.

Significance. This research has implications for airport noise control world-wide. Because of its concern with health it has already generated considerable interest overseas.

Trials of an Earmuff for Use by the Australian Artillery

Investigator: Norman Carter in collaboration with Defence Science & Technology Organisation

Background: It is well known that firing artillery weapons can cause loss of hearing. The current Department of Defence Manual of Occupational Health and Safety specifies that double hearing protection (earplugs and earmuffs) be worn by all personnel who may be within three metres of an artillery gun at any time during firing. However, double hearing protection can impair spoken communication, leading to a conflict between the requirements for hearing protection and operational efficiency. Some

empirical findings also suggest that double hearing protection may not be necessary.

Research Questions: Are presently available earmuffs alone (without simultaneous wearing of earplugs) adequate to protect hearing in artillerymen working within three metres of an artillery gun during firing ?

Procedures: The trials are carried out in collaboration with the Defence Science and Technology Organisation. Volunteer subjects (servicemen and some civilian employees of the Department of Defence) are exposed to simulated howitzer noise while wearing earmuffs. The levels of the 'gun' noise commence at relatively low levels and are gradually increased on successive days. The volunteers' hearing thresholds and evoked otoacoustic emissions are measured before and immediately after each noise exposure.

Findings: The studies are incomplete but results so far suggest that good quality heavy duty earmuffs may be sufficient to protect hearing, without simultaneous use of earplugs. 'Talk through' earmuffs could provide the person to person communications required by artillery personnel.



A volunteer subject wearing experimental earmuffs, being exposed to simulated howitzer noise.

HEARING LOSS PREVENTION

(also known as Hearing Conservation Research).

The Hearing Loss Prevention Research Unit's current program is directed at developing a better understanding of the causes of hearing loss through biological research plus objective measurements designed to characterise the state and rates of ageing of human ears. An important object is to research and develop new strategies for preventing hearing loss.

In the past, programs for limiting noise-induced hearing loss in industry, although extensive, have not proven to be highly effective and research has been mainly directed at ways of limiting noise at source or of reducing the amount of noise reaching the ears of workers by the use of hearing protection. Hearing protector research programs have been directed at defining attenuation characteristics of available protectors and determining which type of protector is most appropriate to a particular application.

The advent of preliminary predictors of hearing loss described below makes it possible to improve the success of hearing loss prevention programs. These studies, carried out by physiologist Dr. Eric LePage, audiologist Mrs. Narelle Murray and engineer Khanh Tran, concern cochlear mechanics and ageing of the cochlea, and apply the information gained to prevention services and screening programs.

Normative click-evoked otoacoustic emission data, their relationship to pure tone audiometry and their age dependence.

Investigators: Eric LePage, Narelle Murray, Khanh Tran

Background: The characteristics of otoacoustic emissions can provide much information about the state of damage to the inner ear. A primary interest in otoacoustic emissions worldwide has been to determine if the new technique could be used to obtain a faster, object-



Eric LePage

ive measure of audiometric threshold. This pursuit draws attention to the widespread tacit assumption that the most sensitive measure of auditory acuity, the pure tone audiogram, is also the most sensitive measure of ear damage. By contrast, a morphological study in guinea pigs by Altschuler et al., (1992) recently showed that

behavioural thresholds do not decline at any frequency until outer hair cell loss at the appropriate location is extensive, i.e. all three rows are largely depleted. If this finding could be demonstrated also for humans, it would have profound implications for the clinical practice of audiology and otology. This is because the ear's high sensitivity to sound does not necessarily imply high sensitivity to its own state of damage. Evidently the ear's mechanism contains a great deal of redundancy or excess performance. When the excess capacity is used up, further damage results in hearing loss.

Using a variety of measures, the effectiveness of industrial hearing conservation programs in the USA has been difficult, if not impossible to demonstrate, despite the high level of motivation to conserve hearing once real difficulties are experienced. A salient fact is that despite the efforts and expense of large numbers of professionals running such programs, a significant shift in hearing threshold (15 dB) must take place in order to identify a person is at risk (Royster, 1993). In terms of the morphological evidence referred to above, this means that an ear may need to progress from, say, 80 percent depletion to 90 percent depletion of outer hair cells before any warning bells ring that the noise exposure is excessive. Thus current hearing conservation programs based upon pure tone audiometry appear to possess a major flaw at the outset — they provide no early warning of accumulated inner ear damage until

the outer hair cell population is substantially reduced, maybe even decimated.

Research Questions: If it became routinely possible to screen for the number or functionality of those cells remaining in any ear, would the approach provide a sound basis for early warning detection and prevention? Success with this objective would theoretically allow hearing conservation programs to evolve by (a) determination of individual risk long before symptoms present, (b) carrying out screening much more quickly and cost-effectively than previously by targeting the 15 percent most at risk, and (c) serving as a much more sensitive indicator of the effectiveness of any hearing conservation program.

A key object of the normative study has therefore been to compare pure tone audiometric thresholds with measures of emission strength for the same ears and to compare how each depends on ageing and noise-exposure. Secondly, susceptibility to hearing loss is gauged by lack of emission strength and varies greatly amongst people with similar audiometric pictures for reasons as yet unknown.

Research Procedures: The project applies an Otoacoustic Emission (OAE) analyser to 1500 males and females in the Australian population, investigating click-evoked OAE characteristics. Data were collected from individuals in all age ranges in screening mode after checking for external or middle ear complications. Pure tone audiograms and aural histories were also sought from as many subjects as possible within this group. The pure tone audiometry is compared with the net otoacoustic emission strength over the same frequency range (1 to 4 kHz), while the emission strength values: mean, mean \pm 1 standard deviation, maximum and minimum in each age range is plotted versus age after first grouping the results into age ranges spanning 6 years, 0, 1 to 6, 7 to 12 and so on.

Findings: Following data analysis a preliminary definition of Coherent Emission Strength (CES) was derived. It has been found that emission strength values may decrease over most of the normal range before there is any shift in audiometric thresholds (LePage and Murray, 1993; Murray, LePage and Tran, 1994). In a recent study hearing loss in the British community is again shown to be a phenomenon restricted to the elderly (Davis, 1989). Figure 1 shows (LePage et al, 1994) that the distribution of critically-low values of emission

strength in the Australian community overlies the British distribution well. While other indices of net outer hair cell function are being tried, CES is a good candidate for measure of individual susceptibility to hearing loss. All measures to prevent hearing loss are currently levelled at the population as a whole and are almost exclusively directed at occupationally noise-exposed workers and noise-induced hearing loss. A crude measure of individual susceptibility is the CES value, and for any age range, 0, 1 to 6, 7 to 12 etc., the range of values of CES is very wide. That is, while there is a statistically

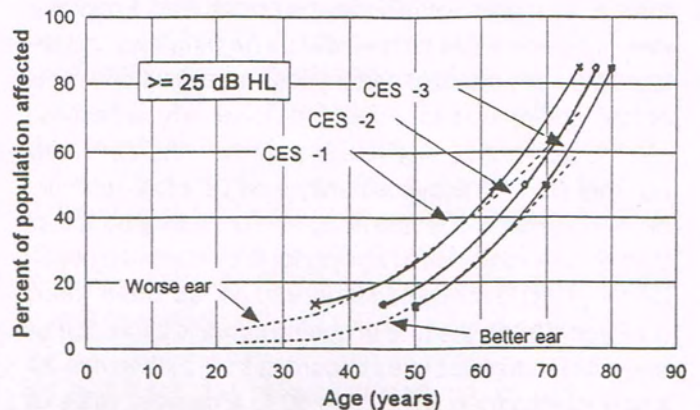


Figure 1 - Coincidence of objective estimation of distribution of critically low emission strengths (choice of CES -1, -2 and -3 dB SPL) with the distribution of hearing loss in the British study as determined for better ear and worse ear.

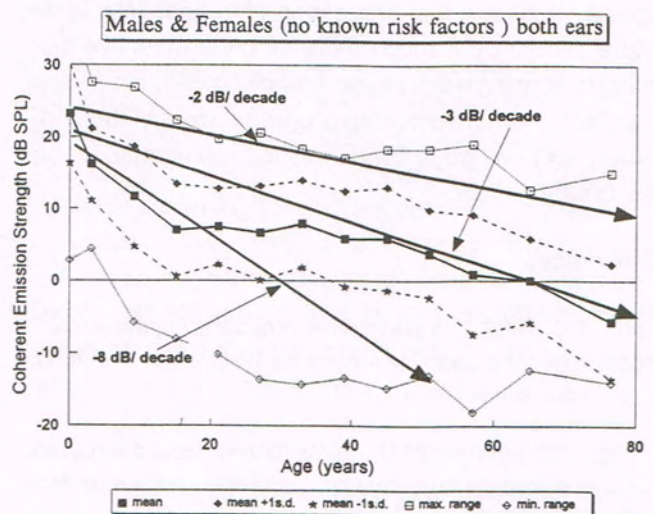


Figure 2. Rates of ageing in the Australian population

significant decline in the mean values with age, the scatter is wide. This suggests that there are highly susceptible individuals at every age in the population. It

follows that different individuals may require a different maximum sound level to avoid accelerated damage.

Significance: It is virtually axiomatic that noise-induced hearing loss is largely preventable, if not entirely avoidable. This is the fundamental basis upon which hearing conservation programs are structured. These results, however, have drawn into focus the fine dividing line between presbycusis and noise-induced hearing loss. The concept of damage accumulation in the cochlea prior to hearing loss being exhibited almost removes this distinction. Both are aspects of ageing of the cochlea; presbycusis may be considered a minimal rate of ageing while noise-induced hearing loss constitutes the early onset of hearing loss as a result of accelerated ageing due to excessive noise, where "excessive" is defined as any rate higher than the observed minimal rate. Presbycusis may now be more formally defined in terms of a rate of ageing occurring no faster than the average rate of ageing for the Australian population (3 dB/decade). However, as we have seen (LePage, 1994), the rate of ageing curve is a function of age with the highest rates occurring from birth to age 20 and declining from ages 20 to 30 to a minimal value of about 3 dB/decade. This new concept immediately highlights the historic state of indeterminacy of the causes of the accumulated ageing. Other factors such as antibiotics (e.g. the aminoglycosides) and environmental toxins (e.g. toluene) may lead to accelerated ageing. It follows that prevention of hearing loss in the future should be a much more tangible objective than trying to conserve after outer hair cell damage is almost complete. Such screening programs exist in the community for analogous conditions e.g. glaucoma, diabetes, breast cancer.

References:

Davis, A.C. 1989. The prevalence of hearing impairment and reported hearing disability among adults in Great Britain. *Int. J. of Epidemiology*, 18, 911-917.

LePage, E.L., Murray, N.M. 1993. Click-evoked otoacoustic emissions: comparing emission strengths with pure tone audiometric thresholds. *Aust. J. Audiol.*, 15, 9-22.

Murray, N.M., LePage, E.L. 1993. Age dependence of otoacoustic emissions and apparent rates of ageing of the inner ear in an Australian population. *Aust. J. Audiol.* 15, 59-70.

Murray, N.M., LePage, E.L., and Tran, K., 1994. Ageing characteristics of the Australian population in terms of otoacous-

tic emission strengths: global and individual picture. *Proc. Better Hearing Australia conference, Adelaide, Aug.7-11th, 1994.*

Measures of susceptibility to hearing loss: otoacoustic emissions and noise exposure.

Investigators: Eric LePage, Narelle Murray with Ken Mikl (WorkCover Authority, NSW)

Background: A pilot study was undertaken to investigate if the apparent predictive ability of the otoacoustic emission test has practical value to individuals exposed to high levels of noise for long periods. Four separate otoacoustic emission investigations were undertaken to quantify the extent of cochlear damage accumulated in environments marked by differing levels of sound exposure consistently maintained over a long period, either occupational or leisure noise exposure. These can be broadly classified as (1) a low sound level office worker population, (2) a moderate exposure to full-time orchestral musicians performing in an orchestra pit, (3) a high level exposure in a pilot study of 24 deep coal mine workers, and (4) a random sample of heavy music industry/media workers where the feature in common is frequent exposure to high-level amplified sound, encountered free field or via headphones.

The WorkCover Authority was requested by the Sydney Opera House Trust to make an assessment of occupational noise problems and to identify acoustic problems in the orchestra pit in the Opera Hall of the Sydney Opera House. The Hearing Loss Prevention Research Unit of NAL was invited to carry out otoacoustic emission testing on members of the Australian Opera and Ballet Orchestra as part of this investigation.

Research Questions: 1. To compare emission strengths of each group and compare with those of the Australian population obtained over the past three years; 2. To test whether the otoacoustic emission test is a useful measure of noise dose and therefore 3. as a "bioassay" for the effectiveness of hearing protection devices; 4. To test whether the definition of emission strength for permanent damage is adequate for temporary disturbance caused by noise exposure. 5. How does the measure of Coherent Emission Strength relate to acoustic measures of the orchestra pit and placement of members relative to other instruments?

Research Procedures: Otoacoustic emission screening tests were carried out on 24 mine workers before and after shifts over a period of ten days, with follow up carried out three months later. Their pure tone audiograms were obtained from their employer and estimations made of their noise dose and wearing of hearing protectors by an accompanying engineer. Otoacoustic emissions were obtained from 102 office workers during one working day. Approximately 90 orchestral musicians were tested three times during 1992, 1993 and 1994 for slow degradation changes. Records from our database of rock musicians, pub workers, recording engineers and frequent radio-headset users were selected and compared. In all cases questionnaire results were used to eliminate records for which there were significant complicating factors such as the presence of middle ear disease or histories of ototoxic drug usage.

Findings: The pilot Coal industry study revealed that it is possible to obtain a sensitive index of the level of outer hair cell motor performance, (Coherent Emission Strength, (CES dB SPL)), which is inversely related to the degree of cochlear damage. The CES values of office workers are comparable with those of a normative Australian population over the same age ranges. The orchestral musicians' emission strengths (mean) were lower than these, while the coal miners were considerably below the orchestra players, and the heavy amplified sound cases on average showed the lowest emission strengths.

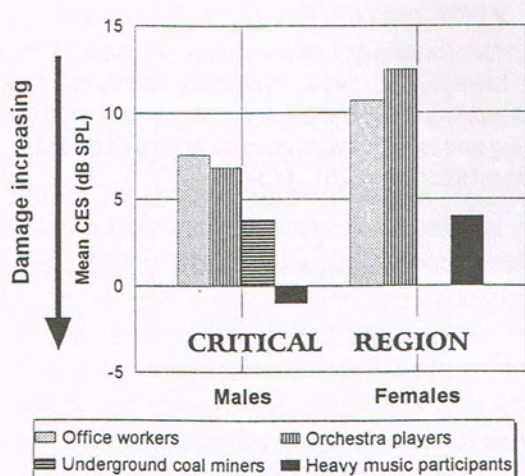


Figure 1 - Comparison of the mean values for the four groups, males and females.

The current definition of CES appears to be a sensitive measure of net outer hair cell performance, and therefore is a sensitive inverse measure of cochlear noise damage. To date the sample size from the coal industry has not been large enough to determine if the approach can be used to assess the long-term or short-term effectiveness of ear protection.

Significance: Coherent Emission Strength appears to be a useful parameter for assessing permanent damage and over a period of time could be useful for assessing the effectiveness of wearing hearing protection devices. A potentially very important application of transiently-evoked otoacoustic emissions for industry is as an objective cross-check on the validity of pure tone audiometry. With the exception of some retrocochlear lesions whose occurrences are relatively rare, emissions present at any frequency suggests that hearing thresholds should be normal at that frequency. The converse is not true. Lack of emissions at any frequency provides no clue as to the extent of hearing loss at that frequency.

Since this initial study was carried out, changes have been made both to the orchestra pit and to seating arrangements within the orchestra. Repeat measurements will be taken in 1995 to see if these alterations have helped maintain the stability of hearing of the orchestra members.

The studies undertaken with the orchestra, the coal miners and non-noise exposed workers have highlighted the value of otoacoustic emission screening for hearing conservation purposes. It has proved to be an easily applied, rapid and, most importantly an objective method of screening for cochlea damage. In particular, to be able to determine susceptibility in individuals amounts to being able to provide early warning of increased risk for hearing loss so that the individual will recognise the importance of taking precautionary measures to avoid excess exposure.

The project also aims to produce a better description of what constitutes excess exposure for the population as a whole and for different subgroups depending on age, gender, occupation, type of exposure, and most importantly depending on assessment of susceptibility for risk. In respect of the latter other definitions of susceptibility are being tried.

LePage, E.L., Murray, N.M. and Macrae, J.H. 1993. Otoacoustic emission assessment of ear damage in coal mine workers: Pilot study May-October 1992. National Acoustic Laboratories Commissioned Report No. 75.

WorkCover Authority, Acoustic Services. 1992. Noise Hazard Assessment - Australian Opera and Ballet Orchestra.

Cochlear Motor Cells

Investigators: Eric LePage with Professor Hans Peter Zenner and Dr Günter Reuter of the University of Tübingen, Germany.

Background: Loss or damage of outer hair cells of the cochlea is the primary cause of sensorineural hearing loss. The manifestation of their motor activity to provide for normal hearing sensitivity is the topic of intense research activity. Models of cochlear function, used extensively throughout hearing science, e.g. in speech processing, are based upon the Nobel prize-winning work of Georg von Békésy which predates the discovery of the motor action of these cells. The outcome of the current research is yielding a new class of cochlear models.

Research Questions: 1. What is the mode of action of the outer hair cells in living cochlear tissue? 2. What is the basis of the so-called cochlear amplifier? 3. Are the outer hair cells strongly involved in a regulation process which becomes disturbed in cases of tinnitus and/or Menière's disease. These questions are being approached by classifying the various motor characteristics by speed and mode of action in living cochlear tissue. All experiments have been carried out at, and funded by the University of Tübingen, subject to the stringent international codes with respect to animal research.

Findings: The outer hair cells display a mode of activity beyond that required of current models of the cochlear amplifier. The changes of length of the outer hair cells (LePage, 1989; Zenner 1993) constitute a major mode of action responsible for how the ear adjusts to changes in stimulus level over a 120 dB dynamic range, accounting for the loss of dynamic range in hearing loss and the effects of loud sound (LePage, Reuter and Zenner, 1993). A significant outcome is a first viable model for tinnitus (LePage, 1993) - unwanted sounds which are generated in the cochlea due to damage.

Significance: A major update to the cochlear amplifier theory with biproducts for explaining the origins of sensorineural hearing loss, variability of individuals in re-

sponse to loud noise which will have application to prevention activities, and a model for tinnitus which will be immediately useful in explaining the condition to tinnitus sufferers.

References:

LePage, E. L. 1987. Frequency-dependent self-induced bias of the basilar membrane and its potential for controlling sensitivity and tuning in the mammalian cochlea. *J. Acoust. Soc. Am.*, 82, 139-154.

LePage, E.L. 1989. Functional role of the olivo-cochlear bundle: A motor unit control system in the mammalian cochlea. *Hear. Res.*, 38, 177-198.

LePage, E.L. 1993. A model for cochlear origin of subjective tinnitus: excitatory drift in operating point of inner hair cells, In, *Tinnitus Mechanisms*, ed. by J. Vernon and A. Möller, Allyn and Bacon, Boston, (in press).

LePage, E.L., Reuter, G., and Zenner, H-P. 1993. Summating baseline shifts and mechanical adaptation in a guinea pig cochlear explant shown with two displacement measuring techniques. In, *Biophysics of hair cell sensory systems*, edited by H. Duifhuis, J.W. Horst, P. van Dijk and S. van Netten. World Scientific Publishing, (in press).

Zenner, H.-P. 1986. Motile responses in outer hair cells. *Hear. Res.* 22, 83-90.

Zenner, H.-P. 1993. Possible roles of outer hair cell d.c. movements in the cochlea. *Brit. J. Audiol.* 27, 73-77.

Zenner, H.-P. Reuter, G., Zimmermann, U., Gitter, A.H., Fermin, C., LePage, E.L. 1994. Transitory endolymph leakage induced hearing loss and tinnitus: depolarization, biphasic shortening and loss of electromotility of outer hair cells. *Eur. Arch. Otorhinolaryngol.* 251: 143-153.

Selective Attention

Investigators: Eric LePage, with Dr Patricia Michie and Dr Nadia Solowij of the School of Behavioural Sciences, Macquarie University.

Background: Pure tone audiometry is an exercise in selective attention. It is a measure of how well any person can extract a pure tone from background noise. The first sign of hearing loss is that this selection process is diminished in conditions of speech in raised background noise. There are several reasons to think that the selection process normally involves activity of the outer hair cells which are connected to the brain and to the responses of the opposite ear (LePage, 1989). Speech discrimination and spatial selection may therefore be fundamentally dependent on normal outer hair cell function which is why noise damage leads to difficulties in discrimination.

Research Questions: Otoacoustic emissions are good measures of the activity of outer hair cells. Therefore, are emissions strongly affected by voluntarily selecting one tone in a pair of tones so that the brain regards the non-attended tone as background noise?

Research Procedures: Normally hearing subjects are tested for otoacoustic emission strength to determine suitability for the experimental protocol. Subjects then listen to an exacting task where they must identify target sounds and respond with a button push to confirm identification of the various tone bursts while the stimulus conditions remain unaltered. The amplitudes of the resulting otoacoustic emissions are compared to determine whether the sounds are being attended to or not.

Findings: A small attentional effect has been seen by a number of investigators. However, thus far our experiments have not confirmed this result, despite considerable theoretical justification for thinking a significant effect exists. Five independent experiments were conducted primarily on fixed target tone bursts; the first four was for switching attention between two targets being presented to the same ear; in the fifth experiment the subject had to alternate attention to one ear and then the other. No significant effects were observed in any of the experiments (Michie et al., 1994).

Significance: The most common complaint of the hearing impaired is the interference to their hearing by background noise. Background noise constitutes the

most significant technical problem to be overcome in new hearing aid designs. A positive result should provide more understanding of the background noise problem and could result in the redefinition of noise as the "loss of voluntary selection". It could also reveal why other investigators have failed to demonstrate selection at the level of the auditory periphery. The negative outcome of these current experiments was surprising considering that two other investigators have seen small but statistically significant effects. On the one hand this might be taken to imply that selection as a process is carried out in higher brain centres as has always been suspected. On the other hand, failure of this experiment tends to indicate that the paradigms tested were not sufficiently comprehensive to show an effect. For example, a stronger effect may be seen with more care taken to reveal spatial selection effects or dynamic focussing effects.

References:

- LePage, E.L. 1989. Functional role of the olivo-cochlear bundle: A motor unit control system in the mammalian cochlea. *Hear. Res.*, 38, 177-198.
- Giard, M-H., Bouchet, P., Collet, L., and Pernier, J. 1992. Auditory selective attention at the cochlear level. Presented at the 5th International Conference on Cognitive Neurosciences (ICON V) Jerusalem, Israel, June 14-19, 1992.
- Puel, J-L., Rebillard, G., Bonfils, P. and Pujol, R. 1989. Effect of visual selective attention on otoacoustic emissions, in *Cochlear Mechanisms*, ed. by J.P. Wilson and D.T. Kemp, Plenum Publishing Corp.
- Michie, P.T., LePage, E.L., Solowij, N., Haller, M., and Terry, Lyn. 1994. Evoked otoacoustic emissions and auditory selective attention. (submitted).

Screening Criteria for Newborns

Investigators: Eric LePage, Narelle Murray, Roger Lovegrove, with Dr. David Starte (Royal North Shore Hospital), AHS staff.

This project is looking at special cases of the Age study, i.e. neonates and infants, for which data is being collected from a variety of sources, with values for specificity and sensitivity being determined. Those who fail the criteria would be referred on for ABR testing or Electrocochleography.

Preliminary results from the study suggests that neonatal screening passes should require CES values of 20 dB SPL, while partial passes should be 14 dB SPL, with failures dB SPL.

Advanced Signal Processing

Investigators: Eric LePage, Dr Michael Harrap (ADFA), Khanh Tran.

Otoacoustic emission methods provide much more information than pure tone audiometry. However, even more information is contained in otoacoustic emission records than is currently being used. At present the spectra of the click-evoked response is an average of all the activity of the record in time. By contrast, using the short-time Fourier transform it is possible to display dynamic variations in the spectra giving a time picture of how the ear responds to a click (LePage, et al., 1993). Since impulse noise, e.g. gunfire, is the most damaging form of noise and represents a major problem for Defence Forces, the project is aimed at deter-

mining susceptibility to hearing loss in terms of how well the cochlea can protect itself by dynamically adapting to the fast pressure wave front. There is a significant commercial potential for the project by virtue of updating current recording methods.

This project has received the lowest priority during 1993/94 and will receive more attention during 1994/95.

Reference:

LePage, E.L., Murray, N.M., Tran, K. and Harrap, M.J. 1993. The ear as an acoustical generator: otoacoustic emissions and their diagnostic potential. *Acoustics Australia*, December 1993.

Prevention of Hearing Loss

Predictions of a rise in hearing loss in young people.

Investigator: Eric LePage

Background: The accumulation of inner ear damage can be tracked using otoacoustic emissions for years prior to a critical level of damage being reached at which time the symptoms of hearing loss become manifest (Murray and LePage, 1993; LePage and Murray, 1993). We have translated the concept of the rise in cochlear damage to that of rates of aging of the ear. In these terms presbycusis, or the normal aging effect may be re-defined as a minimal rate of aging of the ear, or a decline in emission strength of 2 to 3 dB per decade. In the Australian population, the group over 40 years of age seems to be declining at about 3 dB per decade. However, many young people are being tracked at declining at between 20 to 30 dB per decade. Prominent amongst this group are those who regularly engage in heavy music or leisure noise exposure and who are already reporting symptoms of hearing loss, even by age 15. Otoacoustic emission recordings from such ears show very clear signs of early aging, similar to the ears of older workers in heavy industry.

Research Questions: As the ear ages the emission strength declines, a critical level is reached at which hearing loss is reported. Does the dip in the aging characteristic centred on teenagers represent

a group of individuals who will reach this critical level prematurely? How many people would reach this level? What would be the time course of this process? Could the result be regarded as an epidemic? How long would it take for the number of younger people reporting hearing problems to equal the number of older people suffering presbycusis? What would be the costs? Since industrial hearing loss is almost exclusively a male condition, how would the current trends affect the ratio of incidence in males and females? Would this trend cause a rise in communication difficulties in young adults?

Research Procedure: Using ABS figures for the Australian population in past years plus projections for growth in the future, the rates of rise in numbers of individuals reaching critical values of ear damage have been modelled, subject to various assumptions. These assumptions describe a) the current trend, b) the effect on the numbers affected if the rates of aging were reduced. Implemen-

tation of the computer model was a two stage procedure. The first stage was to establish a curve describing the current rates of aging in the population and to show if this varies with age. For this it was necessary to feed in our current 'snapshot' picture of emission strengths with age for males and females and to make some assumptions as to what it might have been like at some stage in the past. It was assumed that the decline in emission strengths were linear with age back in 1980. Linearity of decline was chosen a) because the aging curve above age 40 is linear, and b) because of lack of information which would make any other choice less arbitrary. Ideally one should have started the process several generations ago; then the actual period becomes arbitrary. On the other hand the period had to be sufficiently short so as to adequately represent the current trend. The second stage involved using the derived curve to project how the emission strengths would behave in the future.

Findings: A key output of the computer model is a curve described as a *rate-of-aging* curve, plotted versus age (see Figure 1) which shows that the rate of decline of emission strength, interpreted as the *rate of damage accumulation*, is greatest in the Australian population for young people.

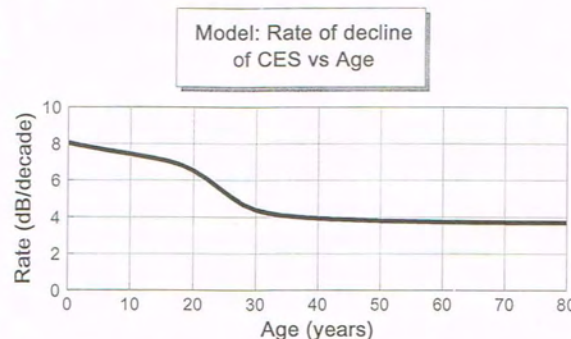


Figure 1. The *rate-of-aging* curve, plotted versus age showing that the decline of emission strength (CES) interpreted as the *rate of damage accumulation*, is greatest in the Australian population of young people.

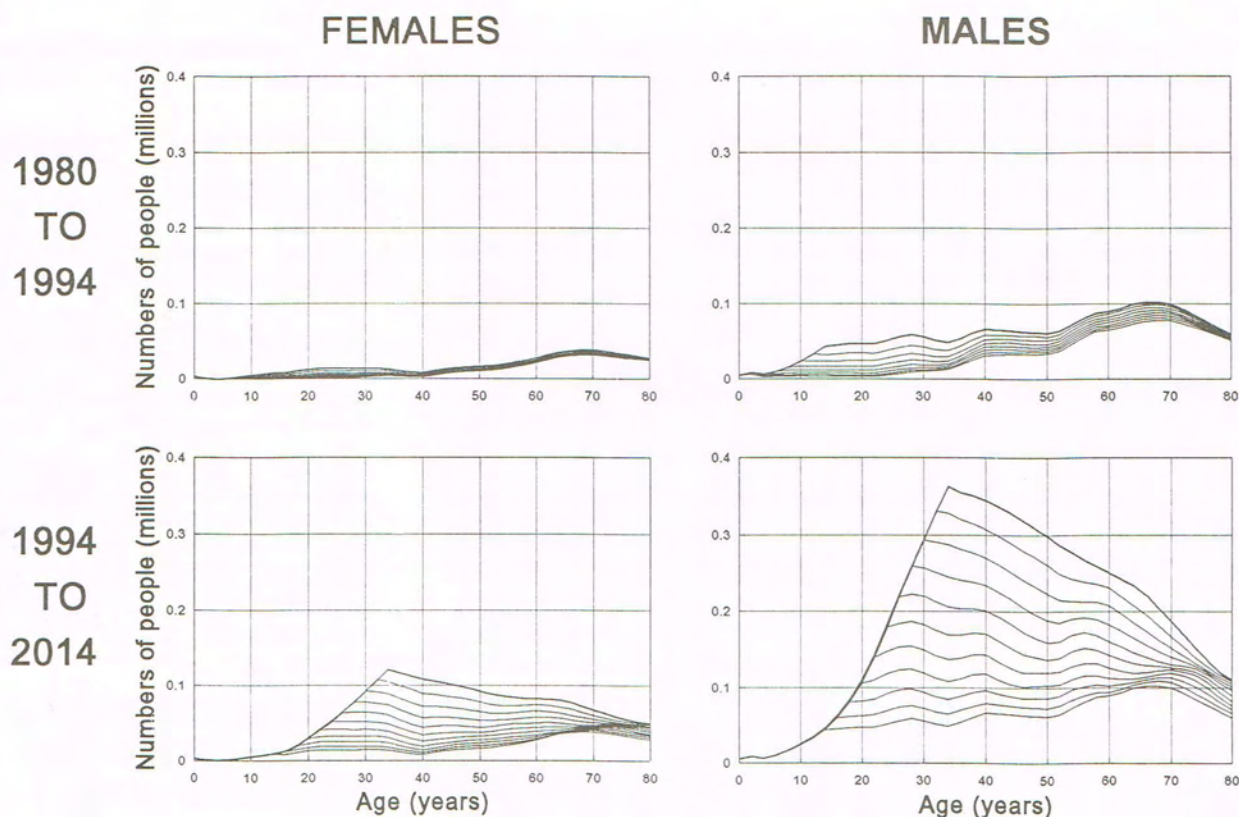


Figure 2. Incidence of Australian Individuals (top panels) who have at least a mild hearing loss (derived from model). The trend-establishment phase (top panels) is used to match the existing incidence of hearing loss in 1994. The ordinate values represent the number of individuals in each two year age bin used in the computation. Each family of lines in the top panels represents the projected numbers affected from 1980 (lowest), 1982, 1984, etc., to 1994 (highest) respectively. The projection phase of the model (bottom panels) reflects the expected numbers for the next 20 years. The lines represent the years 1994, 1996, 1998 etc., to 2014, respectively.

It averages between 7 and 8 dB/decade in the first 20 years and then declines to about 3.5 dB/decade for ages 40 and over. This feature is difficult to explain unless people in their first 20 years, on average, are currently accumulating cochlear damage faster than the previous generation was at the same age. Other explanations for the aging curve are indeed possible. However, our evidence that a high proportion of young people do have more highly damaged ears than older people supports this assertion. This suggestion that premature aging is occurring is in sharp contrast to the expectation that for most people the *presentation of symptoms* will not occur until later in life. Figure 2 shows the incidence of Australian individuals who have *at least* a mild hearing loss. The ordinate values rep-

resent the number of individuals in each two year age bin used in the computation. The model was established to reflect actual numbers in the top two graphs, while the trend shown in Figure 1 is used to

project the expected numbers for the next 20 years.

Note that the current hearing loss picture remains predominantly a phenomenon of old age (presbycusis), but that

Table 1. The total numbers of Australians possibly affected with at least a mild hearing loss (CES values less than -3dB SPL) as shown in the bottom panels of Figure 2.

MALES	1994	2000	2004	2014
Total population (M)	8.5	9.1	9.4	10.2
Population affected (M)	2.5	3.7	4.8	8.0
Percent affected	29	41	51	78
FEMALES				
Total population (M)	8.5	9.1	9.4	10.2
Population affected (M)	0.7	1.1	1.4	2.6
Percent affected	9	12	15	25



Otoacoustic emission testing (l. to r. Khanh Tran, Narelle Murray and Eric LePage).

even now the model shows the rising numbers of young people experiencing a hearing loss. The extrapolations of the model show that hearing loss in the Australian community will become predominately a young-adult phenomenon in the future with steeply rising numbers in the case of males. If the model is realistic in its depiction of the current situation, the continuity which is inherent in the model suggests that a trend toward premature hearing loss is well established in Australian young adults, as the very long term effects of accelerated early ear damage already accumulated eventually become manifest and are confirmed with pure tone audiometry. The projected total numbers of Australians affected is shown in Table 1.

Significance: The costs of such a marked increase in hearing problems -- loss of selectivity, abnormal sensitivity characteristics, and rise in internal noise (tinnitus), can only be guessed. It suffices to suggest here that the figure for direct and indirect costs will be large because very many more people will be affected than is currently the case for noise-in-

duced hearing loss in industry (e.g. the cost of providing hearing rehabilitation services and hearing aids to millions of people). Indirect costs such as the costs of increasingly poor communication between young adults, could be correspondingly larger. Irrespective of the magnitude of the problem, the model indicates that the greatest preventative effort should act to stem the trend as early as possible, e.g. in educating primary school-age children. (The recent production of the videotape directed at this age group, "It Won't Come Back" by AHS Prevention Services is a significant achievement in this regard). The model also shows that if it were possible to achieve an instant change in attitudes to the point of changing the rate of aging curve (down from 8 dB/decade to, for example, a constant 3 dB/decade from age 10), the projected rise could be dramatically reduced so that only 17 percent of the population would be affected by the year 2004 and 21 percent by 2014. Such a change could be pursued only through a massive education program. The costs of such a program would

be high, but much less than the projected costs.

References:

LePage, E. L. and Murray, N. M. 1993. Click-evoked otoacoustic emissions: comparing emission strengths with pure tone audiometric thresholds. *Aust. J. Audiol.*, 15, 9-22.

Murray, N. M. and LePage, E. L. 1993. Age dependence of otoacoustic emissions and apparent rates of ageing of the inner in an Australian population. *Aust. J. Audiol.*, 15, 59-70.

LePage, E. L., 1994. 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. In: Better Hearing Australia conference, 7-11 August, 1994, Adelaide, South Australia.

Tracking the growth of cochlear damage in a cohort study.

Investigators: Narelle Murray, Eric LePage

Background: A key objective of the normative study (LePage and Murray, 1993, Murray and LePage, 1993, Murray, LePage and Tran, 1994) was to compare pure tone audiometric thresholds with measures of emission strength and compare how each depends on aging. A major outcome from this study of the population as a whole was the derivation of a parameter which may be used as a risk factor for hearing loss according to the emission strength, or lack thereof for any ear. Viewing the population decline in emission strength and, subsequently, decline in hearing sensitivity did not provide a description of how these parameters changed in the case of individual ears. Whereas the decline in emission strengths for the whole population was around 3 dB/decade, individual cases were shown as declining at varying rates, up

to 30 dB/decade (Murray and LePage, 1994).

Research Questions: It is of interest to know how individual ear emission performance varies with time. What is the normal test-retest variability for any particular person and ear? Do the emission strengths vary systematically over a longer period? Do the emissions generally decrease with time or may they increase? Does the change in both ears go in the same direction? In the cases of decline in ear activity, is it valid to interpret this as a rise in ear damage? Does ear damage accumulate steadily from one year

to the next, or do individuals have episodes of faster aging? Are such episodes related to age, illnesses, periods of drug taking, episodes of noise exposure? What are the characteristic rates of aging for people in different occupations? Do rates of aging taper off as age increases, e.g. as adulthood is entered? In the cohort, if individuals with lower emission strength do indeed report hearing problems before those with higher emission strength, is this a useful first definition of "susceptibility" to hearing loss?

Research Procedures: A preliminary study was con-

ducted each day for one week on 12 subjects to determine the test-retest variability of the emission strengths, so that any change over a longer period could be assessed as to its significance. Click-evoked otoacoustic emission auditory assessment and pure tone audiometry has been carried out on 50 males and females selected for the main study, 30 of whom were first tested in 1989 and have subsequently been seen annually. On each, tympanometry has also been carried out as a screening procedure to eliminate the possibility of middle ear problems at the time of testing. Aural histories have been updated at

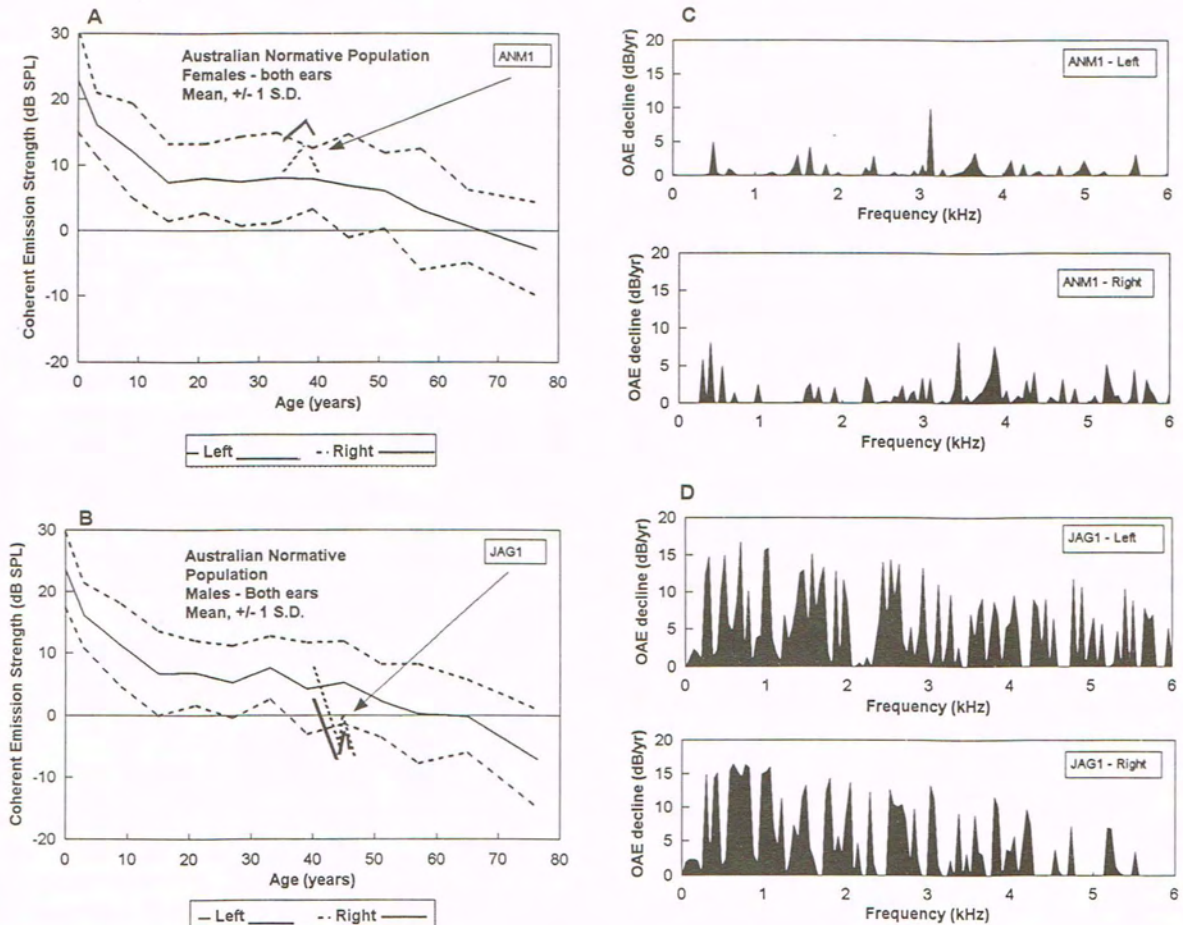


Figure 3. Left panels A and B: Normative age data for ages 0 to 80: females and males - top and bottom panels respectively; Mean (solid line) ± 1 S.D. (dashed lines). Superimposed are results for one female and one male for four years: solid lines connect points for left ears; dotted lines for right ears. Right panels C and D: The height of the peaks represent the average decline in emission spectra versus frequency for each of the subjects in A and B (left and right ears respectively). Regions of negative decline do not show as pronounced spikes since they are not associated with notches in the spectrum and are not shown.

each interview to determine any pathologies which may have occurred since previous testing was carried out.

Findings: Preliminary findings for two subjects, one female and one male, are presented to illustrate the approach. In Figure 3 (left panels), the normative age data are summarised for all ages 0 through 80, showing the mean (solid line) one standard deviation (dashed lines) for females and males (top and bottom panels respectively). On these panels are shown the results for one female and one male for four years in succession. A solid line connects points for left ears while dotted lines connect points for right ears. The average rates of change in emission sound levels (OAE decline dB/yr) is computed for each frequency in the spectrum (right panels). Left ear spectra are above right ear spectra in each case. These panels show that the decline in the emission is not uniform across frequency, but tends to accumulate at localised frequencies as the notches in the spectrum deepen and widen. In the case of the female with high emission strengths, the specific frequencies at which change has taken place are much fewer, and of lower rate of decline, than the regions where change has taken place for the male with low emission strength. The preliminary findings suggest that several individuals who commenced the study with a high Coherent Emission Strength (CES) value have tended to retain this high CES and have a low rate of decline. To the contrary, those who entered the study with a low CES value (in the lower 16% of the population) appear to have a much higher rate of decline. No intervening pathologies seem to account for this latter result, although previous noise exposure may explain this particular subject's initial low result.

Significance: One possible interpretation of these spikes in the right panels is that each represents a patch of damage at some point along the basilar membrane. That is, the frequency plot of spikes may represent some form of map of damage sites or outer hair cell lesion areas. In the case of the male subject these results suggest that damage has been accumulating at many sites quite rapidly over the three year span, whereas for the female, damage is still being seen to occur but only at specific places. The audiograms of these subjects show no signs of change. The otoacoustic emission approach therefore may be offering a method of tracking 'microlesions' or tiny damage sites inside the cochlea, long before any hearing disturbance occurs. As such this is the first time that such lesions have been tracked in humans. All previous work picturing ageing-type damage has occurred in animal studies (Bohne et al, 1990). It is particularly interesting that the results to hand already suggest that the lesion sites may be sites of physical weakness. These results support earlier studies suggesting that noise-induced hearing loss is the end product of the growth of these microlesions in size (Bohne et al., 1987). The approach therefore may be highly useful for hearing loss prevention in terms of being able to determine who has "tough" ears and who has "tender" ears by estimating the number or density of such lesions. Considering the expense and insensitivity of present hearing conservation methods, otoacoustic emissions appear to offer substantial improvement. No longer should the acquisition of a hearing loss due to accelerated aging occur without being anticipated and controlled.

If it is possible to enter life with a high emission strength (as is seen in our normative population results) and ex-

clude some or all of the pathogens and noisiness of life early in life, it may lead to a retention of outer hair cell capacity and a much lower rate of decline which is manifested in a lesser susceptibility to hearing loss later in life. If, however, there is early disturbance (or low emission strength very early in life) the individual can be forewarned and early intervention measures put into place to conserve what cochlear performance remains.

References:

Bohne BA, Yohman L, and Gruner MM. 1987. Cochlear damage following interrupted exposure to high-frequency noise. *Hear. Res.*, 29, 251-264.

Bohne BA, Gruner MM, and Harding GW. 1990. Morphological correlates of aging in the chinchilla cochlea. *Hear. Res.*, 48, 79-92.

LePage, E.L., Murray, N.M., and Tran, K. 1994. Comparison of otoacoustic emission measures of cochlear damage in the Australian population with hearing loss in the Australian and British populations. Proceedings Better Hearing Australia Conference, Adelaide, August, 1994.

Murray, N.M., and LePage, E.L. 1993. Age dependence of otoacoustic emissions and apparent rates of ageing of the inner ear in an Australian population. *Aust. J. of Audiol.* Vol. 15/2, 59-70.

Murray, N.M., LePage, E.L., and Tran, K. 1994. Ageing characteristics of the Australian population in terms of otoacoustic emission strengths: global and individual picture. Proceedings of the Better Hearing Australia Conference, Adelaide, August, 1994.

Defining "dynamic" susceptibility to hearing loss in relation to the problem of impact noise.

Investigators: Eric LePage, Khanh Tran, Michael Harrap (ADFA).

Background: There is a growing view amongst the scientific community that while

loud sounds produce varying amounts of damage, sharp sounds of short duration, e.g. impulses and impacts, may be relatively much more damaging than purely on an equal-energy basis (Price, 1992). Most cases where the ear responds with acute pain or ringing are due to the fact that the bang occurred without warning. Advanced warning of a blast may protect the ear by the action of the stapedius reflex. Indeed some experts believe that noise-induced hearing loss may therefore be very largely caused by the accumulation of the damage caused by mini-traumatic events (Bruehl; Campanella; pers. communications). There are strong theoretical reasons for supporting this view which include a model of the outer hair cells in the cochlea, which are involved in a rapidly acting regulation process. This process continually adjusts the natural sensitivity of the ear according to ambient sound levels. Our working hypothesis is that while the efferent control loop constitutes a very efficient internal cochlear protection mechanism for loud sounds, the protection breaks down when the rise in sound level is very rapid (1 msec). The middle ear-, or acoustic-reflex is about 100 times too slow to protect the inner ear against such transients (unless it is pre-stimulated; see Acoustic Reflex Eliciting Earmuff project -- N.Carter).

Loud continuous sounds will produce a temporary threshold shift which recovers at a rate which decreases with time after cessation of sound. Many studies have shown that the hearing threshold shift and recovery after impacts is much less predictable. In a study conducted on military personnel in France (Dancer et al, 1991), high impact noise often did not produce the greatest hearing loss till the next day, i.e. there was a delayed effect.

These kinds of physiological responses make havoc of conventional experiments designed to quantify, understand and protect against impact noise. We expect that the highly variable outcomes are connected with how well the outer hair cell/efferent protection mechanism functions to protect itself. The efficiency of

the mechanism will depend vitally upon (a) waveforms and spectrum of the blast and (b) the complement and spatial pattern of the remaining outer hair cells. We have shown that otoacoustic emissions are very good at generating data that appear to describe the physical extent of ear damage. In addition, click-evoked otoacoustic

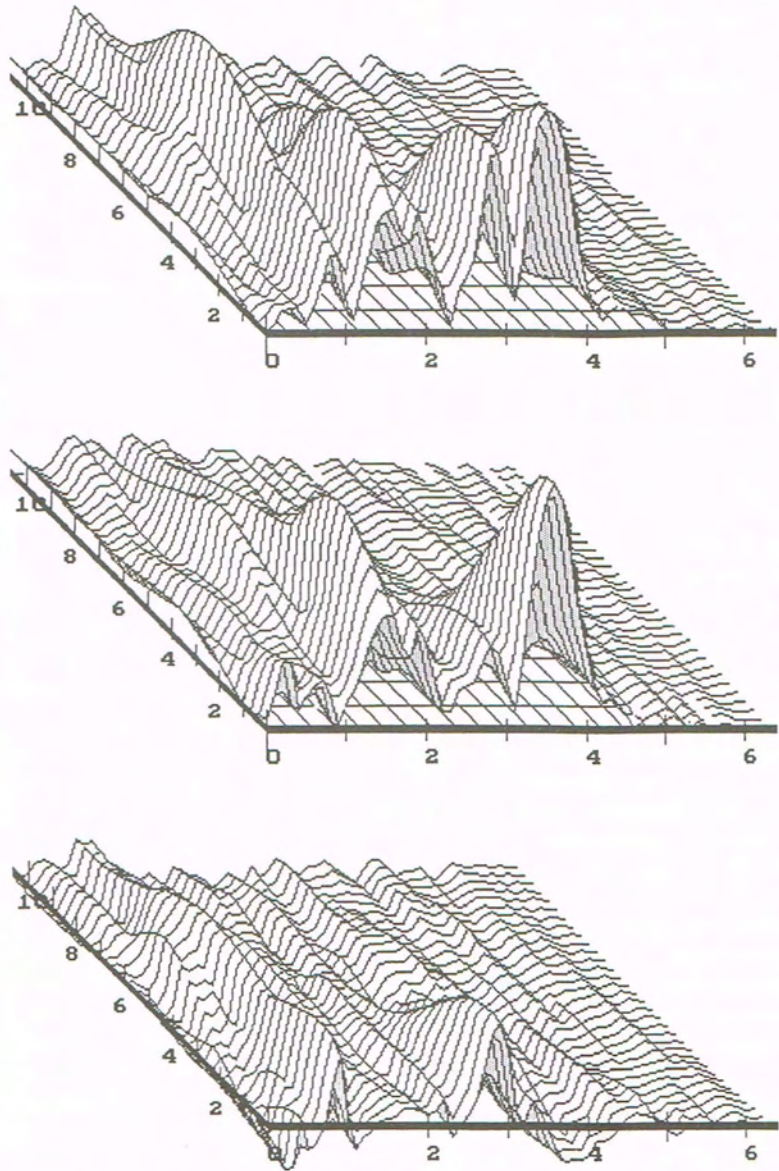


Figure 4. The effect of a pure tone exposure. The three panels are time-frequency surfaces depicting how a spectrum of a click-evoked acoustic emission changes with time as a series of spectra one behind the other. Each response is obtained from standard screening mode, using signal averaging of 1040 clicks. The frequency range shown is from 0 to 6 kHz, while the time axis goes back into the paper from 0 frequency (the highest signal peak is -6dB SPL, the level at the plane is at -16dB SPL). The top panel shows the normal emission before the exposure. The second panel is for a record completed six minutes later. In between the ear was exposed at 3 kHz for one minute at 90dB SPL (80dB HL). The early emission peak shows the difference (before minus after). Low frequencies in the response have also been affected.

emissions are particularly suited to describing dynamic, protective-type responses in the human ear. This is because the raw data captured is a time-response from which the frequency spectrum is calculated. It has been possible for example, to make recordings at five minute intervals which demonstrated variations in cochlear mechanical responses which were used to identify a case of a perilymphatic fistula.

Research questions: Do the dynamic responses captured with otoacoustic emissions reflect a protective reflex which gives rise to temporary threshold shift? Is it possible to use the technique to characterise the susceptibility of a person to hearing loss (permanent shift) more precisely in terms of the ear's dynamic protective response to sound? Our working hypothesis is that it may be possible to obtain a series of recordings on a subject, and at some point deliver a stimulus to the ear which would evoke the outer hair cell protective response, then watch the extent of the shift and the rate of recovery. A healthy ear might be expected to produce a rapid reaction to the acoustic challenge and recover quickly, a more susceptible ear react more slowly and recover more slowly. In the past only relatively high level sounds could produce a clear temporary audiometric shift, or an acoustic reflex, but with otoacoustic emissions, much smaller doses may produce a usable result without the complication of the acoustic reflex. With the ample frequency specificity of click-evoked otoacoustic emissions, it should be possible to show how the pattern of existing damage affects the resulting exposure and damage picture, and manipulate it with contralateral stimulation to simulate real world protection.

Research Procedures:

The first stage of this project seeks to obtain a much more complete display of all the information available in the standard click oto-emission and to use this to provide a time-frequency map of the emission, which we expect to be interpretable as changes to the internal protection mechanism. The second stage is to test the power of the approach with deliberate exposures to pure tones and noise, an approach which would not have been practical or acceptable on ethical grounds in times past. The primary difference now made possible by the higher sensitivity of emissions over pure tone thresholds is that non-traumatic exposures (e.g. 90 dB SPL for one minute) can produce a marked change. For this preliminary experiment subjects with normal hearing and sizeable emissions were chosen for recording before and after a small acoustic dose (actually well within the limits described by Australian Standard AS1269 -Hearing Conservation). The results were analysed with time-frequency analysis to display the time dependence of the emission spectrum, akin to a voice spectrogram. The raw data was collected with an Otodynamics analyser and the displays generated with an Ariel "Cyclops" card employing a TMS320C40 processor.

Findings: Figure 4 shows an example of the application of the approach. The three panels show otoacoustic emissions from the right ear of a female subject with sizeable emissions. The three-dimensional display is a series of curves one behind the other, showing how the spectrum of the emission to 6 kHz varies with time going 10 ms back into the page. The actual features of the top plot are strongly characteristic of the ear. Click-evoked otoacoustic emissions generally decrease in their high frequency

content with time. The middle panel shows the same ear immediately following a 3 kHz, 90 dB SPL sound exposure lasting just one minute. This plot shows quite marked changes in the features of the spectrum, in particular a reduction in the size of the emission peak at around 3 kHz and the rise in the peak on the high frequency side of it. The third panel shows changes in the form of a surface representing the change in performance of the ear (before minus after). The difference plot also shows that the response to frequencies below 1 kHz were also affected by the tone.

Significance: This result is of strong theoretical interest considering the "half-octave shift" phenomenon which causes a temporarily deafened ear to lose sensitivity, not at the frequency of exposure, but at a higher frequency. It means that the active elements inside the cochlea which generate the emissions at 3 kHz are likely generating less energy at 3 kHz and more at 4 kHz following the exposure. This may imply that as part of the protection mechanism, the mapping of sound frequency to place of detection is warped. The result may be interpreted as the protection mechanism acting to disperse the sound energy (LePage, 1991). This is the first time *physical* evidence of remapping has been shown for the *human* ear, suggesting the mode of action of the cochlear protection mechanism which is much more finely attuned than the stapedius reflex. Of practical interest, is the realisation that otoacoustic emissions provide a sensitive measure of ear disturbance analogous to temporary threshold shift, i.e. producing a detectable change for a smaller noise dose than required to show an audiometric shift. The high sensitivity explains the difficulty in interpreting earlier results in which

coal miners were tested before and after shifts (LePage, et al., 1993) and military personnel were tested for response to acoustic impact (Carter, et al., 1995). Evidently these high level exposures upset the delicate regulation mechanism so that the results appear random. Such new understanding could modify approaches to handling impulse noise and be used as an improved measure for susceptibility of workers to noise, particularly impact noise.

References:

Carter, N.L., French, T. and LePage, E.L. 1995. Evaluation of the aural reflex eliciting (AR) earmuff. Progress Report No. 2. National Acoustic Laboratories, Sydney

Dancer, A. L., 1992. Status and shortcomings of military noise standards. In: Noise-Induced Hearing Loss, edited by A.L. Dancer, D. Henderson R. J. Salvi and R. P. Hamernik. Mosby Year Book, St.Louis, 513-520.

LePage, E. L., 1992. Hysteresis in cochlear mechanics and a model for vari-

ability in noise-induced hearing loss. In: Noise-Induced Hearing Loss, edited by A.L. Dancer, D. Henderson R. J. Salvi and R. P. Hamernik. Mosby Year Book, St.Louis, 106-118.

LePage, E. L., Murray, N. M., and Macrae, J. H., 1993. Otoacoustic emission assessment of ear damage in coal mine workers: Pilot study May - October 1992. In: National Acoustic Laboratories Commissioned Report 75, Sydney, Australia.

Price, G. R., 1992. Importance of spectrum for rating hazard: theoretical basis. In: Noise-Induced Hearing Loss, edited by A.L. Dancer, D. Henderson R. J. Salvi and R. P. Hamernik. Mosby Year Book, St.Louis, 349-360.

Ear damage in orchestral musicians.

Investigators: Eric LePage, Narelle Murray with Ken Miki (WorkCover Authority, NSW)

Background: Since 1992 the WorkCover Authority has undertaken noise measure-

ments in the pit of the Sydney Opera House as well as audiometrically monitoring the hearing of the members of the Australian Opera and Ballet Orchestra. As part of these monitoring procedures the Hearing Loss Prevention Research Unit has carried out auditory assessments of the Orchestra using otoacoustic emission testing.

Research Questions: 1.

To compare emission test data over the three years, 1992, 1993, 1994, for the Orchestra as a whole; 2. To investigate differences, if any, between sections of the Orchestra; 3. To compare individual test results to observe any decline or rise in otoacoustic emission strength.

Research Procedures:

Otoacoustic emission screening tests were carried out on a total of 112 orchestra members, 77 of whom were seen for

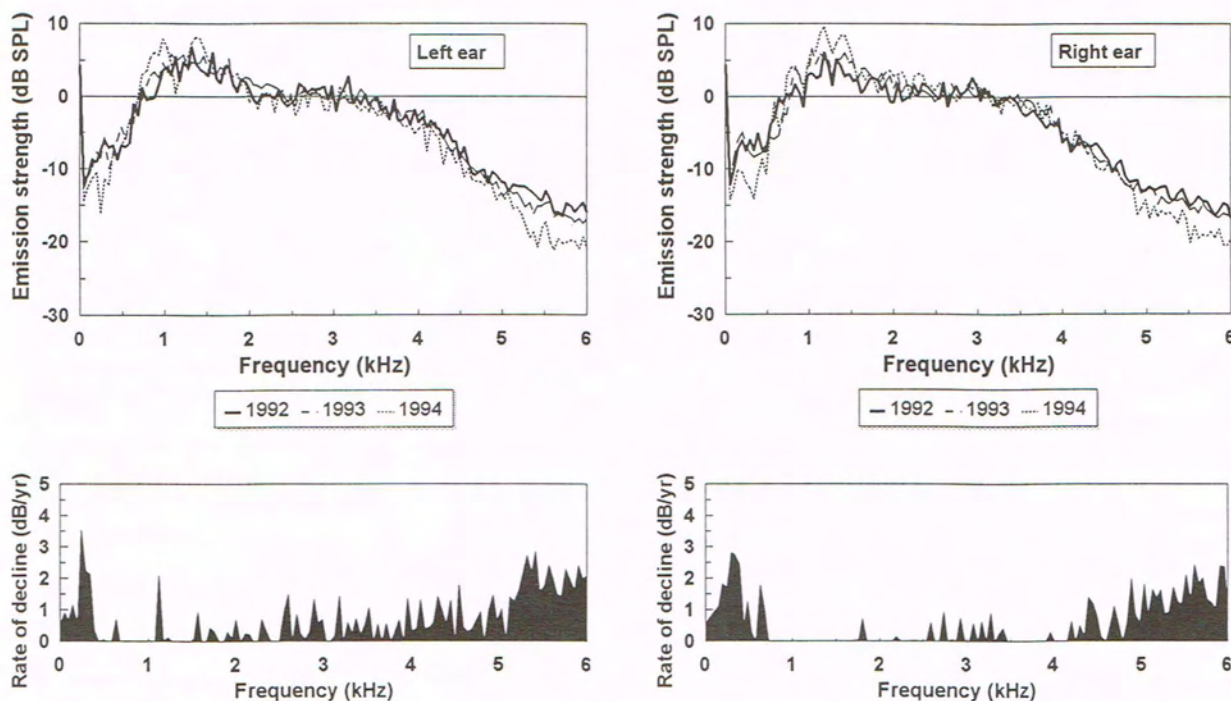


Figure 5. Top panels depict mean spectral emission values averaged across all members of the Orchestra for the years 1992, 1993 and 1994, left and right ears. Bottom panels: the peaks represent the rate of decline in emission spectra over the three year period for the whole Orchestra.



Measuring noise levels during Orchestra rehearsals.

a second time, and 44 of these for a third time (i.e. 1992, 1993 and 1994). Questionnaires were completed, with those seen for a second or third time completing questionnaires supplementing information obtained originally. Pure tone audiometric results were obtained from the WorkCover Authority testers on the same day.

Findings: As seen in Figure 5 the spectral values had the highest rate of decline for low frequencies as well as the high frequencies which are traditionally associated with cochlear damage from exposure to noise. These results were apparent in all sections of the Orchestra, although individual results varied considerably. Pure tone audiometry results remained steady over the three years for the Orchestra as a whole, and did not reflect the trend evident in the emission results. The decline was more marked in left ears and is

consistent with our earlier findings. The lower panels show the spectral shape of the rate of decline in the whole orchestra population. Plots also exhibit a trend suspected previously: a decline in high frequency emissions may be accompanied by a rise in emissions around 1 kHz, a feature of theoretical interest concerning the way the ear fails.

Significance:

The results show that it is possible to track slow declines in ear performance of a group before there are significant audiometric changes. Alterations have been made to the Orchestra pit and hearing protection issued to some members of the Orchestra only after completion of these tests; therefore more significant changes may be apparent after monitoring is carried out in 1995.

Evaluation of the Aural Reflex Earmuff

Investigators: Dr N L Carter (NAL); Dr Han Tin French (DSTO)

Background: Previous research has shown that the aural reflex can protect hearing. The aim of the present study was to determine whether the addition of the acoustically stimulated aural (stapedius) reflex immediately before firing improved the hearing protection of earmuffs worn by artillerymen.

Procedures: Soldiers were exposed to single shots of simulated howitzer noise. Hearing and otoacoustic emissions were tested immediately before and after firing. These tests, using single 'shots' were designated Phase 1 of the research program.

Findings: No effect was found on acuity thresholds nor on otoacoustic emissions, even when the aural reflex was not elicited. These results agree substantially with US research suggesting that the protection afforded by earmuffs against impulse noise is substantially greater than indicated by tests using steady state sounds.

The results may also have implications for Military Occupational Health and Safety regulations requiring all personnel to wear double hearing protection (plugs and muffs) within 5 metres of a howitzer during firing.

Phase II Proposal: A final report on Phase I was written and presented to the Australian Defence Medical Ethics Committee (ADMEC). A new proposal, to carry out Phase II of the project, was presented to ADMEC and the Defence Scientific Adviser. The plan was approved, and preparations were completed. Phase II, the final phase of the project, will test the effects of exposure to multiple 'shots' of simulated howitzer noise.

A New Approach to Hearing Loss Prevention

Investigators: Eric L. LePage,
Narelle M. Murray

Preventative medicine has as its aim the preservation of normal function for as long as possible. Early detection of a debilitating or life-threatening condition is essential for corrective action, and the earlier the warning, the more time exists to apply the correction.

Prevention programs fall into two basic categories. The first includes conditions for which precursors have not yet been identified and which are therefore the more insidious. Secondly, there are those for which precursors exist, which can be identified by screening, but which may proceed to the full blown condition if untreated. Obviously the most successful preventive action can occur if precursors exist. For example, breast cancer is a condition in the first category. The first signs are positive identification of the condition which then requires immediate corrective action which is often radical and traumatic. On the other hand, Insulin Dependent Diabetes Mellitus (IDDM) falls into the second category, since clinical precursors have been identified and so corrective action may be attempted earlier which is less radical than insulin injection (Harrison, 1995).

Until recently, hearing loss was a condition in the first category. The most prominent clinical feature of hearing disability is the subtlety of its onset and this has led to frequent semantic difficulties. For example, the field of hearing conservation has for years used the term "*hearing damage*" which at best is an ambiguous juxtaposition of anatomical fact: *ear damage* and its behavioural outcome, *hearing disability*. In turn

this has led to conceptual difficulties. The most central of these is an implicit assumption that, since the ear is normally extraordinarily sensitive to sound, then it must also be sensitive to its own state of damage.

The Western industrialised nations now expend very considerable resources on Hearing Conservation Programs (HCP) to try to limit the multiple costs of hearing loss (Dobie, 1995). Axiomatic within such programs is the notion that catching the first tiny shift in hearing threshold is the most sensitive early warning measure possible. While most hearing disability occurs in the mid frequency ranges, the first signs of Age Dependent Hearing Loss (ADHL) or presbycusis occurs at the highest frequencies, while Noise Induced Hearing Loss (NIHL) first presents with a notch at 4 or 6 kHz, (Monley et al., 1996; Pelousa, et al., 1995), the latter for more impact exposure. High frequency audiometry is currently receiving a resurgence of interest in the hope of issuing a warning before the hearing loss progresses to the speech frequencies where most disability occurs, (Feghali and Bernstein, 1991).

A concern of the NAL hearing loss prevention research program has been that one cannot really use the same phenomenon, even the same parameter (hearing threshold), which signals the onset of a condition to also provide early warning. In the case of diabetes, some entirely different measure has provided a better precursor than blood glucose levels — the level of antibodies which lead to islet cell destruction. This discovery has shown that rising blood glucose levels provide no warning at all and indicate an advanced stage of the condition.

An advanced precursor for hearing loss

Back in the late 1960s it was believed that the mammalian ear was a simple transducer and bending of the hairs on the tops of the hair cells in the cochlea sent sound information to the brain. Until recently hearing conservation has been based upon the simple notion that if sound is too loud, the hairs will break off. During the 1970s this simple idea was drawn into question and controversy raged. By 1980 a London Congress finally agreed that our ears responded actively to the sound entering. At that meeting two key pieces of information clinched the argument. The first was that the electromechanical interaction with sound, itself produced a byproduct sound, a much softer response, which could be measured with a microphone in the ear canal (Kemp, 1978; Kemp & Chum, 1980). Kemp called this an *otoacoustic emission*. The second was a long-awaited confirmation that the behaviour of this mechanism appeared physiologically vulnerable, nonlinear and compressive in the direction that would account for the 120dB dynamic range of the ear (LePage and Johnstone, 1980).

Since 1980 the measurement of otoacoustic emissions has been commercialised and tested widely. There are currently three manufacturers of distortion product equipment and one for the original, click-evoked method. Over 1000 scientific presentations and articles have since revealed the basic characteristics of otoacoustic emissions. The practice of audiology has taken an abrupt right turn with the realisation that most sensorineural hearing loss (particularly NIHL) occurs be-

cause of progressive damage and loss of the active cells, the *outer hair cells* (OHC) and that this leads to a decline in the size of the otoacoustic emission. Moreover this decline can be monitored from the very earliest stages of ear damage, even from birth.

The first application for the technique was to the early detection of hearing problems in neonates and infants who are unable to respond voluntarily to behavioural tests (Bray and Kemp, 1987; Kemp and Ryan, 1991). The United States National Institutes of Health have since published a consensus statement (National Institutes of Health, 1993) recommending that all newborns be screened with the technique as a *first* measure. Since neonates possess a complete set of outer hair cells, the emission is typically very large (over 30 dB SPL) and it is relatively easy to decide if it is "present" or "absent". The emission may still be "absent" in a newborn after amniotic fluid has dispersed, because of maternal infection or birth trauma or a variety of developmental defects, in which case the infant should receive early intervention care and educational help. At NAL, it rapidly became obvious that click-evoked emissions were not simply "all or nothing" but varied in amplitude over a 50 dB range and were very reproducible in the short term for any ear on test/retest, and they declined steadily with age.

We have now acquired click-evoked otoacoustic emission data in some 2500 children and adults, both normal and those with all stages of disability. Since nearly all of the hearing loss cases (ADHL and NIHL) fall within the bottom 20 percent of the range of emission strengths, we found that there was a critical value of emission strength at which the risk for hearing loss rose steeply. Accordingly, we conceived that the use of the technique could pave the way from *hearing conservation* in the first category (no precursors) to *hearing loss prevention*

in the second category (otoacoustic emissions may provide many years of warning).

To illustrate the difference we compare attempting to carry out effective hearing conservation programs using pure tone audiometry with looking through a window with the shade lowered near the bottom, limiting the view (Figure 1A). The addition of click evoked otoacoustic emissions, combined with our index of emission strength (Coherent Emission Strength, CES dB SPL), is like raising the shade so that the full ageing picture of the decline of ear function can be tracked from birth (Figure 1B). Before the use of otoacoustic emissions the rise in ear damage with the consequent decline in emission strength was outside our field of view. Since lifestyle factors are well recognised to play an important part in our hearing health, this new capability is set to revolutionise the kinds of intervention which can now be undertaken during the childhood period where the age curve declines most rapidly (LePage, 1994). The decline in CES is normally slow, about 3 dB/decade offering many years in which to control the factors which accelerate hearing loss.

Basis of the precursor

Remarkably, in the cases of both diabetes (IDDM) and hearing loss, the condition results from a decline in the performance of certain cells vital for normal function. In both cases the decline in performance in the long term is due to decline in numbers of these cells: in the case of the ear, the outer hair cells (Ryan and Dallos, 1975); in the case of sugar utilisation, the islet beta cells of the pancreas (Harrison et al., 1990). The time course of the two phenomena are remarkably similar. While compensation mechanisms may give the perception that the degree of disability wavers, the underlying accumulated damage is permanent. It appears that in both cases normal function may be maintained

while only a fraction (about one fifth to one tenth) of the original numbers of the cells at birth remain.

We have come to term this ageing characteristic (Figure 1A) the "fuel tank" model. The contents of the tank decline steadily throughout life and for any individual may go through periods of more rapid decline due to the factors which produce "accelerated ageing". As the tank approaches empty, the symptoms which begin to present go through various stages. At NAL we have obtained a fairly definitive value for the critical value of emission strength (LePage et al., 1994) below which people begin reporting symptoms of at least a mild hearing loss. This value, at which the tank warning light comes on is about -2dB SPL. Some time prior to this stage, people may even be suffering acute symptoms of trauma to loud noise exposure, the most notable of which is tinnitus which presents in many ways (LePage, 1995).

Using otoacoustic emissions therefore, it is possible to extend the estimation of a person's hearing status to level of risk for a hearing loss, subject to some caveats. One concern is the transmission of the sound energy forwards and backwards through the middle ear. If the measured emission strength is found to be high this tends to rule out transmission problems through the middle ear. On the other hand if the result is low then tympanometry should routinely be performed at the time (Sutton et al, 1996) of the test to determine if the low result can be due to fluid or fixation of the ossicles. With the exception of problems of incomplete development of the middle ear, these conditions are transient. In addition, the existence of a pressure differential across the ear drum has been tested — it gives up to a 6 dB reduction in emission strength for ± 200 mm water pressure in the external canal. We may safely assume therefore that over any period of repeated measure-

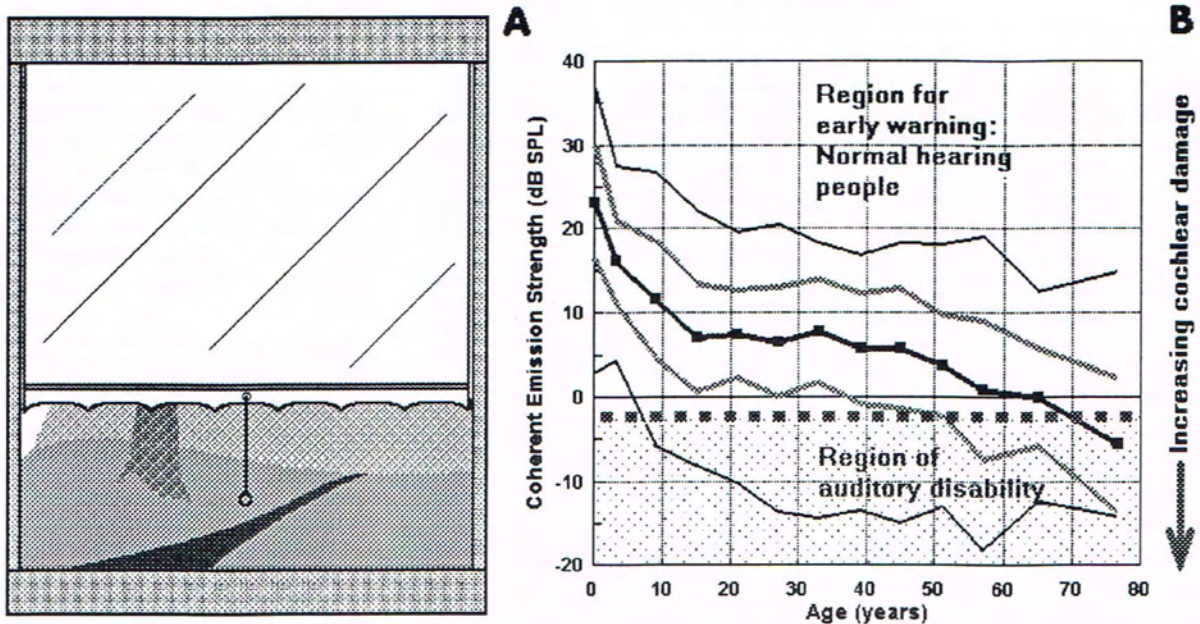


Figure 1 represents schematically the change which otoacoustic emissions have brought to audiometry. Our data have mostly been obtained with the Otodynamics Analyser in standard screening mode, using the "nonlinear" click-stimulus paradigm. Figure 1B (right) shows the change in emission values with age in the Australian population. The central (heavy line with the squares) is the mean value for each 6 year span in the Australian population. The two flanking lines are mean \pm one standard deviation (containing 70 percent of the population) while the thin top and bottom lines represent the maximum and minimum values. Importantly there is a critical value of emission strength (dashed line) below which is a shaded region which contains most incidences of hearing problems. Above the line is the clear region during which the preclinical values of emission strength may be determined. Figure 1A (left) shows a window scene with the window shade drawn revealing only a part view. By way of analogy click evoked otoacoustic emissions have raised the shade so that the full extent of ear performance may be appreciated. The full view has far reaching consequences because it offers early warning.

ments the highest value may be taken to represent the best estimate of net activity of the ensemble of outer hair cells. The curves of Figure 1B have been prepared subject to this assumption, while all known forms of abnormality (excepting noise overexposure) have been eliminated.

A remarkable feature of Figure 1B is the wide spread of otoacoustic emission (CES) values at every age. Indeed, at any age the spread of emission strength values is almost 80 percent of the total range for all ages. This spread may account for why some investigators concluded there was no age-dependence on click-evoked emissions (e.g. Stover and Norton, 1993). Since observing the age-dependent effects in our cohort, it is clear that many factors contribute to outer hair cell destruction and that many young people already have extensive preclinical damage. Attempts have been made to factor out the possibility that ear

maturation accounts for the rapid decline in the first decade. Some dependency upon ear canal size may exist to age 8, but at least these results are presented for constant screening test conditions, in particular the clicks are presented at a pre-set value of 80 dB SPL which equates to about 50 dB hearing level. Since the outer-hair-cell/medial-efferent-system is effectively mature at birth or soon after, the rapid decline in the first decade may not be due to maturation and so it requires some additional explanation.

An illustration of the need for a precursor other than hearing loss

The high incidence of low values in the first two decades particularly emphasises our basic assertion: *it is invalid to take baseline audiometry as an adequate measure of ear damage*. This may be illustrated by way

of a hypothetical example which has factual basis in our records. Take three people who have audiograms within normal limits. The first one is a female in her late teens who has been socially active and attended many concerts and discos and who regularly listens to her headset radio while jogging. A second person is a female who has had a quieter life, living in a country town. The third person is a male who has not lived a particularly noisy existence but who had had a continuous series of middle ear infections before he was six years of age. His infections were treated with the fitting of T-tubes (grommets) and with several courses of various antibiotics. All three people present for work at Company X and baseline audiograms are taken. The working conditions are identical but the male starts to have problems and an audiogram a year later reveals a mild hearing loss. At the same time the first female is tested and her hearing is normal, with perhaps a

slight drop at 4 kHz. The second female's hearing remains normal. After five years the male has a compensable hearing loss while the females have a slight high frequency loss and a normal audiogram respectively. Both cases involving hearing loss are deemed to be work-related since baseline audiograms were normal. In retrospect the male was designated as having more susceptible ears because a lower dose of noise "produced" the hearing loss and Company X was deemed liable to pay compensation. In these cases the situation would appear completely different had all three had baseline otoacoustic emission screens. It would have been readily apparent at the beginning of employment that the male had lower level cochlear emissions and that stronger, more focussed measures could then have been taken to protect his hearing.

Such a hypothetical example illustrates well why HCPs based only upon monitoring sound levels and behavioural measures may characterise the size of the problem of noise-induced hearing loss, but may not be effective in reducing its incidence (Hetu et al, 1990). Indeed, the monitoring of workers' pure tone hearing thresholds has been singled out as particularly ineffective and therefore should be abandoned (Noble, 1978). Otoacoustic emissions explain why this is so. The essential difference offered by otoacoustic emissions is that the approach can indeed move from *hearing conservation* of the current state which may include hearing loss, to actual *hearing loss prevention*, or at least knowledgeably delaying the onset of symptoms by years.

Implications for the future

The advent of otoacoustic emissions as an early warning approach raises a whole new set of possibilities. There will always be people who are more susceptible to the fac-

tors causing hearing loss. It has been shown that if the decline in emission strength could be limited to an average of less than 3 dB per decade (Murray et al., 1994), an individual need never suffer hearing loss during his or her lifetime. With a birth value of emission strength at 24 dB SPL on a linear decline basis, that would afford 8 decades before the critical value is reached. Such an outcome is not hypothetical. Evidently there have been communities which have managed to avoid hearing loss to a great age (Rosen, et al., 1962), a phenomenon which at the time had no scientific explanation beyond the fact that this was a study of a low cardiovascular risk group. By contrast, we are tracking many individuals with much higher rates of decline, some teenagers as much as 30 dB per decade and who have already reached critical levels of preclinical ear damage (Murray et al., 1994). Ultimately, social entities, industries, governments and schools will take responsibility for situations which will accelerate ear damage, particularly in young people, so that the population which is living for longer may retain their hearing for longer.

This new approach has profound consequences not only for exercising greater control over NIHL, but hearing loss due to all causes. Other contributory factors may be much more significant than previously thought in influencing an individual's susceptibility to NIHL. These predisposing factors seldom are revealed in baseline audiometry but using otoacoustic emissions their role has been shown to be very important. In the hypothetical example above, the risk of significant latent damage in children due to use of antibiotics is now more easily appreciated, despite the fact that the damage has been documented previously but only after hearing loss has occurred. Aminoglycoside administration is well known to cause massive outer hair cell destruction in the basal turns, amounting to an estimated 1000 dB/decade decline

in emission strength. More commonly used erythromycin has been determined to be ototoxic (Brummett, 1993), and to produce destruction equivalent to rapid ageing of the cochlea. Recent studies using otoacoustic emissions have now begun to document *preclinical* changes in otoacoustic emissions due to streptomycin use (Furst et al., 1995), while another recent study looking at Ampicillin has found little effect on click evoked emissions (Zorowka et al., 1993). The doubt surfaces, however, that some "childhood" antibiotics may be equally ototoxic. It certainly fortifies a more general cautionary approach to the widespread use of antibiotics, and encourages the introduction of otoacoustic emissions to monitoring toxic side effects whenever multiple courses are indicated. More generally, many drug trials are still being published in which hearing loss is the primary indicator of ototoxicity. Many "alternate" medical treatments and diets available (of which the liqueur Absynthe is an historical example, causing the painter Van Gogh extreme tinnitus) could be causing extensive latent OHC damage which, many years later are attributed to other causes, such as heredity.

NAL Hearing Loss Prevention Research is therefore aimed at determining the sensitivity and specificity with which otoacoustic emissions might be incorporated into hearing conservation programs. These parameters require long-term monitoring of a cohort of individuals to show that the otoacoustic emission approach is a valid predictor. We are well advanced in such a study. (see Longitudinal study of permanent shifts in otoacoustic emissions)

The "fuel tank" concept carries an important consequence in respect of industrial HCPs. It implies that *the need to take into account individual risk* is at least as an important factor as *sound level* in limiting hearing disability. We therefore

strongly advocate a two-pronged approach 1) conventional reduction of sound levels taking care of exposure of the majority of most (non-susceptible) individuals, and 2) individual screening with the cheaper early warning methods which may be far more cost effective and alert those most susceptible before hearing loss occurs.

The Australian measure of hearing protector attenuation, the SLC₈₀ attenuation rating figure was based upon the notion of protecting 80 (actually 84) percent of the population. We are now questioning whether in fact, the remaining 16 percent, not "protected" from noise by hearing protectors, remain the group most at risk by virtue of their basic susceptibility. If this were true, it would help account for why it is difficult to show that the wearing of hearing protectors reduces the incidence of hearing loss (Dobie, 1995). With further refinement otoacoustic emissions should be able to shed some more light on these complex issues.

In summary, the potential for click-evoked otoacoustic emissions to provide early warning allows the possibility that there is a significant transition towards delaying of the onset of hearing loss, not just for NIHL but for all the other causes synergistically contributing to ear damage (McFadden et al., 1995). Once the otoacoustic emission screening test becomes as common as the intra ocular pressure test for glaucoma, it may begin to have a larger impact on the incidence of premature hearing loss. As a tool for education in our hands it has had a significant impact on individual awareness of the processes of ageing of the ear and indeed upon the difficulties in dealing with negative attitudes previously exhibited toward HCPs (Hetu, et al., 1994). It is an assuring thought that the resulting increase in hearing awareness could lead to a general increase in hearing appreciation, so that future hearing conservation programs may

begin to focus more upon the positive aspect — *conserving normal hearing* for its advantages rather than merely conserving what remains after irreparable damage is done.

References

- Bray, P. and Kemp, D., (1987). An advanced cochlear echo technique suitable for infant screening. *Brit. J. Audiol.*, 21, 191-204.
- Brummett, R. E., (1993). Ototoxic liability of erythromycin and analogues. *Otolaryngol-Clin-North-Am.* Oct, 26, 811-9.
- Dobie, R. A., (1995). Prevention of noise-induced hearing loss. *Archives of Otolaryngology Head & Neck Surgery*, 121, 385-391.
- Feghali, J. G. and Bernstein, R. S., (1991). A new approach to serial monitoring of ultra-high frequency hearing. *Laryngoscope*, 191, 825-829.
- Furst, G., Maurer, J., and Schlegel, J., (1995). [Monitoring ototoxic side effects in streptomycin therapy of tuberculosis patients with transitory evoked otoacoustic emissions TEOAE] *Pneumologie*. 49, 590-5.
- Harrison, L. C., (1995). Antigen-specific therapy for autoimmune disease: prospects for prevention of insulin-dependent diabetes. *Molecular Medicine*, 1(7), 722-727.
- Harrison, L. C., Campbell, I. L., Colman, P. G., Chosich, N., Kay, T. W. H., Tait, B. D., Bartholomeusz, R. K., Aizpurua, H. J., Joseph, J. L., Chu, S., and Kielczynski, W. E., (1990). Type 1 Diabetes: *Immunopathology and Immunotherapy Adv. Endocrinol. Metab.*, 1, 35-94.
- Hetu, R., (1979). Critical analysis of the effectiveness of secondary prevention of occupational hearing loss. *J. Occupational Medicine*, 21, 251-254.
- Hetu, R., (1994). Mismatches between auditory demands and capacities in the industrial work environment. *Audiology (Basel)*, 33, 1-14.
- Hetu, R., Getty, L., and Waridel, S., (1994). Attitudes towards co-workers affected by occupational hearing loss. II: Focus groups interviews. *Br-J-Audiol.* 28, 313-25.
- Hetu, R., Tran Quoc, H., and Duguay, P., (1990). The likelihood of detecting a significant hearing threshold shift among noise exposed workers subjected to annual audiometric testing. *Annals of Occupational Hygiene*, 34, 361-370.
- Kemp, D. T. (1978). Stimulated acoustic emissions from within the human auditory system. *J.Acoust.Soc.Am.*, 64, 1386-1391.
- Kemp, D. T. and Chum, R. (1980). Properties of the generator of stimulated acoustic emissions. *Hear. Res.*, 2, 213-232.
- Kemp, D. T. and Ryan, S., (1991). Otoacoustic emission tests in Neonatal screening programmes. *Acta Otolaryngol. (Stockh); Suppl.*, 482, 73-84.
- LePage, E. L., (1992). Hysteresis in cochlear mechanics and a model for variability in noise-induced hearing loss. In *Noise-Induced Hearing Loss* edited by A. Dancer, D. Henderson R. J. Salvi and R. P. Hamernik. *Mosby Year Book*, St.Louis. 10, 106-115.
- LePage, E. L., (1994). 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. In: *Better Hearing Australia Conference*, Adelaide, South Australia, 7-11 August, 1994.
- LePage, E. L., (1995). A model for cochlear origin of subjective tinnitus: excitatory drift in the operating point of inner hair cells In *Mechanisms of Tinnitus* edited by Jack A. Vernon and Aage R. Moller. *Allyn and Bacon*, Boston. Chapter 11, 115-148.
- LePage, E. L. and Johnstone, B. M., (1980). Nonlinear mechanical behaviour of the basilar membrane in the basal turn of the guinea pig cochlea. *Hear. Res.*, 2, 183-189.
- LePage, E. L., Murray, N. M., and Tran, K., (1994). Comparison of otoacoustic emission measures of cochlear damage in the Australian population with hearing loss in the Australian and British populations. In: *Better Hearing Australia Conference*, Adelaide, South Australia, 7-11 August, 1994.
- McFadden, D., Plattsmier, N. S., and Pasanen, E. G., (1995). Temporary hearing loss induced by combination of intense sounds and nonsteroidal

anti-inflammatory drugs. *Am. J. Otolaryngol.*, 5, 235-241.

Monley, P., West, A., Guzeleva, D., Dinh, D. A., and Tzvetkova, J., (1996). Hearing impairment in the Western Australian noise exposed population. 12th National Conference of the Audiological Society of Australia, Brisbane, 29th April, 1996 to May 2nd 1996, Audiological Society of Australia, p51

Murray, N. M., LePage, E. L., and Tran, K., (1994). Ageing characteristics of the Australian population in terms of otoacoustic emission strengths: global and individual picture. In: Better Hearing Australia Conference., Adelaide, South Australia, 7-11 August, 1994.

National Institutes of Health, (1993). NIH Consensus Statement: Early identification of hearing impairment in infants and young children. NIH Consensus Statement. US Dept. of Health & Human Services, Bethesda, MD, Vol. 11(1): 1-24.

Noble, W., (1978). Monitoring Audiometry: Protection for Whom? Occupational Hearing Loss: Conservation and Compensation, edited by Waugh, R. and Macrae, J., Sydney, 1978, Australian Acoustical Society, 164-179

Pelousa, E. O., Abel, S. M., Simard, J., and Dempsey, I., (1995). Prevention of noise-induced hearing loss in the Canadian military. *Journal of Otolaryngology*, 24, 271-280.

Rosen, S., Bergman, M., Plester, D., El-Mofty, A., and Satti, M. H., (1962). Presbycusis study of a relatively noise-free population in the Sudan *Ann. Otolaryng.*, 71, 727-743.

Ryan, A. and Dallos, P., (1975). Effect of absence of cochlear outer hair cells on behavioural auditory threshold *Nature*, 253, 44-46.

Stover, L. J. and Norton, S. J., (1993). Boys Town Natl. Res. Hosp., 555 N. 30th St., Omaha, NE 68131, USA The effects of aging on otoacoustic emissions. *J. Acoust. Soc. Am.*, 94(5), 2670-2690.

Sutton, G.J., Gleadle, P., and Rowe, S.J., (1996). Tympanometry and otoacoustic emissions in a cohort of special care neonates *Br. J. Audiol.*, 30, 9-17.

Zorowka, P., Schmitt, H. J., Eckel, H. E., Lippert, K. L., Schonberger, W., and

Merz, E., (1993). Serial measurements of transient evoked otoacoustic emissions (TEOAEs) in healthy newborns and in newborns with perinatal infection. *Int-J-Pediatr-Otorhinolaryngol.* Oct, 27, 245-54.



Canadian Pianist Jon Kimura Parker has toured for the ABC three times since 1990, each time playing with ABC orchestras around Australia. The concerts just presented at the Sydney Opera House were "standing room only", such is his mastery of the classical repertoire topped off with his "liquid jazz" encores. Maybe his local popularity says something about Australians loving his more relaxed style, while his own homepage which comes complete with his e-mail address says something about his accessibility. He willingly "lent" his ears for our NAL cohort study of musicians, to add to our small number of percussion players. His were high level emissions indicating no hearing problems, but they

nevertheless showed tell-tale patches of accelerated ageing. The early report on the status of his ears was much appreciated.

"I often tell young musicians that their most important asset in making music is not their fingers, or their instruments, but their ears. Safeguarding one's hearing is not just a good idea, it's crucial to the longevity of anyone's enjoyment of music and sound. And you can always spot a musician in New York: they're the ones standing on the subway platform with their fingers in their ears every time a train whizzes past!" Regards, Jackie Parker

(quoted with permission)

Prevention of Hearing Loss

Longitudinal study of permanent shifts in otoacoustic emissions.

Investigators: Narelle Murray, Eric LePage

Background: For some six years now as an adjunct to a study of otoacoustic emissions in a normative Australian population, 50 males and females from this study have been followed up annually with pure tone audiometry and otoacoustic emission testing. Having derived the parameter, Coherent Emission Strength, which may be used to estimate the risk factor for hearing loss, and applied this to the population as a whole, the reasons for and the extent to which this parameter changes in individual ears has to be determined.

Research Questions: The questions remain as to normal test-retest reliability; the interpretation of changes individual ear activity; whether the accumulation of ear damage is systematic or not, and to what it could be related; and, what is a useful definition of "susceptibility" to hearing loss.

Research Procedures: Analysis of Variance of Repeated Measures was carried out on the preliminary study of otoacoustic emission measures on 12 Subjects to determine test-retest variability. This was undertaken on a spectral analysis covering 1) all frequencies in total, 2) 1 kHz frequency bands and 3) on the derived parameter Coherent Emission Strength. Analysis of the click-evoked otoacoustic emission ear assessment as well as the pure tone audiometry of the 50

males and females in the Cohort Study is continuing.

Findings: Although the small amplitude click-evoked otoacoustic emissions exhibit considerable intersubject variability it was found that there was excellent intrasubject test-retest repeatability of both the emission spectrum and the Coherent Emission Strength parameter. Calculated Standard Error of Means values of 1.02 (both ears) would indicate that on retesting, a deviation of greater than 2.04 dB (2 SEMs) from a subject's original emission spectrum would be expected in only 5% of cases and could be taken to represent a real change in the measured emission. Similarly it would appear that any change in Coherent Emission Strength >1.5 dB would also represent a real change in cochlear activity.

Significance: In clinical practice, it should be noted that variability may depend on maintaining test conditions, such as the use of the same probe and the same software. However, the high test-retest reliability of click-evoked otoacoustic emissions may now mean that damage to the cochlea over time can be estimated either in terms of decline in spectral emission or in Coherent Emission Strength, on the proviso that possible changes in middle ear transmission is ruled out. CES as a simply derived single-number parameter does, in particular, appear to be a viable method of assessing coherent activity in the cochlea. It has lead to what we have dubbed the "fuel tank" model of ear ageing in which the first signs of hearing loss do not appear until the tank is nearly empty. Not just noise contributes to the decline, but many other factors throughout life. These conclusions will now be applied to the results of the Cohort Study.

Longitudinal study of hearing of orchestral musicians.

Investigators: Narelle Murray, Eric LePage, with Ken Mikl (WorkCover Authority, NSW)

Background: In 1995 noise measurements in the pit of the Sydney Opera House, audiometric monitoring and otoacoustic emission assessments for members of the Australian Opera and Ballet Orchestra were again carried out. In all, since 1992, 115 members of the orchestra have been tested, with 17 having been tested on all four occasions.

Research Questions: To compare otoacoustic emission test data for any significant changes which may have occurred over the four year test period a) on an individual basis, b) for each orchestral section and, c) for the whole orchestra.

Research Procedures: In 1995 click-evoked otoacoustic emission screening tests were carried out in the same manner as previous years. Questionnaires regarding past aural and noise exposure history were again completed and pure tone audiometric results obtained from testers of the WorkCover Authority on the same day.

Findings: A wide variation was found in individual ears across the orchestra. For example (Fig. 1) shows in Panel A results for a String player and Panel B for a Brass player. It should be noted that in both cases the spectra for 1995 are lower than those for 1992. If it is accepted that a difference over time between two emission spectra is >2dB, or in CES values >1.5dB, is

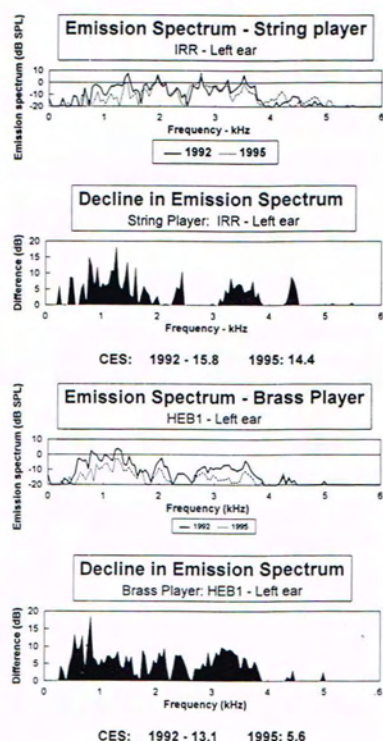


Figure 1. A. Top panel: Emission Spectrum of a String Player in the Orchestra; bottom panel: decline in the emission spectrum from 1992 to 1995. B. Top panel: Emission Spectrum of a Brass Player in the Orchestra; bottom panel: decline in the emission spectrum from 1992 to 1995.

significant, then it would appear that there is no decline in outer hair cell activity over the 4 years in the left ear of the String player but a significant decline in outer hair cell activity in the left ear of the Brass player. However, on a frequency specific basis there is a significant decline in emission level of the string player's left ear below 2kHz.

As in individual data, the String section shows a much smaller difference between the 1992 and 1995 results than the Brass section; indeed the Strings show a slight improvement (Figure 2. - String and Brass Sections). This is because a number of older members of the String Section of the Orchestra tested in 1992 had left and been replaced by young new members in 1994 and 1995 and this may have given rise to the higher emission levels. The same did not happen in the Brass section, with the majority of that Section being tested in both 1992 and 1995. Utilising the measure of two Standard Error of Means (SEM), it can be seen (Figure 3) that with the exception of the String Section, the other sections of the Orchestra have greater variation than the SEM calculated for the

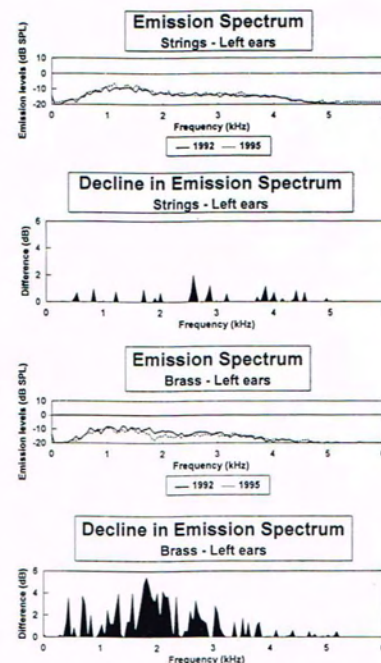


Figure 2. Emission Spectra for both the String and Brass sections of the Orchestra, together with the decline in those spectra between 1992 and 1995.

“baseline” reliability study (solid horizontal line), as discussed for the cohort study. Although it is noticeable that in most cases the SEMs for 1995 are greater than those for 1992 the only Section where the difference is significantly higher is the Percussion Section.

Significance: The results continue to indicate that it is possible to show significant declines in ear damage with otoacoustic emissions over a period of four years.

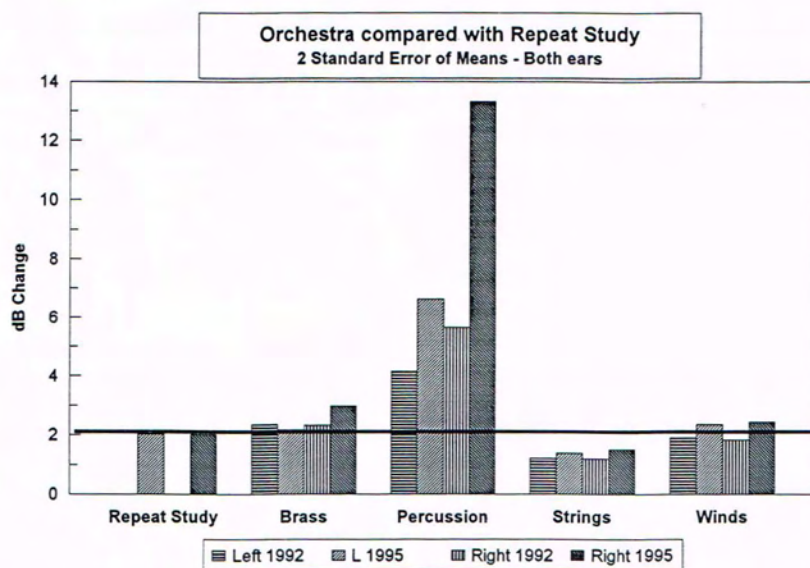


Figure 3. Changes occurring from 1992 to 1995 in each section of the Orchestra in terms of Standard Error of Means compared with a Reliability Study carried out to determine a level of significant change in emission strength over time.

Role of efferent control loops: Selective attention in hearing. An otoacoustic analog and its potential role in hearing loss prevention.

Investigators: Patricia T. Michie*, Eric LePage, Nadia Solowij*, Michael Haller* and Lyn Terry* (*Macquarie University)

Background: The interest in selective attention or sensory focussing in hearing stems from considering the most common symptom of hearing loss reported to audiologists at first contact with any client. Typically, a client will describe how they can hear satisfactorily if the background noise is low. However, once the background noise level rises they are unable to cope with attending to another's voice. A middle-aged male will typically describe how, in a restaurant, he will be struggling to have a conversation with the wife while the wife will be able to pick out and repeat parts of conversations from each table. Often such abilities of the wife may be erroneously attributed to additional expertise which females possess or have acquired due to their gender. Females are, in fact, no less prone to difficulties in understanding speech in background noise. The essential difference has little to do with gender traits, but more to do with the degree of accumulated ear damage, specifically the hair cells of the inner ear. Females and males may exhibit the same range of ear damage, although on average, female ears exhibit less damage than male ears. Male damage accumulates to critical levels ten

years earlier than does female ear damage.

Research Questions: The research psychological literature has for many years recorded a clash of opinion as to whether selective attention is exclusively mediated by central processing in the brain, maybe even at the cortical level, or whether it also necessarily involves the peripheral sense organ in question (Lukas, 1981; Hirshhorn and Michie, 1990). The literature refers not just to the sense of hearing but all senses, since one can, for example, selectively attend to visual stimulation while ignoring hearing, taste, touch, heat and cold, and pain (Driver and Mattingley, 1995). Due to human cross-modal selection abilities there are strong reasons to believe that the basic mechanisms of selection are central. On the other hand the fact that selection in hearing breaks down with damage to the ear suggests that the within-mode selection mechanism may involve the outer hair cells (OHC) (Avan and Bonfils, 1991; Froelich et al, 1993; Kawase, 1993). This suggestion is strongly fortified by considering the way the nerves innervate the ear. There are important tracts of fibres (the medial efferent system) which connect control signals from the brain stem to the OHC. The OHC primary mode of action is controlling sensitivity at specific frequencies throughout the hearing range and several lines of evidence suggest that the efferent tract may be "wired" specifically to facilitate heightened sensitivity at specific frequencies, which can be chosen within the brain stem for optimum signal to noise ratio (LePage, 1989). Moreover, they may further have a specific role in allowing a person to choose the level of lateral suppression of non-meaningful frequencies (Kawase et al., 1993).

Research Procedures and Findings: The project was conceived in several stages and ARC funding of \$80000 was obtained for the first series of experiments. Six

groups of experiments were run in the current series, five different paradigms plus a repeat of the first. In each series 12 subjects were used. Each subject was presented with a series of one of two brief tone pips and the otoacoustic emissions for each trial were averaged and stored for comparison. In the first series of experiments the results were characterised in terms of the strength of the emission in the neighborhood of the two frequencies. All trials were randomised and counterbalanced. In each of the five experiments the conditions of the stimuli were varied, e.g. the difficulty of the identification task varied, or the pairs of stimuli delivered to the same ear or opposite ears. In the first series of experiments the noise level was low and the results were characterised as "on-frequency" responses. The outcome of the first series was, remarkably, no significant effect, to be compared with a small (1dB) effect of attention when switching between the visual and auditory modes seen by Puel et al. 1988; Meric and Collet, 1992; Giard et al., 1994. The first series of experiments actually revealed some significant effects, but not in the directions for which the experiment was designed. The results have been submitted, revised and accepted and will shortly appear in *Hearing Research* (Michie et al., 1996).

Since the set of experiments were conducted, the importance of crossmodal coordination in selective attention has been highlighted (Driver, 1996), in particular in relation to understanding speech with visual cues (lip-reading). Ours were a preliminary series of experiments. Further experimental series in the future may investigate the role of varying background noise to simulate hearing loss and tracking tasks to simulate more realistic listening conditions. The outcome of the project nevertheless presents a paradox. The outer hair cells play a vital role in determining "non-voluntary" selection, i.e. the selectivity

exhibited by single auditory nerve fibres and this tuning characteristic is also reflected in the tuning of the medial efferent neurons. Some results not presented in the paper actually suggest a possible resolution, the voluntary selection task is influencing the frequency mapping of the cochlea (LePage, 1990; Allen, 1996) in such a way that the expected attentional effect is minimised (Michie et al, 1996).

Significance: As we have shown, the performance of the OHC may be measured with otoacoustic emissions. They have proven to be a very sensitive overall measure of ear damage. By determining if the otoacoustic emissions change with voluntary focussing of attention one might develop a test for listening to two test sounds which could shed important light on the role of the OHC in this normal hearing process. If click-evoked otoacoustic emissions could show specific changes with attention, then repeated measurement over time may yield a sensitive objective rapid test for the decline in the capacity of the normal hearing ear to process signals in noise, e.g. speech under noisy conditions. Such basic information could be very useful in two major ways. Firstly, the sensitive test could be much more potent for use in prevention programs for monitoring the decline in normal ear processing power. Secondly, it could suggest possible future strategies for hearing aid design where the object is to process speech so as to improve the signal to noise ratio.

References:

- Allen, J.B. (1996). OHCs shift the excitation pattern via BM tension. In: *Diversity in Auditory Mechanics*, Edited by Lewis, Long, Narins and Steele, Berkeley California, June 24-28, 1996, 1-8.
- Avan P. and Bonfils P., (1991). Analysis of possible interactions of an attentional task with cochlear micromechanics. *Hear. Res.*, 57, 269-275.
- Driver, J., (1996). Enhancement of selective listening by illusory mislocation

of speech sounds due to lip-reading. *Nature* (London), 381, 66-68.

Driver, J. and Mattingley, J. B., (1995). Selective attention in humans: normality and pathology. *Curr-Opin-Neurobiol.* 5, 191-197.

Froehlich, P., Collet, L., and Morgon A., (1993). Transiently evoked otoacoustic emissions amplitudes change with changes of directed attention. *Phys. & Behav.*, 53, 679-682.

Giard, M. H., Collet L., Bouchet P., and Pernier J., (1994). Auditory selective attention in the human cochlea. *Brain Res.*, 633, 353-356.

Hirschhorn, T. N. and Michie, P. T., (1990). Brainstem auditory evoked potentials (BAEPs) and selective attention revisited. *Psychophysiology*, 27, 495-512.

Kawase, T., Delgutte, B., and Liberman, M. C., (1993). Antimasking effects of the olivocochlear reflex. II. Enhancement of auditory-nerve response to masked tones. *J. Neurophysiol.*, 70, 2533-2549.

LePage, E. L., (1989). Functional role of the olivocochlear bundle: A motor unit control system in the mammalian cochlea. *Hear. Res.*, 38, 177-198.

LePage, E. L., (1990). Helmholtz revisited: direct mechanical data suggest a physical model for dynamic control of mapping frequency to place along the cochlear partition. In *The Mechanics and Biophysics of Hearing*, edited by P. Dallos, C. D. Geisler J. W. Matthews M. A. Ruggero and C. R. Steele, Springer-Verlag, 278-287.

Lukas, J. H., (1981). The role of efferent inhibition in human auditory attention: An examination of the auditory brainstem potentials. *J. Neurosci.*, 12, 137-145.

Michie, P. T., LePage, E. L., Solowij, N., Haller, M., and Terry, L., (1996). Evoked otoacoustic emissions and auditory selective attention. *Hear. Res.*, (in press).

Meric, C. and Collet, L., (1992). Visual attention and evoked otoacoustic emissions: a slight but real effect. *J. Psychophysiology*, 12, 233-235.

Puel, J. L., Bonfils, P., and Pujol, R., (1988). Selective attention modifies the active micromechanical properties of the cochlea. *Brain Res.*, 447, 380-383.

Temporary shifts in otoacoustic emissions

Investigators: Eric LePage, Narelle Murray, Khanh Tran, Michael Harrap (Department of Aerospace and Mechanical Engineering, Australian Defence Force Academy, Canberra)

Background: Hearing loss is often classified as temporary or permanent. A sound exposure which produces a temporary threshold shift (TTS) produces a series of changes occurring within the cochlea which may be broadly classed as self protection compensation. The actual mechanism responsible has not been determined in detail, but certain experiments have shown that substantial shifts in the position of the basilar membrane may occur in response to very loud tone stimulation (LePage, 1989) which have been regarded as significant (Zenner, 1993; Zenner et al., 1994). Since then very considerable evidence has accumulated concerning axial force generation by the outer hair cells particularly in regard to the slow force generation for frequencies up to 200 Hz. From experiments using brief intense tone bursts it would appear that exponential changes in the receptor potentials of inner hair cells display many similar temporal characteristics of hearing sensitivity. It is likely therefore that the decrease in sensitivity which is TTS may be due to sustained displacement of the cochlear partition so that the operating point of the IHC is displaced away from its point of maximum sensitivity. Contraction or elongation of the OHC is basically to shift the mapping of the cochlea to detune the mechanical filter and to result in dispersion of the energy as a defocussing manoeuvre (LePage, 1990; Allen 1996). The recovery time for TTS therefore depends upon the rate at which two maps return to full alignment. These

maps are the current (active) map and the map defined by the passive tapered properties. These maps need to be fully aligned for maximum cochlear sensitivity. This complex relationship together with considerable hysteresis (LePage, 1992) underlies the difficulties of extrapolating PTS from the amount of TTS in the past. However, in mechanical terms, the response of the cochlea to a broadband click stimulus will indicate, for each point along the cochlear partition, the current degree of warp of the map. The amplitude and latency of the reverse propagating wave will reflect the local stiffness of the partition. It follows that click-evoked otoacoustic emissions should not only reveal the place of the greatest change according to latency, but also the time course of the mechanical recovery. In turn the mechanical recovery can be categorised for a population of ears with determined histories and general OAE characteristics.

Research Questions: We have shown that a single figure index of emission strength for an ear, the Coherent Emission Strength (CES dB SPL) shows a decline for most of its range before the measured ear displays a permanent threshold shift (PTS). The main question is whether such a wide range of performance variation exists for temporary mechanical disturbance, the mechanical analog of TTS. What is the recovery relationship for otoacoustic emission following loud tone overexposure?

Using the power of the short term Fourier transform to describe a three-dimensional surface for a particular response, and therefore describing a snap-shot picture of the basilar membrane in a state of recovery, how does this surface change before, during exposure and during subsequent recovery. In particular, is it possible to demonstrate the mechanical equivalent of the half-octave shift. The obvious advantage of being able to arrive at a mechanical description of loud tone

disturbance is that this will immediately offer an avenue to rapidly determine a particular person's susceptibility — the longer the recovery, the more disturbance approaches a permanent state. The immediate and salient advantage of clicks over distortion products for this application is that the surface obtained describes the whole surface as at the same average period. Distortion products would need to scan the surface with discrete points, during which time the ear's biomechanical characteristics are expected to vary.

Research Procedures: In human studies on TTS conducted in the past very loud sound has been required to show any change in hearing threshold. In this project we have already shown that much less sound is necessary to evoke a biomechanical protective response from the ear. Subjects have been taken and exposed to only 80 dB tones for 1 minute as opposed to 105 dB for 10 minutes. The size of the biological response has varied considerably in the few subjects so far tested. The protocol requires monitoring the emissions at one minute intervals and, after five minutes exposing the ear to an 80 dB tone at 3 kHz for one minute, then monitoring the recovery in similar fashion for the next 20 to 30 minutes. Equipment is currently being setup to run the experiment on a short-faster time basis still. The emissions are plotted as previously, but also the time-frequency responses are also being compared. Some of these data were presented at the recent conference on Auditory Mechanics at the University of California, Berkeley (LePage et al, 1996).

Significance: Much basic research is carried out along the lines of "perturb the system, watch the response". This approach is useful up to a point, but unless the procedure is conceived in terms of generating a practical outcome there may be little or no real connection made with clinical objectives without still

more study. In this project, the objective is to lead to a practical understanding of how quickly any *human* ear responds to a challenge then recover from it without the fast time course of events being obscured by the slow, subjective response as in the past. This approach has already proven clinically beneficial within a five year period — we have previously used the click-evoked otoacoustic emissions to determine normative values for overall emission strength and this has led to the "fuel tank" model of ear ageing. Once we have established a procedure to watch a human ear's response to noise in real time the next step is to streamline the method of data collection and reduction and to obtain normative population statistics for clinical comparison. The approach is expected to give considerable insight into the response of the human ear to loud sound, whereas such information has only previously been available through animal experiment.

References:

- Allen, J.B. (1996). OHCs shift the excitation pattern via BM tension. In: *Diversity in Auditory Mechanics*, Edited by Lewis, Long, Narins and Steele, Berkeley, California, June 24-28, 1-8.
- LePage, E. L., (1989). Functional role of the olivocochlear bundle: A motor unit control system in the mammalian cochlea. *Hear. Res.*, 38, 177-198.
- LePage, E. L., (1990). Helmholtz revisited: direct mechanical data suggest a physical model for dynamic control of mapping frequency to place along the cochlear partition. In *The Mechanics and Biophysics of Hearing*, edited by P. Dallos, C. D. Geisler J. W. Matthews M. A. Ruggero and C. R. Steele, Springer-Verlag, 278-287.
- LePage, E. L., (1992). Hysteresis in cochlear mechanics and a model for variability in noise-induced hearing loss. In: *Noise-Induced Hearing Loss*. A. Dancer, D. Henderson R. J. Salvi and R. P. Hamernik, ed(s). *Mosby Year Book*, St. Louis, 10, 106-115.
- LePage, E. L., Murray, N.M., Tran, K. And Harrap M.J. (1996). Excitation pattern shifts in response to a pure tone:

time-frequency evidence from click-evoked otoacoustic emissions. Presented at conference on Diversity in Auditory Mechanics, Berkeley California, June 24-28, 1996.

Zenner, H. P., (1993). Possible roles of outer hair cell d.c. movements in the cochlea. *Br. J. Audiol.* 27(2), 73-7.

Zenner, H. P., Reuter, G., Zimmermann, U., Gitter, A. H., Fermin, C., and LePage, E. L., (1994). Transitory endolymph leakage induced hearing loss and tinnitus: depolarization, biphasic shortening and loss of electromotility of outer hair cells. *Eur. Arch. Otorhinolaryngol.* 251(3), 143-53.

Prevention of hearing loss in children

Investigator: John Macrae

In order to prioritise measures taken to reduce the incidence of hearing loss in children, it is necessary to know the prevalence of various causes of childhood deafness. The most recent statistics on causes of hearing loss in children under the age of 17 years provided with hearing aids by Australian Hearing Services show that, for 47% of the children, the cause of hearing loss is unknown, for 22% the cause is conductive disorder, for 12% the cause is familial, for 4% the cause is meningitis and that other causes are relatively infrequent, of the order of 1%. The cause of loss is, therefore, recorded as unknown for almost 50% of the children. This lack of knowledge hampers attempts to assign priorities to preventive measures. However, it can be shown that at least half of these losses of unknown cause are probably inherited.

About 50% of congenital sensorineural hearing losses are genetic in origin (Fraser, 1976) and, since it is likely that almost all of the hearing losses in the unknown cause category are congenital sensorineural hearing losses, at least 23% (0.50

x 0.465) of the losses in this category are probably of genetic origin. The findings of France and Stephens (1995) support this conclusion. After thorough investigation of 47 hearing losses of unknown cause, they found that 13 (28%) were genetic hearing losses. The AHS statistics identify about 12% of hearing losses as genetic in origin. The estimated total percentage of hearing losses of genetic origin is, therefore, at least 35%. Comparing this percentage with those for the other known causes of hearing loss, it is apparent that genetic disorder is probably the most common cause of hearing impairment in Australian children.

It follows that, from the point of view of prevention, high priority should be given to identification and elimination of genetic hearing loss. Adults who are affected by a hearing loss of genetic origin may wish to prevent their children from being similarly affected. Locations of mutant genes are known and genetic markers are already available for many forms of genetic hearing loss. Genetic markers can be used in the identification of carriers and in confirmation of a particular type of genetic loss and make the option of termination of pregnancy available in circumstances where the foetus is affected.

References

France E.A., & Stephens S.D.G. 1995. All Wales audiology and genetic service for hearing impaired young adults. *J. Audiol. Med.* 4, 67-84.

Fraser, G. 1976. The causes of profound deafness in childhood. Johns Hopkins University Press, Baltimore.

The role of audiometry in occupational noise management

Investigator: John Macrae

It has often been suggested that annual monitoring audiometry provides a means of early detection of failures in occupational noise management. Annual monitoring audiometry consists of a reference audiogram taken before occupational noise exposure has begun, followed by monitoring audiograms at yearly intervals during the course of exposure. However, it can be shown that annual monitoring audiometry is not effective in the early detection of noise-induced hearing loss, where early means within about one year of the beginning of a failure in the program (Hetu, et al., 1990).

A significant threshold shift criterion of 15 dB has been widely used in monitoring audiometry. The average worker will only obtain a noise-induced permanent threshold shift (NIPTS) of 15 dB at 4 kHz, the frequency most affected by noise exposure, after about 5 years of exposure. Even the more susceptible workers will only obtain a NIPTS of 15 dB after more than 2 years of exposure and the least susceptible workers may not obtain a NIPTS of 15 dB in a working lifetime. Annual monitoring audiometry is, therefore, not effective in early detection of NIPTS.

A considerable amount of damage to the inner ear will occur before the effects of shortcomings in a noise management program become apparent in audiograms. In the absence of an effective noise management program, audiometry serves merely to record progression of hearing impairment in workers and as a source of information about liability for compensation. Audiometric testing is not itself a protective measure and

is relevant only within the context of a comprehensive noise management program.

If audiometry is to have a role to play in occupational noise management programs, it is essential to increase its effectiveness for the early detection of noise-induced threshold impairment. This can be achieved, firstly, by averaging thresholds across frequencies and repeated tests and, secondly, by using audiometry to detect noise-induced temporary threshold shift (NITTS). Instead of taking one measurement on each test occasion, it is better to take more than one measurement on each occasion and average the outcomes to obtain a result. By this means, the retest variability of the result is reduced.

Macrae (1994) has confirmed that retest variability diminishes at various frequencies as the number of tests included in the average is increased. Thresholds can also be averaged across frequency. Dobie (1983) has shown that retest variability diminishes when thresholds are averaged across frequency, with half-octave spacing between the frequencies included in the average. He found that the optimum number of frequencies to include in the average is three. On the basis of these findings, Macrae (1994) has recommended the use of a procedure based on averaging of thresholds at the frequencies 3, 4 and 6 kHz across two tests. These are the frequencies most likely to be affected in the early stages of noise exposure. For this average, a change of 4 dB is statistically significant.

In order to increase the effectiveness of audiometry in the prevention of noise-induced hearing loss, it seems sensible to use it to detect NITTS, as suggested by Noble (1978). It can be shown that, if noise exposures are reduced to amounts where they do not cause NITTS, then it is unlikely that they will cause permanent hearing impairment. Macrae (1994) has shown

that, if predicted group average amounts of daily NITTS are close to 0 dB, then predicted group average amounts of eventual NIPTS will also be close to 0 dB. It follows that occupational noise management programs should be oriented towards prevention of NITTS.

References

- Dobie, R.A. 1983. Reliability and validity of industrial audiometry: implications for hearing conservation program design. *Laryngoscope* 93, 906-927.
- Hetu, R., Quoc, H.T., & Duguay, P. 1990. The likelihood of detecting a significant hearing threshold shift among noise-exposed workers subjected to annual audiometric testing. *Ann. occup. Hyg.* 34, 361-370.
- Macrae, J.H. 1994. Improved detection of hearing threshold impairment caused by occupational noise exposure. *Aust. J. Audiol.* 16, 25-35.
- Noble, W.G. 1978. Monitoring audiometry: protection for whom? In: Waugh, R.L. & Macrae, J.H. (Eds), *Occupational Hearing Loss: Conservation and Compensation* (pp.164-177). Australian Acoustical Society, Sydney.

NOISE AND HEARING LOSS PREVENTION

A major objective of NAL research is to increase understanding of the harmful effects of noise on people and to contribute to the prevention of hearing loss. The following four sections describe NAL research which addresses these objectives.

Two sections are historical features, one on hearing loss prevention research and one on the development of community criteria for the acceptability of military noise. NAL has been concerned with hearing loss prevention throughout its history and, indeed, in the time of its predecessors, the Acoustic Testing Laboratory and the Acoustic Research Laboratory. Thus, over 50 years are covered in the historical overview of NAL's hearing loss prevention research. The second historical feature covers a more limited time span and a more specialised topic. It is one to which NAL research made a unique contribution through a series of studies conducted over several years.

The other two sections present projects concerned with hearing loss prevention and community noise. The latter provides an update on the investigations of noise associated with the operations of Sydney airport whereas the former presents six varied projects. Broadly these are all concerned with hearing loss prevention but different projects focus on different risk groups such as young people listening through stereo headsets, industrial workers on extended workshifts, orchestral musicians, and telephonists.

NAL Engineer, Warwick Williams measuring noise at a borehole drilling operation – Dead Bullock's Soak open cut pit. Granites Gold Mine, Northern Territory.



Hearing Conservation – from before the beginning

Historical Feature: Narelle Murray and Eric LePage

*NAL's predecessor, the Acoustic Research Laboratory, was established to investigate problems of hearing loss associated with military service. Part of that brief was the **prevention** of hearing loss and one of the laboratory's first accomplishments was the design of an effective earplug. Another invention was the Murray speaking tube which assisted communication and provided hearing protection within the noisy cockpit of an aircraft. Throughout its history, NAL has been the leading Australian organisation in promoting hearing loss prevention through its research, public education, and its vigorous participation in Standards activities. Research includes the development of a variety of hearing protection devices, the development of valid methods for testing hearing protectors, the development of a scientifically based method for assessing compensable hearing loss, and the development of methods for detecting ear damage before a significant hearing loss occurs. Thanks largely to NAL's efforts, Australia is one of the most advanced countries in protecting its people's hearing. The following feature outlines some of NAL's accomplishments in hearing loss prevention.*



One of the first inventions of the acoustic laboratories was the Murray speaking tube, used for communication and to provide some hearing protection to air force personnel.

Five years before the Commonwealth Acoustic Laboratory came into being its predecessors, the Acoustic Testing Laboratory (ATL) and the Acoustic Research Laboratory (ARL), under the direction of Norman E. Murray, were investigating the problems of noise, hearing and communication particularly in the Army and Air Force. The first "noise" report ("Noise in Australian AC1 Cruiser Tank")¹ was written in 1942, followed by the first hearing conservation reports written in late 1943 when earplugs were developed by the ATL and tested against gunfire noise and blast. These were followed by other earplugs developed in 1945 including the first report of a ventilated earplug developed by N.E. Murray and A.H. Pollard² for use by the RAAF. During this time Murray and Pollard also developed the Murray speaking tube system for intra-aeroplane communication in the RAAF incorporating both 'efficient communication and attenuation of external noise'³. Further reports during the 1940s on both temporary and permanent effects of gunfire on hearing followed together with the initial design of earpad, helmets and headsets for acoustic efficiency in noise. The 1950s saw the design of the Calmuff, which allowed reasonable ability to communicate and perceive warning signals while providing sufficient protection from hearing damage by most industrial and aircraft noises. By 1960 this was commercially available from the Protector Goggle Co. Pty. Ltd⁴.

Sound Level Conversion, or SLC₈₀⁷ formed the basis of the Australian Standard AS1270 and is still used in the acoustical measurement of the attenuation of hearing protectors.

Also during the 1950s, a major emphasis was investigations at RAAF air stations where exposure of personnel to the high level noise from jet aircraft had become a serious problem. As a result of a 1957 investigation (published in 1959 by N. E. Murray, R. Piesse and J. Rose as "Noise and hearing conservation in the RAAF"⁵), hearing conservation programs were set up early in 1960 at eight RAAF bases both in Australia and Malaya (as it was then). By 1961 Murray had agreed to assist with an extensive and extended audiometric survey amongst members of the Australian Military Forces.

Further work was carried out during the 1960s by Ray Piesse on the effects on hearing of armoured personnel carriers, together with evaluation of hearing protection for use in the M551 armoured fighting vehicle (N. Carter, R. Waugh and D. Brigden)⁶. At the end of 1961 the Standards Association of Australia authorized the formation of a Technical Committee on Acoustics and C.A.L. was invited to join the first committee. This association has continued ever since. Norman Murray was also invited to represent Australia at International Standards Organisation Committee Meetings in Germany in 1962. Again, staff of the Laboratories have served continuously on ISO Committees since that time.

The 1970s saw a rise in interest in the effects of noise exposure. Associated with this was the development of a means of rating ear protector performance by Dick Waugh. Known as Sound Level Conversion, or SLC₈₀⁷ (a guide for protection for 80% of wearers) this formed the basis of the Australian Standard AS1270 – Hearing Protectors and is still used in the acoustical measurement of the attenuation of hearing protectors. Also arising from the interest in the effects of noise, John Macrae developed procedures for assessment of hearing loss as a percentage loss of hearing⁸ and, with modifications made in the 1980s, these procedures are still used in all hearing loss compensation claims throughout Australia. "Criteria for assessing hearing conservation audiograms"⁹ (Waugh and Macrae, 1980) are also incorporated in the Australian Hearing Conservation Standard.

With the increased awareness of the effects of leisure as well as occupational noise on people, Norman Carter and associates, from 1975 through to 1982 conducted a series of surveys and audiometric testing of young people ranging in age from 10 to 33 years¹⁰. Carter also began studying non-auditory health effects of occupational noise¹¹, and the effect of noise on cardiac arrhythmias during sleep¹².

Occupational hearing loss still engaged the attention of researchers during the 1980s with research by N.M. Murray and R. Waugh on ambient noise levels for audiometric testing for hearing conservation purposes outside a sound booth. Their recommendations were accepted into the Australian Hearing Conservation Standard¹³. A national strategy for the prevention of noise-induced hearing loss, prepared by Dick Waugh, had its basis ultimately accepted by the National Occupational Health and Safety Commission as its National Standard.

There was some continuation of interest in hearing protection for hearing conservation purposes during the 1990s, with studies carried out on the effect of hearing protectors on localisation of sound (Noble, Murray and Waugh)¹⁴, and the development of an aural reflex eliciting earmuff for use in impulse noise by Carter¹⁵.

The introduction by Eric LePage in 1989 of otoacoustic emissions has resulted in a dramatic shift from hearing conservation based solely on sound exposure, to include studies of *prevention* of hearing loss based on cochlear physiology¹⁶. This recognised the impact of many factors which result in ear damage and/or early deterioration of hearing capabilities including tinnitus; influences such as noise (both occupational and leisure), antibiotics, head injuries, and ageing have now also been taken into consideration. In 1995 LePage published a model for cochlear origin of

Otoacoustic emissions has resulted in a dramatic shift from hearing conservation based solely on sound exposure to include studies of prevention of hearing loss based on cochlear physiology.

subjective tinnitus¹⁷. A model forecasting the prevalence of hearing loss in the Australian population based on trends in the decline of otoacoustic emission strength¹⁸ was also published by LePage together with rates of ageing and ageing characteristics of the Australian population (Murray et al)¹⁹ and comparisons of otoacoustic emissions and pure tone audiometry (LePage and Murray)²⁰.

Granted that noise is still possibly the most widely acknowledged of these factors impinging on the auditory system, studies using otoacoustic emission testing techniques have been undertaken in various sub-populations such as coal miners, orchestras, prisons, and primary and high schools. An interest in leisure noise has again surfaced from observations emerging from questionnaires applied to these groups, particularly in relation to exposure to loud amplified music (both live and through personal stereo cassettes).

References

1. Murray, N.E. Dec. 1942. Noise in Australian AC1 Cruiser Tank. *ATL Report No. 2*.
2. Murray, N.E., & Pollard, A.H. Nov. 1945. Design of a perforate earplug. *ARL Informal Report 24*.
3. Murray, N.E., & Pollard, A.H. July 1945. The Murray speaking tube system. *ARL Report 11*.
4. Piesse, R.A. 1962. Ear Protectors, *CAL Report No. 21*.
5. Murray, N.E., Piesse, R.A., & Rose, J.A. 1959. Noise and hearing conservation in the R.A.A.F. *CAL Report 15*.
6. Carter, N.L., Waugh, R.L., & Brigden, D. 1968. Evaluation of the CVC helmet as a communications headset and ear protector for use in the M551 armoured fighting vehicle. *CAL Report 47*.
7. Waugh, R.A. 1976. Investigation of sound level conversion as a means of rating ear protector performance. *Am. Indust. Hygiene Assoc. J.* 37, 239-45.
8. Macrae, J.H. 1977. Documents concerning the NAL procedure for determining percentage loss of hearing. *NAL Informal Report 45*.
9. Waugh, R., & Macrae, J. 1980. Criteria for assessing hearing conservation audiograms. *NAL Report 80*, AGPS, Canberra.
10. Carter, N.L., Murray, N.M., & Bulteau, V.G. 1985. Amplified music, recreational noise and hearing in young people aged 16-21 and 28-33 years. *Aust. J. Audiol.* 7, 79-83.
11. Carter, N.L. 1986. Non-auditory health effects of occupational noise. *J. Occup. Health Safe. Aust. NZ*, 2, 210-215.
12. Carter, N.L., Hunyor, S.N., & Kenna, L.C. 1986. Rationale and method of an ongoing study of the effect of noise on cardiac arrhythmias during sleep. *Proceedings, Community Noise Conference*, Toowoomba, Queensland.
13. Murray, N.M. & Waugh, Dick. 1988. Estimated Maximum Acceptable Background Noise Levels for Audiometric Testing when using sound-excluding earphone enclosures. *Aust. J. Audiol.* 10, 7-10.
14. Noble, W., Murray, N.M., & Waugh, R. 1990. The effect of various hearing protectors on sound localisation in the horizontal and vertical planes. *Am. Ind. Hyg. Assoc. J.* 51, 370-377.
15. Carter, N.L., French, H.T., & LePage, E.L. 1995. Evaluation of the Aural Reflex Eliciting (AR) Earmuffs – Progress Report No. 2. National Acoustic Laboratories, Sydney.
16. LePage, Eric L. 1992. Hysteresis in cochlear mechanics and a model for variability in noise-induced hearing loss, in: *Noise-Induced Hearing Loss*, Eds. Dancer, D. Henderson, R.J. Salvi, R.P. Hamernik, Mosby Year Book, Vol. 10, St. Louis, 106-115.
17. LePage, E.L. 1995. A model for cochlear origin of subjective tinnitus: Excitatory drift in operating point of inner hair cells. In: *The Mechanisms of Tinnitus*, edited by J. Vernon and A.R. Moller. Allyn and Bacon Publishers, Boston, Chapter 11, 115-147.
18. LePage, E.L. 1994. 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 Better Hearing Australia Conference*, Adelaide, August 7-11, 1994.
19. Murray, N.M., LePage, E.L., & Tran, K. 1994. Ageing characteristics of the Australian population in terms of otoacoustic emission strengths: global and individual picture. *Proceedings Better Hearing Australia Conference*, Adelaide, August 7-11, 1994.
20. LePage, E.L., & Murray, N.M. 1993. Click-evoked otoacoustic emissions: comparing emission strengths with pure tone audiometric thresholds. *Aust. J. Audiol.* 15, 9-22.

Procedures for assessment of hearing loss as a percentage loss of hearing⁸ are still used in all hearing loss compensation claims throughout Australia.

An interest in leisure noise has again surfaced.

Hearing Loss Prevention

Are people aware that loud noise can damage hearing? Do they believe it can happen to them? Do they care? Is leisure noise a problem? Can we measure ear damage before hearing loss occurs? Are extended, noisy workshifts a special problem? How can we reduce "noise" for orchestral musicians? How do we prevent telephone headsets from emitting damaging noises? These are some of the many, diverse questions being addressed by NAL's hearing loss prevention research. The prevention of hearing loss raises a host of attitudinal, social, technical, legal and economic issues. NAL's innovative research program addresses these issues and thereby continues to serve the Australian community and contributes to scientific knowledge.

Survey: Awareness of hearing issues among the Australian population

Investigators: Eric LePage, Sheralyn Shacknoffsky (AHS NSW), Narelle Murray

Background: A survey was conducted because it was felt that people, particularly young people, are loath to take warnings about loud noise and hearing loss seriously. We hypothesised firstly that loud sound, particularly loud music, represents an attractive vibrant lifestyle and secondly that while young people hear warnings that loud sound damages their hearing, this is not borne out by their own experience. They can repeatedly expose themselves to very loud sounds or music for years and they judge that it has no effect. The survey was designed to find out what thoughts, if any, young people had about the causes of hearing loss and what having a hearing loss might be like.

Methods: Questionnaires were designed to ask a series of multiple choice questions which could be fitted on a single page. Fifteen hundred copies were distributed to as many places as could be managed in two weeks. They were designed to be folded in three and included a reply-paid address. Responses were returned from approximately 300 people, predominantly students and young adults, but all ages and many occupations were represented.

Results: People were asked to rate in importance how bad it would be to have one of ten kinds of disability. Loss of vision was regarded as the worst kind, followed in order by loss of mobility (paraplegia), loss of memory, hearing, speech, touch/feeling, writing hand, appearance, smell/taste, other hand. Hearing was thus perceived to be important, but overall rated fourth in the list.

People were asked to rate how likely they thought that they would ever have a hearing loss. Only 6 percent thought it likely whereas the actual incidence is about double that. Forty percent allowed that they might, while the rest rated the chance very low, or had never thought about it.

Does normal hearing mean no ear damage? Only one tenth allowed that damage may prelude actual hearing loss; one third said "yes", one third didn't know and one quarter didn't answer the question. This outcome generally reflected the traditional notion that hearing is fine until a mild hearing loss occurs. The approximately equal number of responses in the negative indicate that few people have ever thought that the common term "hearing damage" can be clearly distinguished into cause, "ear damage", and effect, "hearing disability" or that the effect may be a much-delayed outcome.

The next question was designed to assess how much empathy people feel for others who have a hearing loss since hearing loss has long been described as a hidden disability. Only ten percent thought that someone they knew was seriously disabled by their disability, two thirds sensed some disability, while one fifth were not aware of a problem or didn't answer the question. This outcome rather suggests that people with hearing problems are considered to cope quite well, so the disability cannot be too serious.

To the question, "Which best describes hearing loss?" The answers were almost equally divided between the following diverse manifestations each of which has some factual basis: volume control up; volume control down, no escape from noise, loss of focus, indistinct sounds, loss of clarity, mumbled speech. Two percent answered with loss of direction. Again these answers suggest that the average person really has only a very vague idea of what it is like to have a hearing loss. The responses highlight the fact that hearing loss prevention education programs can be considerably more specific in highlighting loss of the normal ability to select, locate or sort sounds as being responsible for most of the frustration in hearing loss.

The next question was designed to assess the extent to which people are aware how much their ears have been traumatised if they experienced tinnitus subsequent to a loud sound exposure, or how much they were able to ignore the trauma the moment it ceased. Only one fifth thought such trauma might have a permanent effect, nearly half thought the problem was temporary and nearly forty percent gave it no further thought. Overall the responses indicate that people do take their hearing for granted and can readily ignore symptoms since they are not generally associated with pain.

Some years back it was regarded as unmasculine for men to wear ear protection, so the question was designed to obtain current attitudes to the wearing of hearing protection devices: ear muff or ear plugs. "If you were in a very noisy workshop for just 5 minutes and hearing protectors were provided would you use them?" The results were approximately equally divided. Of the 46 percent who would not, two fifths would not because of their need to converse, two fifths wouldn't bother while a fifth still regarded the suggestion as unmasculine.

To gauge what people regard as uncomfortably loud, people were asked to rate a series of public events according to discomfort. There was a clear distinction between the too loud category (rock concerts, discos, motor races and starting guns) compared with fewer people regarding fireworks, cinemas, orchestra concerts, football matches, supermarkets and tennis matches as uncomfortably loud.

Peer pressure is an important factor in fixing and assessing people's response to loud sound. The next three questions were designed to test the level of embarrassment people feel in displaying signs of "weakness". Forty-three percent actually indicated they would ask a party host to turn the music down, thirty percent would do nothing, twenty percent would move away and four percent would use ear plugs.

Much of the problem in mounting effective hearing loss prevention strategies stems from the non-impact of hearing issues on general thinking until disability strikes.

At a noisy sports venue nearly two thirds would wear no ear protection, but would use their hands when necessary; one third would buy earplugs if available. Three percent would try to use paper tissue as earplugs. These responses clearly indicate that people prefer the discomfort of ear trauma rather than not being able to converse. Those who would use paper tissue admit the need, but show no knowledge that paper tissue provides little attenuation.

Most people own sunglasses to protect their eyes against overstimulation or glare, so the next question was designed to determine whether ears are regarded similarly with caution. Only just over a quarter of respondents indicated they had ever bought a pair of foam earplugs. This may indicate that possessing optical accessories is almost as much a fashion statement as a desire to reduce discomfort. It may also reflect the fact that people are much more reluctant a) to insert objects into their ears and b) to suffer the additional difficulties in verbal communication which result from the wearing of plugs. Certainly those who frequently do wear foam earplugs tend to wear them not fully inserted to trade off protection versus continued ease of communication.

Much attention has been drawn to noise as a cause of hearing loss over the past 100 years. The next question was designed to test people's awareness of other factors. Of the factors suggested, (loud noise, infections, head injury, birth trauma, drugs, cap guns) all scored highly as expected. It was revealing, however, that many fewer people rated their hearing at risk due to solvents and toxins, antibiotics and aerobics.

The second last question was designed to check people's impressions about whether loud music is less damaging overall than noise. One fifth of respondents thought music was less damaging than noise. The question might well have been split to gauge responses to the relative effects of different kinds of music. Our otoacoustic emission evidence is that the way most people use music it can be as damaging as noise.

The last question was designed to test the penetration of the \$250,000 AHS publicity campaign "Noise is murder on your ears" conducted just prior to this survey. One fifth of the respondents said that they had heard of the campaign.

Conclusions: The survey revealed that much of the problem in mounting effective hearing loss prevention strategies stems from the non-impact of hearing issues on general thinking until disability strikes. There appears to be a high level of ignorance which the average person possesses in respect of how their hearing works and that education based upon actual risk may be an important prelude to achieving changes in attitudes and behaviour.

Significance: This investigation is a part of an ongoing evaluation of the reasons for engaging in hearing loss prevention research, the costs to our society for ignoring an apparent rise in leisure-related noise trauma and the strategies for making research and clinical programs more effective.

Repeatability of click-evoked otoacoustic emission measures

Investigators: Narelle Murray, Eric LePage and Khanh Tran

Background: Evoked Otoacoustic Emissions (EOAEs) have been revealed to be a useful, noninvasive method of examining cochlear function which are reproducible. However, for any procedure to be accepted, particularly for clinical use, it is important that the outcome of any test result can be generalised across time. Therefore, to determine whether changes in test results are actual functional changes in the cochlea rather than measurement error, it is necessary to determine the test/retest reliability of the procedure itself.

Research Questions: Apart from the question of the test/retest reliability of the procedure itself, the question remains as to its sensitivity. That is, what changes over time constitute a real change in cochlear activity and not purely measurement error? Does the empirically calculated Coherent Emission Strength constitute a viable alternative for assessing changes in cochlear function?

Research Procedures: 12 subjects (6 males and 6 females aged between 28 and 47 years) were tested daily for six consecutive (work) days. An Otodynamics Otoacoustic ILO88 Analyser set at peak stimulus level of 80 (± 1.5) dB SPL with 300 repetitions of the pulsed click train was used (LePage & Murray, 1993). This repetition number resulted from the particular test procedure used. This rate gives a maximal signal to noise improvement of 25dB which is 1 dB better than that obtained with the standard ILO default settings of 260 repetitions, commonly used as a screening method. Statistical analyses were undertaken for both the calculated Coherent Emission Strength and the cross power spectra for each individual.

Findings: Results indicate that otoacoustic emissions are strongly reproducible, viz. there were no statistically significant intra-subject differences ($p < 0.01$) either for the spectra analysed as a total spectrum or for most of the 1 kHz-wide frequency bands analysed. A 5% change in emission strength was found to indicate a real change in cochlear function.

Significance: Our research has confirmed that otoacoustic emissions are highly reproducible and set a limit on the variation above which further testing is recommended.

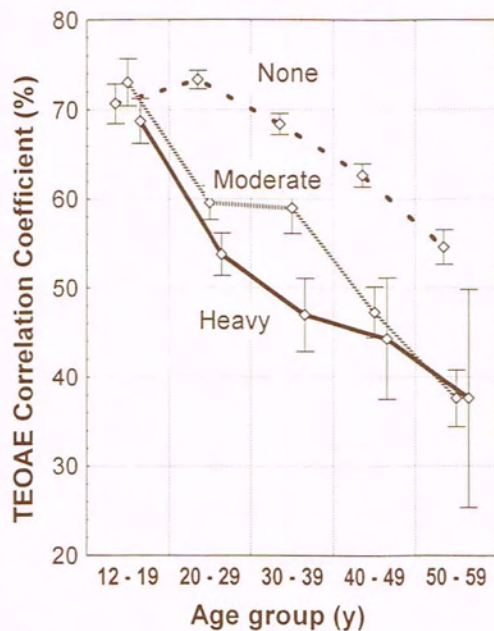
Accelerated Ear Damage in Listeners to Headset Exposure

Investigators: Eric LePage and Narelle Murray

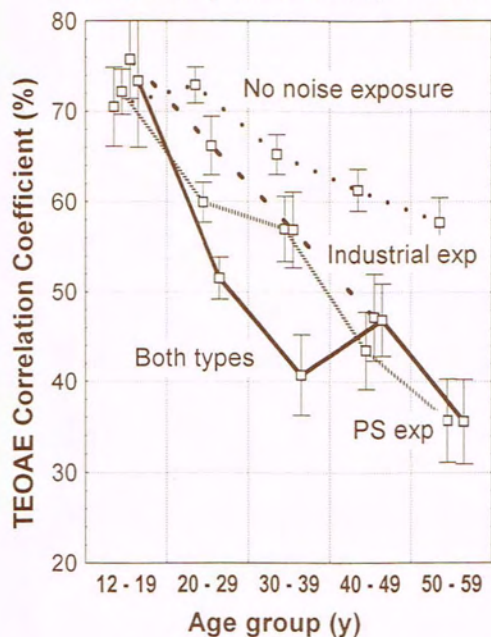
Background: This study is the classic case of the need to repeat earlier studies because of developments in understanding or technology. Otoacoustic emissions provide such a fundamentally new insight into the processes of hearing in humans that previous models of phenomena such as presbycusis (the loss of hearing with advancing age) require re-evaluation. Until the advent of otoacoustic emissions presbycusis was defined in terms of the numbers of people whose hearing was presenting them with problems in later life and the extent to which each person's hearing threshold had risen. Now it is not necessary to wait until later life to observe the aging process in operation – we can redefine presbycusis as the process of accumulating damage due to all causes. We have shown that transient-evoked otoacoustic emissions have a strength which declines with age at something normally between 2 and 3 dB/decade. At that rate, peoples' otoacoustic emission strength, on average, reaches a critically low value between 50 and 60 years of age when the first symptoms of hearing loss appear. The decline in emission strengths with age can now be monitored from birth. Since emission strengths in babies are typically the highest seen it is very easy to identify babies who are beginning life with cochlear damage. Indeed, not infrequently, cochlear emission strengths are low in newborns due to genetic problems, maternal health or behaviour during pregnancy or birth trauma. Screening these children allows parents and communities to undertake earlier intervention.

In an early otoacoustic emission study we examined otoacoustic emissions in a team of coal miners in the Newcastle area and found that on average their emission strengths were significantly below the emission strengths not just for the whole Australian population, but significantly below other males. Only a few had obvious hearing problems. If it were possible therefore to observe preclinical ear damage in a group with a marked exposure to loud noise, it may be possible

PS use: none, moderate & heavy
Males & Females



Exposure: None, PS, industrial, both
Male mutually exclusive populations:



Correlation Coefficient of the transient evoked otoacoustic emission. Data shown are the means and standard errors of the different population subgroups. Where the ranges are clearly disparate, the populations are significantly different ($p \leq 0.001$).

to observe early damage in other groups who are subjected to high levels of sound. Accordingly, it was decided to be more systematic in obtaining client histories for correlation of hereditary and environmental factors with the emission picture. Of particular interest has been concern expressed since the Walkman radio was introduced in 1980 that personal stereo (PS) headsets may more rapidly lead to hearing loss since users commonly have them loud enough for others to hear the headphones from a distance. Several earlier studies, including some conducted at NAL (Carter et al, 1982; 1984; 1985) failed to show any effect.

Methods: This present report is the byproduct of several other studies which have contributed to our normative database from approximately 2500 people. The data are exclusively transient evoked otoacoustic emission data obtained with the Otodynamics Analyser (ILO88). Many individuals in the database have provided multiple records so this dataset of 13,500 records was effectively reduced to a single record for each ear which represented the mean values of the extracted parameters. The pruned database is from 2453 persons. It comprises 4250 records from individual ears, 2587 male and 1663 female ears. Answers to detailed questionnaires were obtained from 1325 people. On the basis of these answers and any other comments offered, each response was reclassified as 0, 1 or 2 respectively according to whether their PS exposure was negligible ($n_{\text{ears}}=2227$), moderate (<6 hours per week, $n_{\text{ears}}=693$) or heavy (>6 hours per week, $n_{\text{ears}}=405$). Likewise each subject was classified as 0 or 1 according to whether they worked in noisy industry.

Results: The left panel of the figure for males and females combined shows that there are significant differences in emission strength between the three groups: (none, moderate and heavy), in all but the first age range. It indicates that there is a marked inverse dependence of emission correlation coefficient upon estimated PS exposure. The effect exists for both males and females beyond the lowest age range. The rapid decline in the second age range indicates that accumulation is marked in the teenage males who use these devices relative to those who do not. PS exposure, however, will only account for some of this rapid decline.

The database was alternately partitioned into four mutually exclusive groups based upon questionnaire answers: (1) no noise risk factors ($n_{\text{ears}}=649$), (2) PS exposure alone ($n_{\text{ears}}=470$, groups moderate and heavy from left panel), (3) Industrial noise exposure alone ($n_{\text{ears}}=233$), and (4) PS plus industrial exposure of the same people ($n_{\text{ears}}=365$). The right half of the figure shows for males only that the industrial noise-exposed group exhibits a similar decline in correlation coefficient but in the case of the PS exposure it is tending to occur a decade earlier, i.e. the PS exposure has evidently produced similar damage but earlier in life. For the PS plus industrial exposed group the mean values are significantly below all other groups and also significantly below the other noise groups up until the fifth decade (>40 years).

Discussion: The effect of protracted use of personal stereos is more marked for males, but other factors paired in lifestyle may have influenced the data. It was expected that PS users would have accumulated less damage than workers in

heavy industry yet the right panel shows that both groups produced similar emission strength values across the young adult age range. These findings imply that indeed typical use of personal stereo headsets causes latent ear damage comparable with occupational noise damage. Moreover, the lowest curve points to a significantly higher rate of damage accumulation in young males exposed to both types of noise. All three exposure groups showed values significantly below the mean values for the non-exposed population. Personal stereos have been around for less than 20 years and the fact that the differences are significant up to about age 40 is remarkably consistent with the popularity of these devices with young people.

Significance: We have previously shown there is evidently a very long lead time required for sufficient cochlear damage to accumulate to become evident as a hearing disability. Twenty years is evidently not enough time for the accelerated aging effects to become manifest as a marked increase in the number of young adults seeking help. On the other hand these data provide very clear evidence that these devices (and any other paired factors) do result in significantly increased cochlear damage. The results tend to support the cautionary advice offered based upon existing guidelines for limits to sound level exposure for occupational noise laid down by Statutory Regulations of the various Australian States and Territories ($85 \text{ dBA}_{\text{shr}}$) since with the high volume levels used these limits are, in some cases greatly exceeded.

Remarkably only 39 questionnaires from PS users reported any hearing difficulties. The primary significance of this research program is that click-evoked otoacoustic emissions may detect the precursor of observable hearing loss, and that many young people are exceeding rates of damage accumulation greater than 2 dB/decade. In some instances we have tracked young people with emission strengths declining at greater than 15 dB/decade (LePage and Murray, 1993). These people may expect to suffer hearing loss significantly earlier in their lives without modification of their habits.

In summary, our OAE estimates of risk are not based upon sound levels, but upon a physical measurement used to estimate permanent cochlear damage. They indicate that personal stereos produce rapid aging of the cochlea comparable to industrial noise trauma. Secondly, general guidelines based only upon recommended volume levels may be applicable for one person but irrelevant for another. Thirdly, otoacoustic emissions potentially offer new precision in individual risk determination in adults.

References

- Carter, N.L., Waugh, R.L., Keen, K., & Murray, N.M., 1982. Bulteau V.G. Amplified music and young people's hearing. *Med. J. Aust.* 2:125-8.
- Carter, N.L., Murray, N.M., Khan, A., & Waugh, R.L. 1984. A longitudinal study of recreational noise and young people's hearing. *Aust. J. Audiol.* 6:45-53.
- Carter, N.L., Murray, N.M., & Bulteau, V.G. 1985. Amplified music, recreational noise and hearing in people aged 16-21 and 28-33 years. *Aust. J. Audiol.* 7:79-83.
- LePage, E.L. 1994. 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.*
- Murray, N.M., LePage, E.L., & Tran, K. 1994. Ageing characteristics of the Australian population in terms of otoacoustic emission strengths: global and individual picture. *Proceedings of the Better Hearing Australia Conference, Adelaide, 7-11 August, 1994.*

Typical use of personal stereo headsets causes latent ear damage comparable with occupational noise damage.

Noise and Hearing Loss Prevention Research

A major objective of NAL research is to increase understanding of the harmful effects of noise on people and to contribute to the prevention of hearing loss. NAL research that addresses these objectives is described in the following two sections.

The first two items question the justification for currently accepted recommendations that compensation for hearing loss should begin at 10% percentage loss of hearing (PHL) and that noise levels should be limited to 85 dBA. It is agreed that a significant proportion of people with PHLs of 5-10% require hearing aids and that the 85 dBA noise limit will do little more than halve the problem of industrial deafness. A further item describes a longitudinal study of ear damage in orchestral musicians. This work has implications for designing and managing a more comfortable and safer environment for musicians who, of necessity, are frequently exposed to high sound levels.

Hearing loss prevention research has been using otoacoustic emission testing over several years. The first item in the following section presents one outcome of that line of research. Analyses of repeated tests of people followed for up to nine years suggest particular ways in which the functioning of the ear changes as damage accrues. It is expected that further data treatment will permit the identification of specific indicators of risk of hearing loss for individuals.

The second section provides a brief update on NAL's participation in the Sydney airport health study. This multifaceted study has wide implications for a large proportion of Sydney's population and, through its contributions to scientific knowledge, to managing the noise problems that occur wherever busy airports are sited near population centres. Other items describe a criterion for predicting community annoyance caused by impulsive noise and the development of noise protection shields for musicians.



Eric LePage and Narelle Murray, scientists responsible for hearing loss prevention research.



Hearing Loss Prevention

Workers Compensation for Industrial Deafness

Investigator: John Macrae

Background: Throughout Australia, hearing disability is assessed for compensation purposes in terms of percentage loss of hearing (PLH), determined from the hearing threshold levels of the compensation claimant (Macrae, 1988). In 1994, the Industry Commission Report on Workers Compensation in Australia (Industry Commission, 1994) recommended that a common Table of Injuries be developed to apply across all Australian jurisdictions. As a result, the Heads of Workers Compensation Authorities included this as a part of the national harmonisation process. A review of PLH thresholds formed part of the review under the Standardised Measurement of Impairment Project. In 1997, the final report of the Heads of Workers Compensation Authorities (Heads of Workers Compensation Authorities, 1997) recommended that:

- a PLH threshold of 10% apply for compensability; but
- a PLH of 5% or greater will trigger rehabilitation for the worker and workplace assessment as a preventive initiative;
- where the threshold for compensability is attained, the full PLH is compensated; and
- subsequent claims must demonstrate at least a further 5% deterioration from the previous PLH.

The following work was carried out in order to obtain information that could be used to assess these proposals.

Research Questions: Two relevant matters were investigated: first, the relationship between PLH and requirement for hearing aids; second, the retest variability of PLH

Procedures: The relationship between PLH and requirement for hearing aids was investigated in two ways. In the first approach, a sample of the hearing thresholds of 436 child and age pensioner clients provided with hearing aids by Australian Hearing was drawn at random from files and the PLHs of the clients were calculated from their thresholds. In the second approach, the associated PLH was calculated from the hearing thresholds of 282 war veterans whose threshold impairments were mainly due to noise exposure and whose requirements for hearing aids were known.

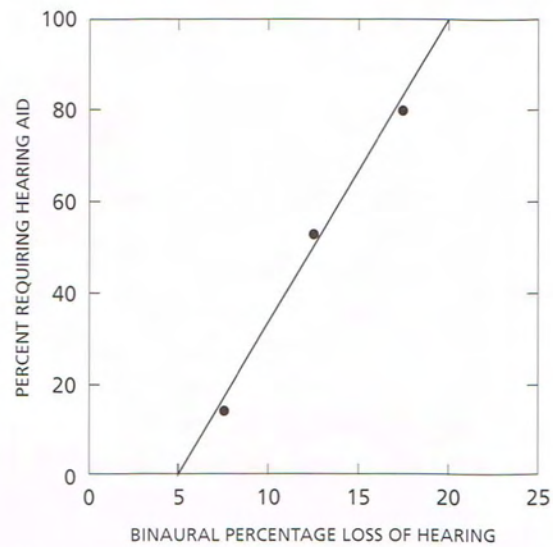
The retest variability of PLH was investigated in the following manner. Since there is retest variability associated with hearing thresholds and PLH is derived from hearing thresholds, the retest variability of PLH was derived from the known retest variability of hearing thresholds. It is well known that, in the absence of a real change in threshold sensitivity, hearing thresholds vary on retest in accordance with the law of random error and the changes are, therefore, normally distributed and that there are no correlations between the random variations of the thresholds at the various test frequencies. The audiograms of 684 war veteran, child and age pensioner clients who had been provided with hearing aids by Australian Hearing Services were obtained from files and the associated PLH was calculated. The thresholds were then varied randomly by computer, the associated PLH was re-calculated and the differences between the PLH before and after the random changes were determined.

Findings: The PLHs of the 436 child and age pensioner clients provided with hearing aids by Australian Hearing Services are shown in the Table, opposite, far right. The one client in the category 0-4.9% had a PLH of 4.9%. This result indicates that some clients with a PLH of about 5% require hearing aids.

The relationship between PLH and requirement for hearing aids of the 282 war veterans is shown in the Table below. No veterans with a PLH in the range 0-4.9% required hearing aids. All of the veterans with a PLH of 20% or greater required hearing aids. In the intermediate ranges, the percentage of veterans requiring hearing aids gradually increased. A graph of the findings with a straight line fitted to the data is given in the Figure, right. The results of this approach also indicate that the requirement for hearing aids begins at a PLH of about 5%. Given the illustrated linear relationship, it was calculated that approximately 16% of claimants with a PLH between 5 and 9.9% will require hearing aids.

The standard deviation of the distribution of differences between PLH before and after the random changes in thresholds of the audiograms of the 684 war veterans, children and age pensioners was found to be 1.942%. This means that a change in PLH of 3.2 % is significant at the 5% level or, adopting a more stringent criterion of statistical significance, a change in PLH of 4.5% is significant at the 1% level.

Significance: The Heads of Workers Compensation Authorities recommend that PLH threshold of 10% apply for compensability and that a PLH of 5% will trigger rehabilitation for the worker and workplace assessment as a preventive initiative. In the light of the information presented in this article, it would seem more reasonable to set a PLH threshold of 5% for compensation. The Figure shows that about 33% of those with a PLH of 10% can be



Relationship between percentage loss of hearing and requirement for hearing aids.

expected to require hearing aids.

Requirement for hearing aids begins at a PLH of about 5% for some clients and approximately 16% of claimants with a PLH between 5 and 9.9% will require hearing aids. If a 5% threshold is adopted then there is no need for a trigger for rehabilitation but a trigger for workplace assessment as a preventive initiative should be set at a PLH of 0.1% or greater, since a considerable amount of threshold impairment occurs before the onset of hearing disability. Monitoring audiometry in industry should detect this threshold impairment and trigger preventive action but an extra trigger in terms of PLH may be useful in circumstances where monitoring audiometry is not carried out. If, instead of a threshold of 5%, a threshold of 10% is adopted, the PLH trigger of 5% for rehabilitation and workplace assessment as a preventive

Percentage Loss of Hearing of Children and Age Pensioners with Hearing Aids

% Loss of Hearing	No. of Clients
0-4.9	1
5-9.9	10
10-14.9	22
15-19.9	39
20-24.9	33
25-29.9	31
30-34.9	48
35-39.9	47
40-44.9	28
45-49.9	36
50-54.9	17
55-59.9	26
60-64.9	23
65-69.9	15
70-74.9	14
75-79.9	9
80-84.9	8
85-89.9	7
90-94.9	5
95-99.9	16
100	1

Relationship between Percentage Loss of Hearing and Requirement for Hearing Aids for War Veterans

Percentage Loss (%)	Number of Clients	With Hearing Aids	Without Hearing Aids	Percentage with Hearing Aids
0-4.9	47	0	47	0.0
5-9.9	21	3	18	14.3
10-14.9	17	9	8	52.9
15-19.9	20	16	4	80.0
20+	177	177	0	100.0

initiative becomes especially important. The approved rehabilitative measures should include the provision of hearing aids, where appropriate.

The Heads of Workers Compensation Authorities also recommend that an increase in PLH of 5% must occur before any subsequent claim can be made. This seems to be a reasonable proposal in view of the results presented in this article. If the more stringent 1% criterion of statistical significance is adopted, then a change in PLH of 4.5% is required before a real change in PLH can be considered to have occurred. This becomes 5% when rounded to the nearest whole percentage point. The error rate for a significance level of 1% is 1 in 100, i.e., for 1 out of every 100 claimants with an increase in PLH of 5%, the increase will not be real. However, for the remaining 99 claimants, a real increase in PLH has occurred. This is a suitably low rate of error. The recommendation that an increase in PLH of 5% must occur before any subsequent claim can be made therefore seems appropriate.

References

- Heads of Workers Compensation Authorities 1997. *Promoting Excellence – National Consistency in Australian Workers Compensation*. Final Report, Heads of Workers Compensation Authorities, Melbourne.
- Industry Commission 1994. *Workers Compensation in Australia*. Industry Commission Report No. 36, Australian Government Publishing Service, Canberra.
- Macrae, J.H. 1988. *Improved Procedure for Determining Percentage Loss of Hearing*. NAL Report No. 118, National Acoustic Laboratories, Sydney.
- Macrae, J.H. 1998. Workers compensation for industrial deafness. *Acoustics Australia* 26, 13-16.

An Evaluation of the Australian National Standard for Occupational Noise

Investigator: John Macrae

Background: In response to its concern about the high prevalence of occupational noise-induced hearing loss in Australia, the National Occupational Health and Safety Commission (NOHSC) formally declared, in 1992, the current Australian National Standard for Occupational Noise (National Occupational Health and Safety Commission, 1993). The standard is an $L_{Aeq,8h}$ of 85 dB(A) and an unweighted (linear) peak sound pressure level, L_{peak} , of 140 dB. The National Standard is an advisory document but has affected regulations in the various Australian jurisdictions. By the end of 1996, the Commonwealth and most State and Territory governments had incorporated the National Standard in regulations and had either adopted the National Code of Practice verbatim or incorporated its principles in their own codes of practice (Waugh, 1997).

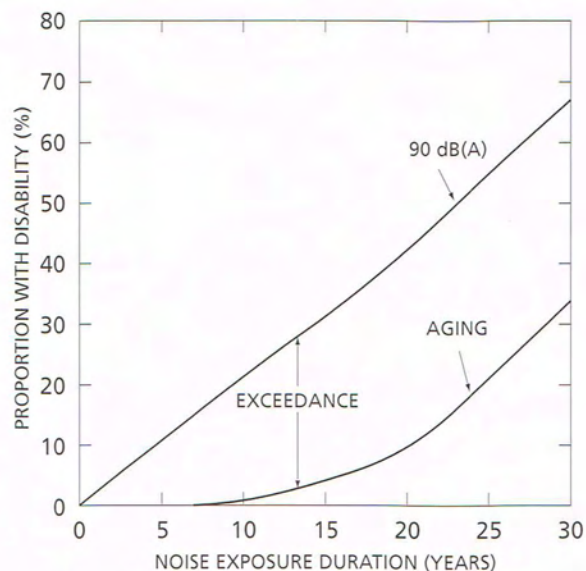
Research Question: Does this National Standard for Occupational Noise go far enough in limiting the permissible noise exposure of workers?

Procedure: The relative effectiveness of different noise exposure levels in preventing the adverse effects of industrial noise exposure on the hearing of workers can be evaluated in terms of hearing disability exceedance. The concept of hearing disability exceedance is illustrated in the figures. The two curves presented in the first figure were derived from values given in a published table of the estimated prevalence of hearing disability in otologically screened, noise-exposed male populations (Macrae, 1986), where otologically screened means free from all signs and symptoms of ear disease other than the effect of occupational noise exposure. The values in the table were calculated by means of equations

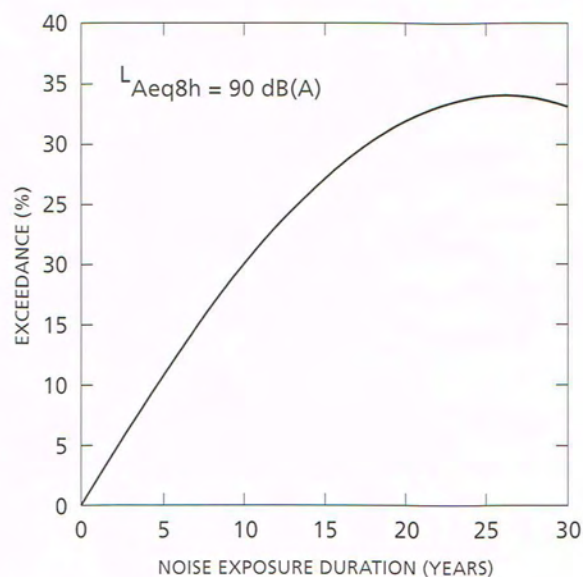
given in International Standard ISO1999 (1990) and the National Acoustic Laboratories (NAL) procedure for determining percentage loss of hearing (Macrae, 1988).

In Australia, hearing disability for compensation purposes is quantified in terms of percentage loss of hearing (PLH), as determined by the NAL procedure. Hearing disability exists if the PLH is greater than zero. Some disability can be expected to occur in some workers not exposed to harmful levels of noise, as a result of the process of aging. This is represented by the lower of the two curves in the graph, above right. For the purposes of the table and the graph, occupational noise exposure is assumed to begin at the age of 20 years. Thus, at the age of 50 years, about one-third of workers not exposed to harmful noise can be expected to have some hearing disability.

The higher of the two curves in the figure shows the proportion of workers who can be expected to have some hearing disability when they are exposed to noise with an $L_{Aeq,8h}$ of 90 dB(A). The difference between the two curves can be described as exceedance, where exceedance refers, in this context, to the amount by which the proportion of noise-exposed workers with hearing disability exceeds the proportion of workers who have hearing disability purely as a result of aging. Subtracting the curve for aging from the curve for 90 dB(A), the exceedance curve shown in the second figure (right) is obtained. Reading this graph, after 25 years of exposure to noise with an $L_{Aeq,8h}$ of 90 dB(A), 34% of the exposed workers will have a hearing disability who would otherwise not have had any hearing disability. In view of the exceedance associated with an $L_{Aeq,8h}$ of 90 dB(A), it is not surprising that occupational noise-induced hearing loss has continued to be a highly prevalent industrial disease in Australia (Macrae, 1997).

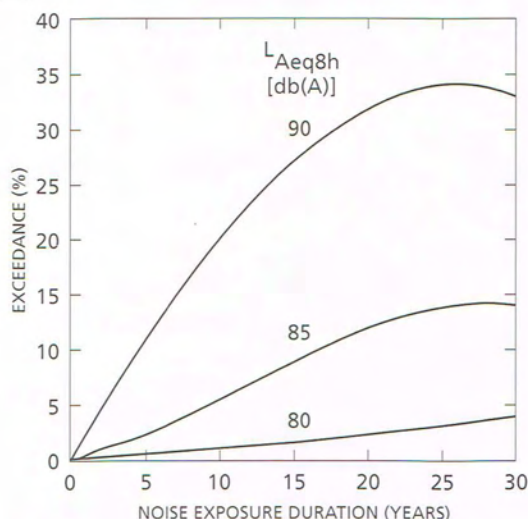


Estimated proportion of a population of otologically screened males with hearing disability, when the population is not exposed to harmful noise (effects of aging alone) and when the population is exposed to occupational noise with an $L_{Aeq,8h}$ of 90 dB(A), as a function of duration of noise exposure, in years. The difference between the two curves at any noise exposure duration is described as hearing disability exceedance. For the purposes of this graph, occupational noise exposure is assumed to begin at the age of 20 years



Hearing disability exceedance (as defined in the previous Figure) for a population of otologically screened males exposed to an $L_{Aeq,8h}$ of 90 dB(A), as a function of duration of noise exposure, in years.

It is apparent that a noise exposure limit of 85 dB(A) will do little better than halve the problem of the high prevalence of industrial deafness in Australia.



Hearing disability exceedance for a population of otologically screened males exposed to occupational noise with $L_{Aeq,8h}$ values of 80, 85 and 90 dB(A), as a function of duration of noise exposure, in years.

In order to assess the relative effectiveness of the noise exposure limit of the National Standard in preventing the adverse effects of industrial noise exposure on the hearing of workers, the predicted hearing disability exceedance associated with noise exposure levels of 80, 85 and 90 dB(A) as a function of noise exposure duration was determined.

Findings: The third figure (above) shows the hearing disability exceedance for noise exposure levels of 80, 85 and 90 dB(A). At a noise exposure duration of 25 years, the

exceedances associated with noise exposure levels of 90 dB(A), 85 dB(A) and 80 dB(A) are about 34%, 14% and 3%, respectively.

Significance: It is apparent that a noise exposure limit of 85 dB(A) will do little better than halve the problem of the high prevalence of industrial deafness in Australia. Given the current magnitude of the problem, a stricter limit seems appropriate. A standard of 80 dB(A) would come much closer to an acceptable solution to the problem of occupational noise-induced hearing loss. Industries would be well advised to aim for a noise exposure limit, $L_{Aeq,8h}$, of 80 dB(A) rather than the National Standard value of 85 dB(A) and serious consideration should be given to reducing the National Standard noise exposure limit to an $L_{Aeq,8h}$ of 80 dB(A).

References

- International Standard ISO 1999 1990. *Acoustics – Determination of occupational noise exposure and estimation of noise-induced hearing impairment*. International Organization for Standardization, Geneva.
- Macrae, J.H. 1986. Occupational hearing loss. *J Occup Health Safety – Aust NZ* 2, 204-209.
- Macrae, J.H. 1988. *Improved procedure for determining percentage loss of hearing*. NAL Report No. 118, National Acoustic Laboratories, Sydney.
- Macrae, J.H. 1997. A discussion of the Australian national standard for occupational noise. *Acoustics Australia* 25, 109-112.
- National Occupational Health and Safety Commission 1993. *Occupational Noise: National Standard and National Code of Practice*. Australian Government Publishing Service, Canberra,.
- Waugh, R.L. 1997. Hearing is believing: Recent developments in Australian occupational noise policy. *Complete Safety Australia* 1, 28-31.

Longitudinal Study of inner ear damage in an Orchestra

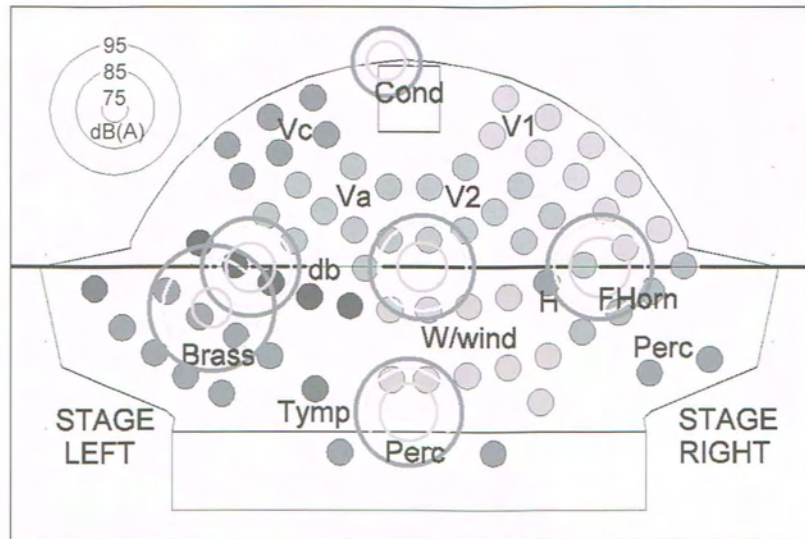
Investigators: Narelle Murray, Eric LePage
with Ken Mikl (WorkCover NSW)

Background: Since late 1992 NAL has been conducting a longitudinal study of the hearing of members of the Australian Opera and Ballet Orchestra who play in the orchestral pit at the Sydney Opera House.

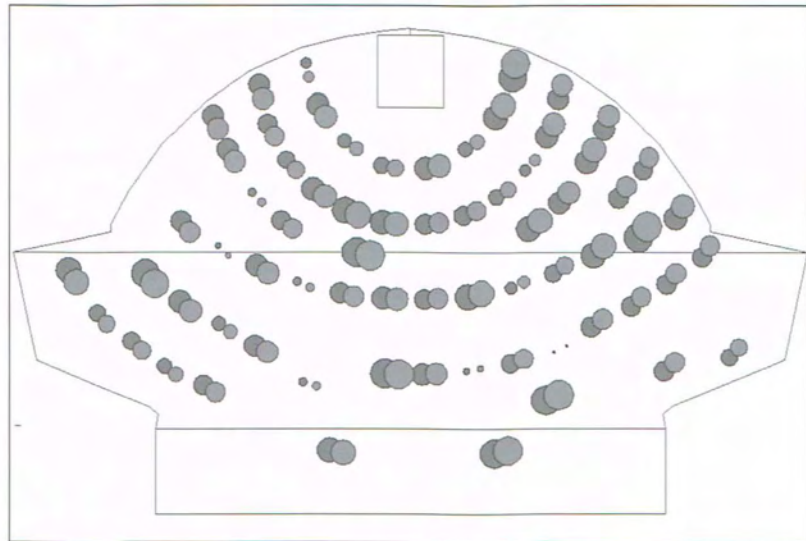
Research Question: By testing with click-evoked otoacoustic emissions and comparing the results with subjective testing of hearing thresholds with pure tone audiometry, can we establish usable parameters to determine how well the hearing of these orchestral musicians survives over a period of five years?

Research Procedures: We have tested 119 members to this point with 74 having been tested on more than one occasion by the objective method of click-evoked otoacoustic emission testing. Pure tone audiometry has been carried out each year by staff of WorkCover (NSW). Each year all musicians completed a questionnaire on their aural health, length of time they had been a musician, length of time in an orchestra and their hours of music exposure per week in performance, rehearsal, private practice and playing at other venues. The questionnaire also assessed other factors previously associated with hearing loss, e.g. tinnitus, antibiotic and anti-inflammatory use, smoking and recreational noise exposure.

Findings: From the five-year data collection results of click-evoked otoacoustic emission testing and pure tone audiometry continue to be evaluated to assess inner ear damage in a moderately noise-exposed population. One interesting result thus far is from a comparison of sound levels (taken by WorkCover) (figure, top right) at six microphone positions throughout the orchestra with Waverepro% otoacoustic emission results (figure, below right) and also with pure tone audiometric hearing levels (figure, overleaf). (These were taken as the maximum of 4 and 6 kHz for each desk and as representative of the most

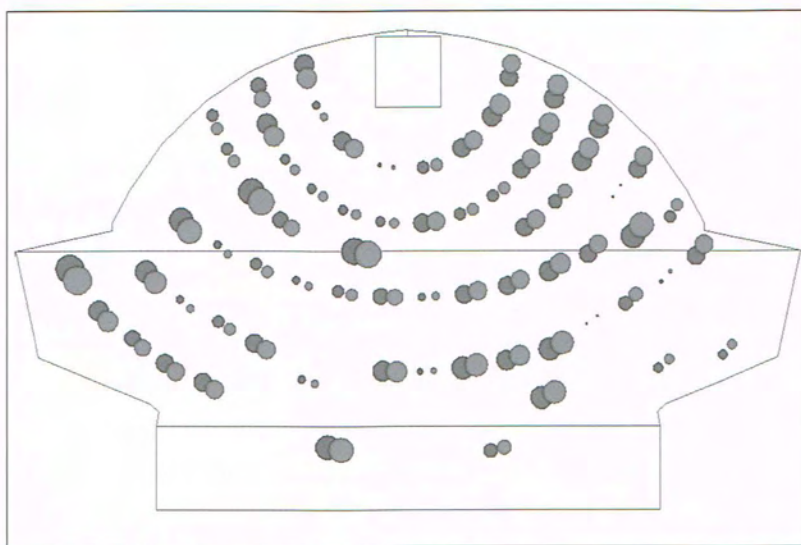


Sound levels recorded at the six microphone positions in the orchestra during performances over one season relative to player positions. The inner circles represent an average of 75 dB(A), while the middle circles are 85 dB(A) and the outer circles 95 dB(A) on average. The instruments are abbreviated as follows: Perc.=Percussion; Tymp.=Tympani; Brass – includes Trumpet, Trombone, Tuba; FHorn=French Horn; H=Harp; W/wind=Woodwind and includes Flute, Piccolo, Clarinet, Oboe, Bassoon; V1 and V2= 1st and 2nd Violins; Va=Viola; Vc=Violoncello; db=Double Bass, while Cond.=Conductor. The dark horizontal line represents the edge of the stage.



Average Waverepro% for all players occupying each desk over the five years. The darker circles represent the left ears, the lighter circles the right ears.

... it is believed that, with refinement of some of the parameters, otoacoustic emissions will be an important adjunct to hearing loss prevention programs, particularly for individual counselling of those working even in moderate levels of noise.



Average hearing levels (maximum 4, 6 kHz) for all players at each desk over the five years. The darker circles represent the left ears, the lighter circles the right ears.

sensitive frequencies indicating the commencement of a noise induced hearing loss.) By averaging the size of the emissions for all players who sat at each desk it has been revealed that those individuals who sat in the "hot spots" of the pit, i.e. in the areas which had recorded the loudest sound levels such as in front of the brass and percussion, appear to exhibit lower values of Waverepro%. The range of values, extending from 98 per cent down to 20 per cent is indicated by the size of the circles. In a similar manner there appears to be an even more significant effect of seating position on pure tone hearing levels. In this case the larger the circle the better the average hearing level at that desk. These effects are evident despite that fact that there is a considerable turnover in orchestral membership from year to year and within any opera and ballet season the players tend to play "musical chairs".

Significance: Further investigation including analysis of high and low frequency components of both the emissions and pure tone thresholds is being undertaken as it is believed that, with refinement of some of the parameters, otoacoustic emissions will be an important adjunct to hearing loss prevention programs, particularly for individual counselling of those working even in moderate levels of noise.

References:

- LePage, Eric L., and Murray, Narelle M. 1993. Click-evoked otoacoustic emissions: comparing emission strengths with pure tone audiometric thresholds. *Aust. J. Audiol.* 15, 9-22
- Murray, Narelle M. and LePage, Eric L. 1998. Otoacoustic emissions and Hearing Loss Prevention in an Orchestra. National Hearing Conservation Conference, Albuquerque, NM. February 1998.
- Murray, Narelle M., LePage, Eric L. and Tran, K. 1997. Repeatability of click-evoked otoacoustic emissions. *Aust. J. of Audiol.* 19, 109-118
- WorkCover Authority, Acoustic Services, 1992. Noise Hazard Assessment Australian Opera and Ballet Orchestra, Londonderry, NSW

Watching the ear age – an examination of otoacoustic emission cohort data

Investigators: Eric LePage and Narelle Murray

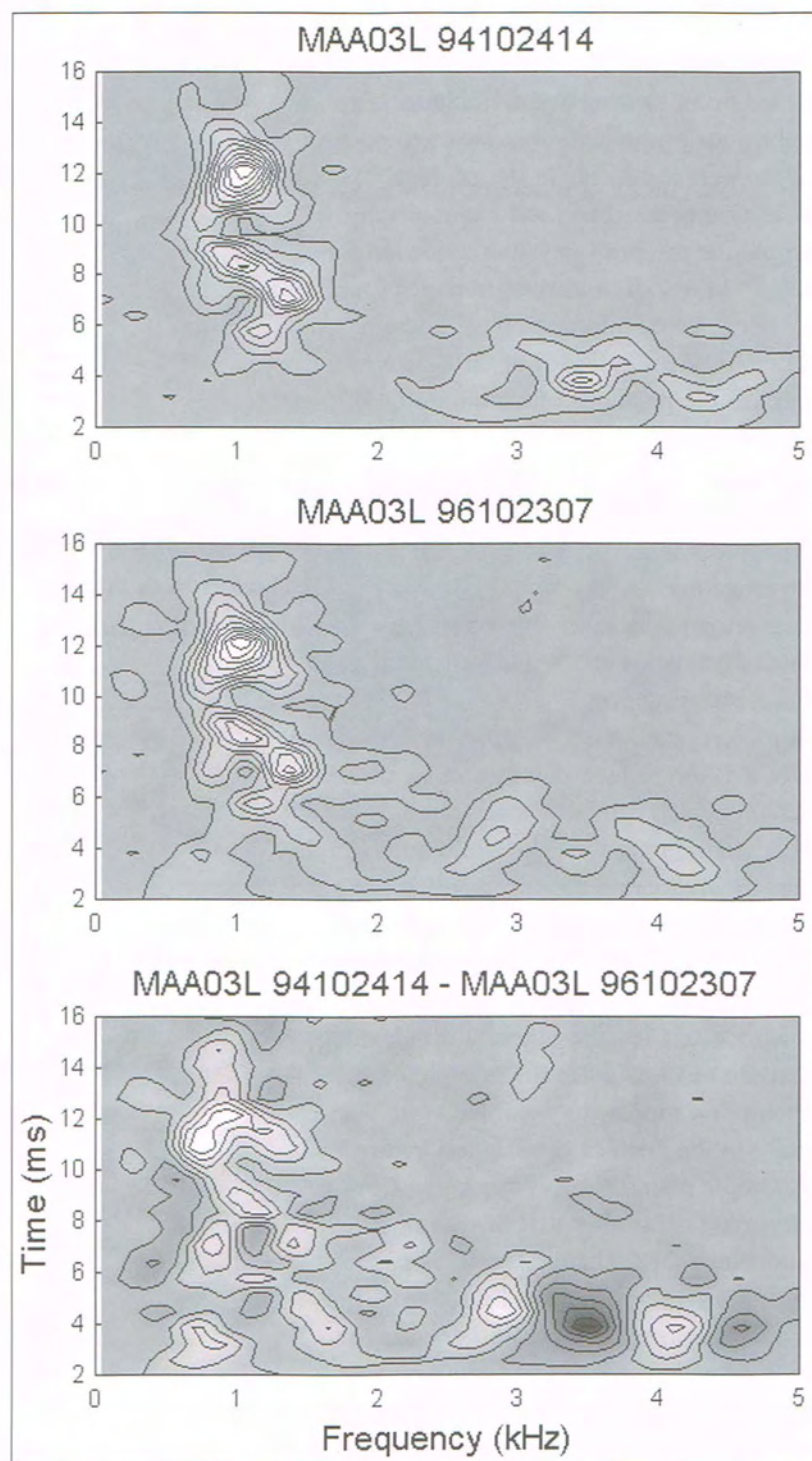
Background: During development the cochlea attains its full frequency range by maturation of the mechanical structure (Pujol et al, 1991; Morlet et al, 1993) and the development of tension in the fibres of the basilar membrane (LePage, 1990; LePage et al, 1997; Allen, 1997) and with it high frequency hearing. The working hypothesis is that the aging process is one of degradation of cochlear mechanics – the normal maturation process in reverse. If so this may be registered as a progressive rise in the size and number of scattered lesions in which the local tension of the basilar membrane breaks down resulting in dislocations in the ordering of the tonotopic map of activity. The high frequency regions (being regions of high tension) are expected to be affected first and the end result is the “ski-slope” characteristic in the pure tone audiogram describing the loss of high frequency reception.

In the past the only way to obtain objective, moderately precise measurement of these kinds of processes was by direct invasion of the cochlea in complex live animal experiments using a variety of probes or scanning laser microscopy, or alternately by tedious studies of the morphology via microscopy in sacrificed noise-exposed animals. With the advent of otoacoustic emissions, however, we have a non-invasive window into the electro-mechanical processes of the human cochlea (Norton et al, 1991) so it follows we should be able to track the accumulation of damage in the preclinical phase. The object of this project is to utilize ear emissions to track individual changes over time in a human cohort and in so doing, discover how the degradation develops and spreads and how much human hearing is affected during this aging process.

The two primary methods of carrying out otoacoustic emission measurements are both based upon looking at the nonlinear behaviour of the emission due to the activity of the outer hair cells in the cochlea. The distortion product method examines the amplitude the primary cubic distortion tone ($2f_1-f_2$) in response to two primaries f_1 and f_2 . The spectral information is generally taken to be an indicator of OHC activity local to the positions of maximum OHC activity the two primaries. While some commercial equipment is investigating delivering multiple sets of primaries at once, the resultant distortion product plot is taken to be a measure of local activity which can be related to the behavioural pure tone threshold audiogram. Like an audiogram the “dp-gram” is not a spectrum in the sense that the responses at all frequencies would be continuously emitted. The distortion product methodology has the inherent disadvantage that the result is generally interpreted that each point represents a particular unique place in the cochlea determined by the tonotopic map. In reality we know from physiological studies that this is not strictly true. Since OHC change length as part of their primary operation, all OHC from the whole length of the cochlea generate low frequency components so low frequency emissions need to be interpreted with great care. In practice low frequency emissions (<1kHz) are not well behaved, consistent with the idea that their site of origin is not “place-specific” in the way that high frequency emissions are generally believed to be.

The click-evoked method of obtaining otoacoustic emissions have some relative advantages and disadvantages relative to distortion products. A key disadvantage is

The working hypothesis is that the aging process is one of degradation of cochlear mechanics – the normal maturation process in reverse.



Time-frequency representations of the ears of an orchestral player measured two years apart. The above panels are for left ear, the opposite panels (far right) for the right ear. The top two panels for each ear are for the raw data and the bottom panel is for the difference showing that over the two years a decrease in high frequency emissions (left ear) and a decrease in the ringing time (right ear).

that the input cannot be so tightly controlled as for distortion products. The best one can hope for is to try to ensure that the stimulus spectrum has a flat frequency response function by placement of the probe. On the other hand the click being a broad spectrum stimulus means that the cochlea is receiving all frequencies at once so that the emission is a reflection of all interactions taking place in the cochlea. We can validly speak of an emission spectrum. Moreover, since the click emission has a useful time waveform it reflects dynamic behaviour of the active process we examine this behaviour in great detail, whereas the temporal behaviour associated with distortion products is normally ignored completely. It seems that this temporal behaviour may have great significance for using otoacoustic emissions for diagnostic purposes. Whereas the choice of the rate of scanning the cochlea using distortion products is somewhat arbitrary but chosen to register a final settled value of distortion product amplitude, the click evoked emission effectively scans the cochlea at the rate determined internal to the cochlea by the travelling wave. Like with distortion products, for screening applications it suffices to consider only the final averaged signal at the end of the sampling period. However, one of the real advantages of beginning with a time waveform is that the Short-Time Fourier Transform can be used to obtain estimates of the spectrum for different latencies following the click. A time-frequency display can be produced to show contours of the power in the otoacoustic emission response to the click after the incident click itself has decayed leaving only the response.

Research questions: If otoacoustic emissions can provide some measure of early warning of the onset of hearing loss, what critical features of the electromechanical process are the emissions detecting?

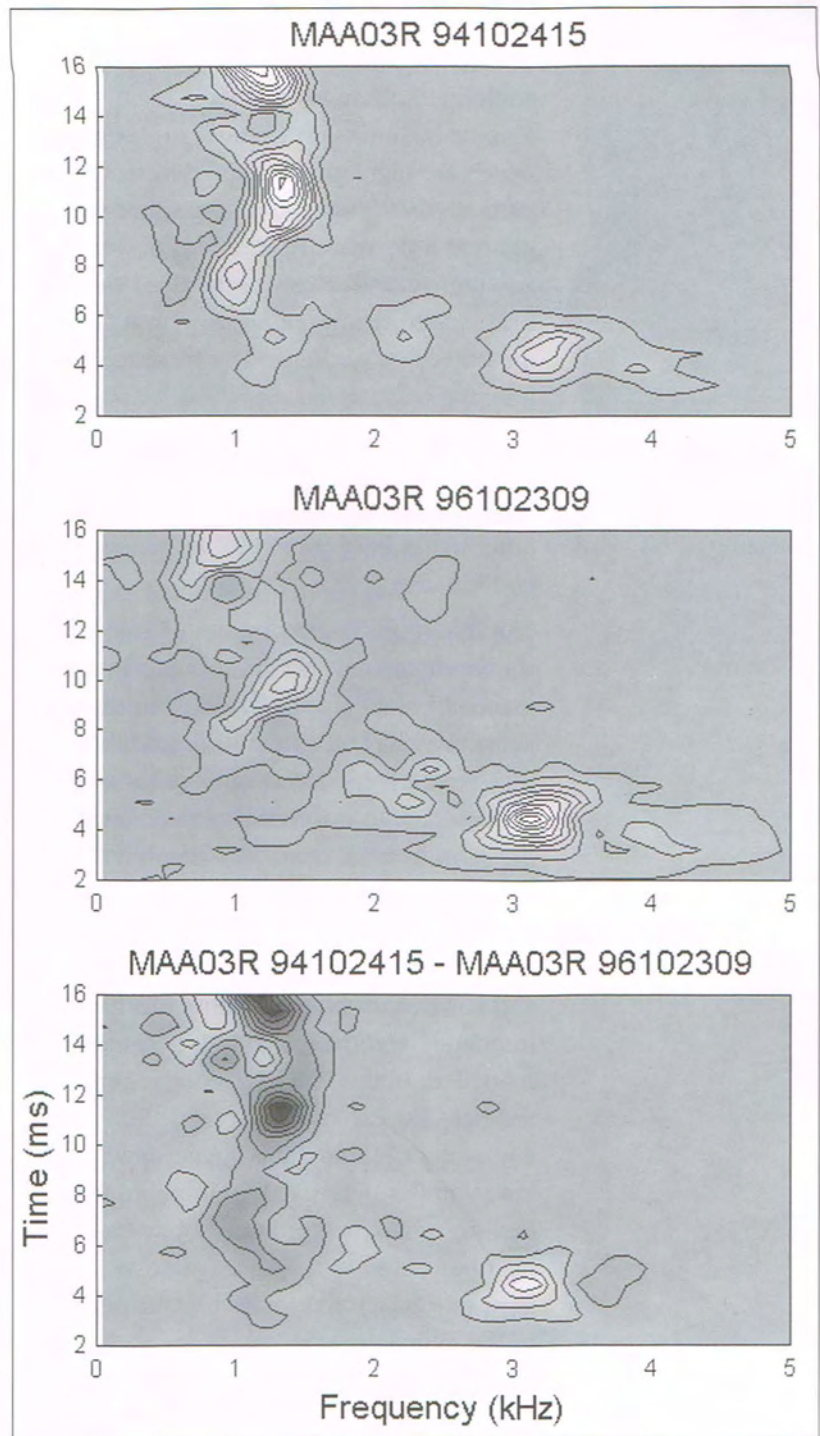
1. How sensitive are otoacoustic emissions to minute changes in the pattern of hair cell activity over a period of time?
2. Considering that emissions provide a "foreshortened view" of the activity in

which the precise point of origin of any component is not known, how do the changes due to aging present?

3. What accumulation and pattern of such lesions are necessary for a change in behavioural hearing?

Research procedures: This project has two phases. The first is concerned with analysing the results of two cohort series, the first are a group of people who have been readily available to our research group for repeated measures over nine years using the ILO88 click-evoked emission analyser. The second group is the data obtained from the orchestral player group in which players have been monitored yearly for five years 1992 to 1996. This group has been subject to many of the changes of other studies on the same people such as highly variable attendance at yearly assessments. However, there has been more than enough data to gain much insight. The second phase concerns generation of new data new apparatus developed in-house for the purpose.

Progress: The figure shows three panels on the left representing the left ear of a male orchestral musician, and three panels on the right representing the right ear. In each case the abscissae are frequency of the emission component (over a subinterval of 128 of the 512, 40 μ s time bins) and the ordinate is the time to onset of the time interval stepped in 8 bin (320 μ s) steps. For each ear the two top panels show emissions from October 1994 and October 1996 respectively. Each ear displays a response pattern which is remarkably similar despite the intervening two year period. In these plots the contours represent linear power so only the highest peaks are shown. Indeed the click-evoked emissions for any particular ear are normally very reproducible and the upper two plots suggest a fingerprint for each ear exists in the time-frequency domain. The lowest panels in each case represent the differences in the time frequency response, highlighting changes which may be very subtle to detect in the upper plots. Mid grey represents zero difference, while white represents regions of increased response over the two year span.



The figure opposite left shows three panels representing the left ear of a male orchestral musician, and three panels above representing the right ear.

Two features are worthy of mention and illustrate the objectives of the study – to characterise how the ear is aging.

However, even over a two year period during which there are no significant audiometric changes there are some remarkable emission changes in this player which are highlighted in the difference plots. In the difference plot black regions indicate a decrease in emission power at any particular frequency and time. Two features are worthy of mention and illustrate the objectives of the study – to characterise how the ear is aging. For the left ear it is seen that there are two prominent peaks at 3.4 and 4.3 kHz. In the middle panel these peaks have declined in power and two other peaks have grown to prominent at 2.9 and 4.2 kHz.

The difference display in the lowest panel shows adjacent peaks and troughs in a manner which has suggested to us that these peaks may have a common origin and that they have moved to lower frequencies consistent with our idea that the activity has the same general characteristics, but that in the intervening period, the basilar membrane tension has declined. This kind of behaviour is repeated for the right ear and has been seen throughout our database frequently and this behaviour is being modelled. In the low frequency region for the right ear exhibits a strong decline in emission at 11 and 15ms suggesting that the emission has decreased in duration. We are investigating whether these subtle changes may be related to the kinds of noise exposure which this subject has experienced in the intervening period.

Significance: Considering that these otoacoustic emission records are obtained typically over 1 minute per each, it is seen that they contain vastly more information than an audiogram. Whereas an audiogram is essentially an untimed test, this method of displaying the ear's response gives much detail not just about which frequencies are contained in the response, but indeed the speed of reaction of the ear. In this sense the otoacoustic emission test is providing some of the kinds of information of a

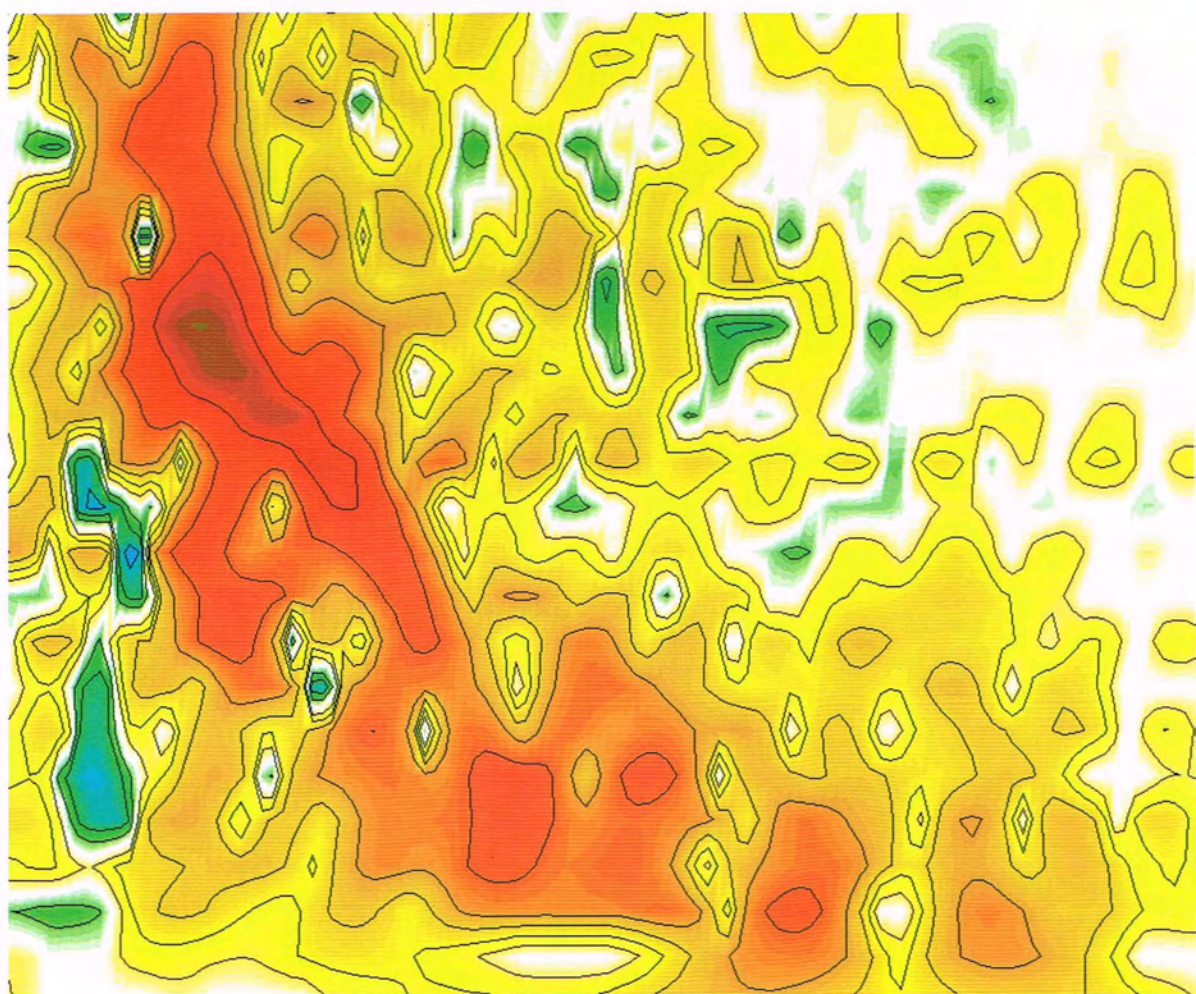
speech discrimination test, but it does so by objective measurement. In particular the data so far analysed tend to support the notion that with aging, perhaps accelerated by noise or music exposure, the ear is winding down in electromechanical power and speed. High frequency activity is being replaced by low frequency activity (left ear), while the ringing time of the emission is also decreasing (right ear). Various methods are being employed in this study to compress these complex data down to some subset of information which will prove to be specific indicators of risk for hearing loss for individuals.

References:

- Allen, J. B., (1996). OHCs shift the excitation pattern via BM tension. In: Diversity in Auditory Mechanics, edited by E.R. Lewis, G. R. Long R. F. Lyon P. M. Narins C. R. Steele and E. Hecht-Poinar., World Scientific Publishing, 167-175.
- LePage, E. L., (1990). Helmholtz revisited: direct mechanical data suggest a physical model for dynamic control of mapping frequency to place along the cochlear partition. In: The Mechanics and Biophysics of Hearing, edited by P. Dallos, C. D. Geisler J. W. Matthews M. A. Ruggero and C. R. Steele, University of Wisconsin, Madison, WI, June 25-29, 1990, 278-287.
- LePage, E. L., Murray, N. M., Tran, K., and Harrap, M. H., (1997). Excitation Pattern Shifts: Time-frequency Evidence From Click-evoked Otoacoustic Emissions. In: Diversity in Auditory Mechanics, edited by E.R. Lewis, G. R. Long R. F. Lyon P. M. Narins C. R. Steele and E. Hecht-Poinar., World Scientific Publishing, 233-240.
- Morlet, T., Collet, L., Salle, B., and Morgon, A., (1993). Functional maturation of cochlear active mechanisms and of the medial olivocochlear system in humans. Acta. Otolaryngol. (Stockh). 113, 271-277.
- Norton, S. J., Bargones, J. Y., and Rubel, E. W., (1991). Development of otoacoustic emissions in gerbil: evidence for micromechanical changes underlying development of the place code. Hear. Res. 51, 73-91.
- Pujol, R., Zajic, G., Dulon, D., Raphael, Y., Altschuler, R. A., and Schacht, J., (1991). First appearance and development of motile properties in outer hair cells isolated from guinea-pig cochlea. Hear. Res. 57, 129-41.



Research & Development Annual Report 1998/99



Hearing Loss Prevention and Noise Research

A major objective of NAL research is to increase understanding of the harmful effects of noise on people and to contribute to the prevention of hearing loss. NAL research that addresses these objectives is described in the following two sections.

Hearing loss prevention research has been using otoacoustic emission testing over several years. The three projects to be described concern

improving methodology for measuring and interpreting otoacoustic emissions and using such data to assess the individual risk of noise-induced hearing loss and for modelling ear damage.

The second section reports four noise measurement studies covering the varied areas of traffic noise, aircraft noise, and the occupational “noise” exposure encountered by orchestral musicians.



The Hearing Loss Prevention research team, Dan Zhou, Narelle Murray, John Seymour, Eric LePage (left to right) with visiting scientist, Åke Olofsson (far right).

Hearing Loss Prevention

Longitudinal Study of a non-noise exposed cohort comparing click-evoked otoacoustic emission techniques and pure tone audiometry

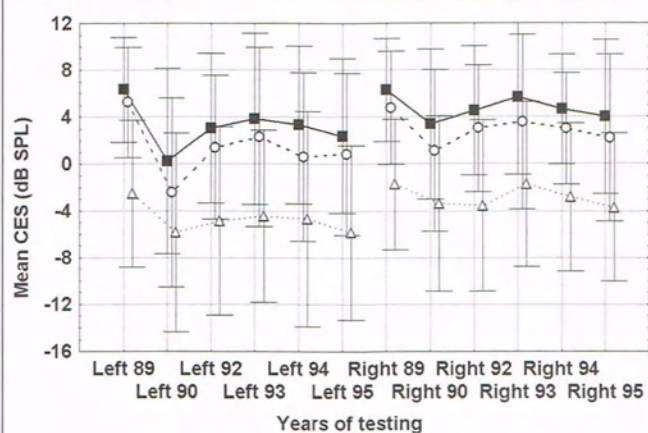
Investigators: Narelle Murray and Eric LePage

Background: Since the introduction of the click-evoked otoacoustic emission technique in Hearing Loss Prevention in 1989, the investigators have been conducting two longitudinal studies involving (a) an essentially non-noise exposed population – staff members of Australian Hearing, and, (b) a moderately noise-exposed population – instrumentalists from the Australian Opera and Ballet Orchestra (AOBO). Reports have been made previously into some aspects of the AOBO study (Murray, LePage & Mikl, 1998). This report is an assessment of some of the results from the cohort study of non-noise exposed individuals.

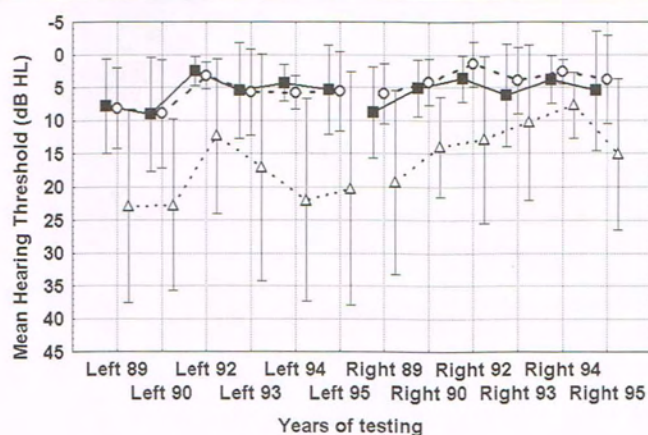
Research Question: The research question for both studies has remained the same. That is, if by objectively assessing the hearing status of individuals by click-evoked otoacoustic

emission testing, and comparing the results with subjective testing of hearing thresholds with pure tone audiometry, can we establish usable parameters to monitor the hearing status of this cohort over a period of up to 7 years.

Procedures: At approximately yearly intervals subjects have been tested with both pure tone audiometry and click-evoked otoacoustic emissions. Pure tone audiometric thresholds were established for each of the frequencies 0.5, 1, 2, 4, 6 and 8 kHz. Click-evoked otoacoustic emissions were measured using the Otodynamics ILO88 Analyser. Two hundred and sixty responses from each ear were averaged at a constant “nonlinear” stimulus level of 80 (± 1.5 dB) peak SPL. The minimum acceptable stability of recording was set at 80%. At each visit subjects completed a detailed questionnaire which assessed their past and present aural health and other factors which have previously been associated with hearing loss, e.g. tinnitus, antibiotic and anti-inflammatory use, smoking and recreational noise exposure. Because of the nature of a longitudinal study, some subjects dropped out because of change of employment, while others were unavailable for testing in certain years. Only those subjects who had a minimum of two test sessions, and, if only two,



Results are shown for each of Broadband – solid squares, Hipass – open triangles and Lopass – open circles – CES values. These are the Mean and ± 1 SD for left and right ears for each of the years testing was carried out.



PTA results are shown for the mean of 1,2,4 kHz – solid squares, 6 kHz – open triangles and the mean of 0.5,1,2 kHz – open circles. These consist of the Mean and ± 1 SD for left and right ears for each of the years testing was carried out.

those sessions at least two years apart, have been selected in any analysis of the Cohort. This accounts for the gaps in the data for some years. Out of a total of 47 subjects tested over the period of the study thus far, analysis has been undertaken on 24 males and 8 females.

In considering the data statistical analysis was undertaken on three parameters: Otoacoustic emission (OAE) Waverepro%, OAE Coherent Emission Strength (CES dB SPL) and Pure Tone Audiometry (PTA). As the study is longitudinal, interest has been focussed on changes which have occurred in all parameters during the life of the study. Results for each of the OAE parameters were calculated on the basis of (a) all frequency bands (referred to as Broadband), (b) frequencies >2000 Hz (Hipass) and, (c) frequencies <2000 Hz (Lopass). The most appropriate PTA comparisons with these OAE frequency bands were felt to be the mean of 1,2,4 kHz, 6 kHz and the mean of 0.5,1,2 kHz respectively.

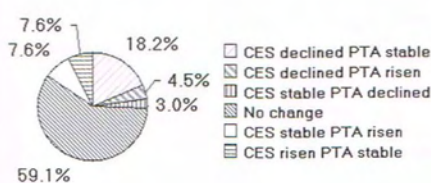
Findings: The results of ANOVA for each of the three parameters of both Waverepro% and CES showed no significant variations between years for either left or right ears ($p < 0.05$). Similarly, ANOVA for the three PTA frequency bands selected showed no differences

either between ears or between years ($p < 0.05$).

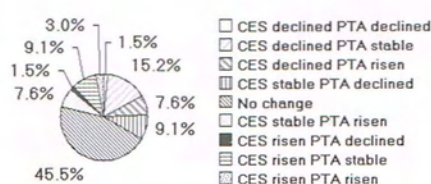
The two figures opposite left show results for each ear and each year for CES and PTA respectively. It does appear that, despite there being so little change in any of the parameters measured, CES does identify more ears with significant changes over time (figure below). CES also appears to identify many more ears with low emissions which still have pure tone thresholds within normal limits. (Murray, N., LePage, L. & Tran, K., 1997; Murray, N., 1999) CES would, therefore, seem to be a better early warning indicator than PTA of those ears which are more vulnerable to early ear damage.

Although there is little change over time in overall group data, there are wide variations in results, with "improvements" as well as declines being observed in both OAE and PTA results. While any "improvement" may be deemed a learning process in the test-retest situation of the subjective pure tone audiometric testing, this could not be applied to the results of otoacoustic emission testing. However, it is possible that these widely variant, almost random, results may be consistent with a system which is nonlinear and not at all well ordered. Gold (1989) first predicted this disorder and indeed, Shera and Zweig (1993) and Wit et al

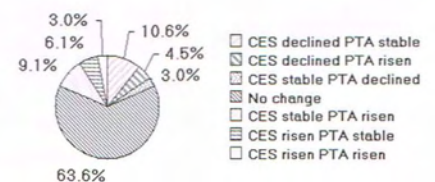
Cohort (1) Broadband CES vs PTA 1,2,4 kHz



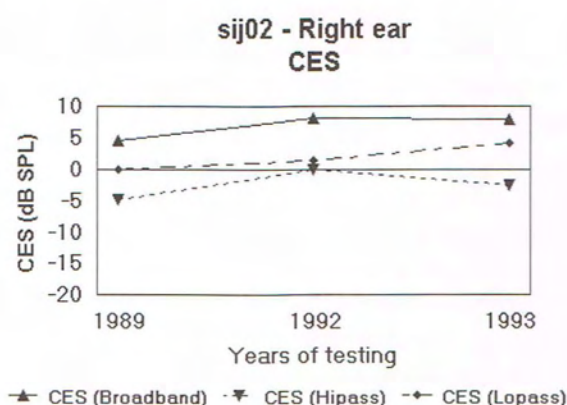
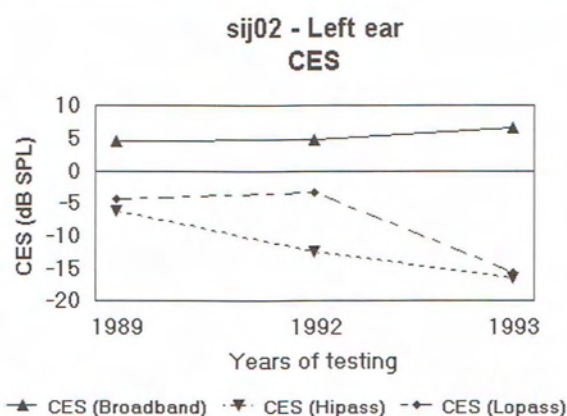
Cohort (1) Hipass CES (>2000 Hz) vs PTA 6 kHz



Cohort (1) Lopass CES (<2000 Hz) vs PTA 0.5,1,2 kHz



Comparisons of significant changes occurring over time between the three CES parameters (a. Broadband, b. Hipass and, c. Lopass) and the corresponding PTA thresholds (mean 1,2,4 kHz, 6 kHz and 0.5,1,2 kHz). In all three comparisons there is predominantly no change over time. However, it can also be seen that amongst the several other variations, the largest percentage in each case is that of CES values declining while PTA remains stable indicating that CES may be of value as a better early warning indicator of hearing changes over time.

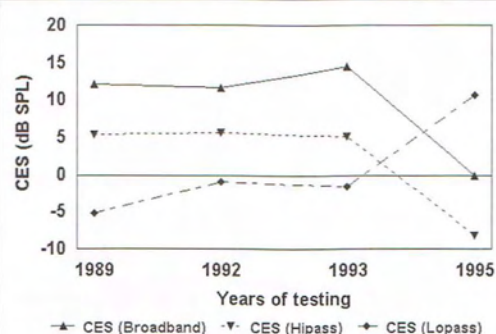


An example of binaural 'see-saw' where the trend in the left ear is downward for both hipass and lopass CES while the trend in the right ear is upward for these parameters.

One of the most interesting features of the otoacoustic emission data is what we term the 'see-saw' effect.

(1994) (both cited in Avan et al, 1996) proposed 'that a certain amount of disorder, either anatomical or functional, should be assumed to exist in the cochlea for TEOAEs to be detected with their characteristic temporal and spectral patterns'.

One of the most interesting features of the otoacoustic emission data is what we term the 'see-saw' effect. This has been observed both binaurally and monaurally. Binaurally it occurs when one ear declines over time and the other appears to compensate for this decline by 'improving' over time. This trend is apparent, although the differences are not statistically significant, in the subject in the figure above (sij02). Monaurally it can occur particularly when the high frequencies appear to decline while over the same period of time the low frequencies appear to 'improve' (figure, above) (wiw01).



An example of monaural 'see-saw'. Hipass CES falls between 1989 and 1995 while the lopass CES rises over the same period.

Significance: Analysis of high and low frequency components of both emissions and pure tone thresholds has enabled us to look more closely at the suggestion of LePage (1992) of the possibility of "permanent re-mapping of the cochlear partition in the case of OHC loss, thereby transferring the frequency representation away from regions in which there is OHC loss to regions where OHC remain". This is the most likely explanation for the monaural 'see-saw' effect. Otoacoustic emissions has allowed non-invasive investigation of this phenomenon in the human cochlea.

References:

- Avan, P., Bonfils, P., Loth, D. 1996. Effects of acoustic overstimulation on distortion-product and transient-evoked otoacoustic emissions. In: Scientific Basis of Noise-Induced Hearing Loss. A. Axelsson, H. Borchgrevink, R.P. Hamernik, P-A. Hellstrom, D. Henderson, R. J. Salvi (Eds.), Thieme, N.Y., 65-81.
- LePage, E.L. 1992. Hysteresis in cochlear mechanics and a model for variability in noise-induced hearing loss. In: Noise-Induced Hearing Loss. A. Dancer, D. Henderson, R.J. Salvi and R.P. Hamernik (Eds.), Mosby Year Book, St. Louis, Chapter 10, 106-115.
- Murray, N.M., 1999. Evoked otoacoustic emissions as a predictive factor of hearing loss. Ph.D. Thesis, submitted, Macquarie University.
- Murray, N.M., LePage, E.L., Mikl, K. 1998. Inner ear damage in an Opera Theatre Orchestra as detected by Otoacoustic Emissions, Pure Tone Audiometry and Sound Levels. *Aust. J. Audiol.* Vol. 20., 67-78.
- Murray, N.M., LePage, E.L., Tran, K. 1997. Repeatability of click-evoked otoacoustic emissions. *Aust. J. Audiol.* Vol. 19, 109-118.

Hearing Status of Aboriginal Prisoners

Investigators: Eric LePage, Narelle Murray, Tony Butler*

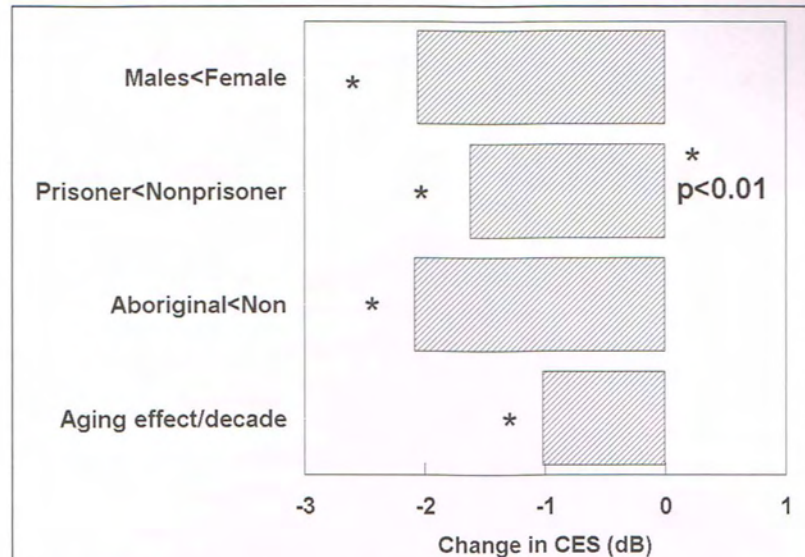
* NSW Department of Health

Background: The NSW Department of Health extended an invitation to NAL Hearing Loss Prevention Research to participate in their very extensive health study of men and women in NSW Correctional Centres by way of hearing assessment using click-evoked otoacoustic emissions.

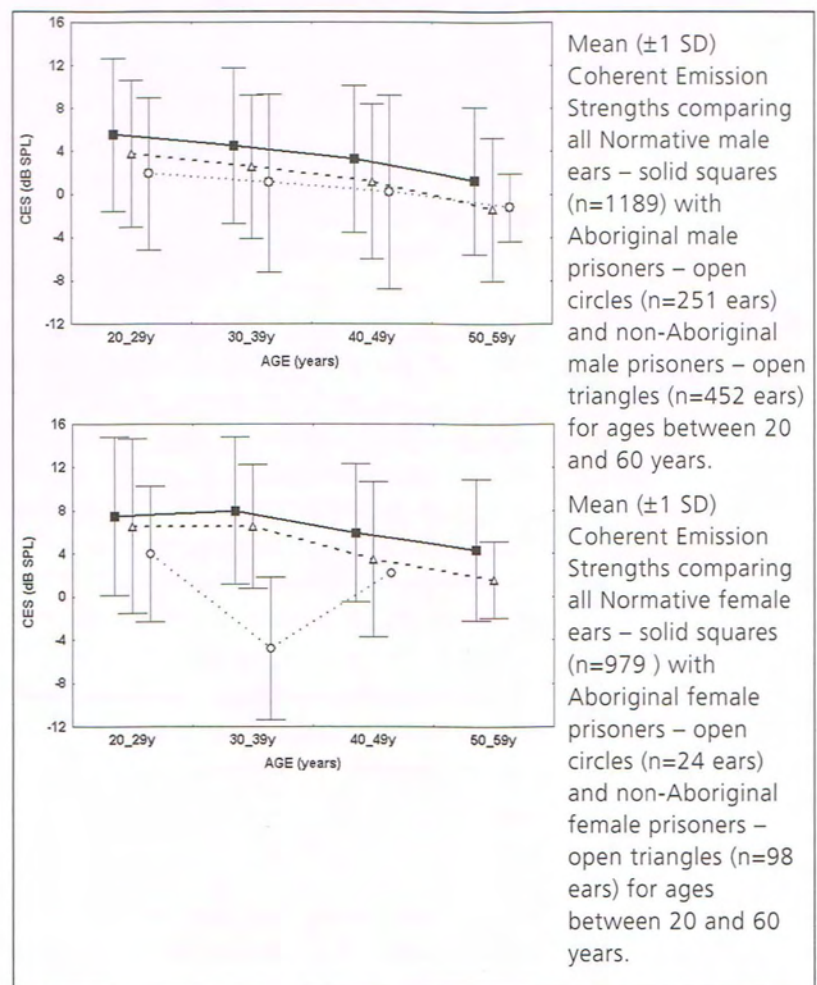
Research Question: To investigate whether the hearing of prisoners differed in any way from that of our predetermined normative Australian population. Because of the quite large population of Aboriginal and Torres Strait Island (ATSI) prisoners it was of particular interest to see if their hearing status differed a) from other prisoners and b) from the normative Australian population.

Procedures: Training was provided for the nurses in the Department of Corrective Services who were involved in the total study to carry out our standard clinical procedures for otoscopic examination and recording click-evoked otoacoustic emissions. All subjects completed a brief questionnaire which included questions relating to hereditary hearing loss, aural pathology, hearing aids, tinnitus, occupational noise, listening to loud music and head injury. Data were collected on 705 male and 122 female ears. Of these, there were 251 male and 24 female Aboriginal ears. The Analysis presented here was carried out on prisoners aged between 20 and 60 years which resulted in 675 male ears (251 Aboriginal) and 114 female ears (24 Aboriginal). The NSW Department of Health provided a breakdown of the numbers of Aboriginal and non-Aboriginal prisoners, together with other basic demographic data. Comparisons were carried out using the derived single number index Coherent Emission Strength (CES dB SPL) (LePage & Murray, 1993).

Findings: The first figure shows the result of a Multiple Regression Analysis carried out on the total data. For the age groups in question this can be interpreted that Aboriginals are more at risk of hearing impairment than non-Aboriginals, prisoners more at risk than non-



Results of multiple regression on all 3074 subjects aged between 20 and 60 years ($n=3074$ ears) showing the difference in CES values between males and females, prisoners and non-prisoners, Aboriginals and non-Aboriginals and between age groups one decade apart. The differences are those indicated by the regression coefficient.



Our data shows ... that ear functionality as assessed by otoacoustic emissions in adult Aborigines is poorer than that of the rest of the population.

prisoners and that males are more at risk than females. The second and third figures show the variations which occur between our predetermined male and female normative population for each of the four age decades under discussion and male and female Aboriginal and non-Aboriginal prisoners in those same age groups. It is not possible to determine from this study whether any hearing loss in the Aboriginal prisoners is of a sensorineural or conductive nature. Low emission strength can be indicative of a conductive hearing problem as well as a sensorineural hearing problem and tympanometry which would have elucidated this was not carried out. However, with the exception of the oldest age group where the numbers in each case are not significant, it can be seen there is a steady decline in ear damage from the normative groups through the non-Aboriginal prisoners to the Aboriginal prisoners.

From the questionnaires it also appears that Aborigines are more likely to suffer head injuries and, although not of significant difference, those with head injuries, in the main, have lower emission levels than either those Aborigines or non-Aborigines with no head injuries. This may have an important bearing on the lower emission levels in general of Aborigines in custody.

Significance: It has been well known for some time that the hearing of Aboriginal children is affected by chronic otitis media leading to conductive hearing losses. It has been assumed that there would be some legacy of this childhood affliction into adulthood. Our data shows that this is quite likely and that ear functionality as assessed by otoacoustic emissions in adult Aborigines is poorer than that of the rest of the population. It would appear that hearing health care programs aimed at helping Aboriginal children would need to be continued into their adulthood.

Reference:

Murray, N.M. and LePage, E.L. 1993. Age dependence of otoacoustic emissions and apparent rates of ageing of the inner ear in an Australian population. *Aust. J. Audiol.* 15/2, 59-70.

Modelling transient evoked otoacoustic emissions in noise-induced hearing loss

Investigators: Eric LePage and Åke Olofsson*

*Unit of Technical Audiology, Department of Ear and Skin, Karolinska Institute, Stockholm, Sweden.

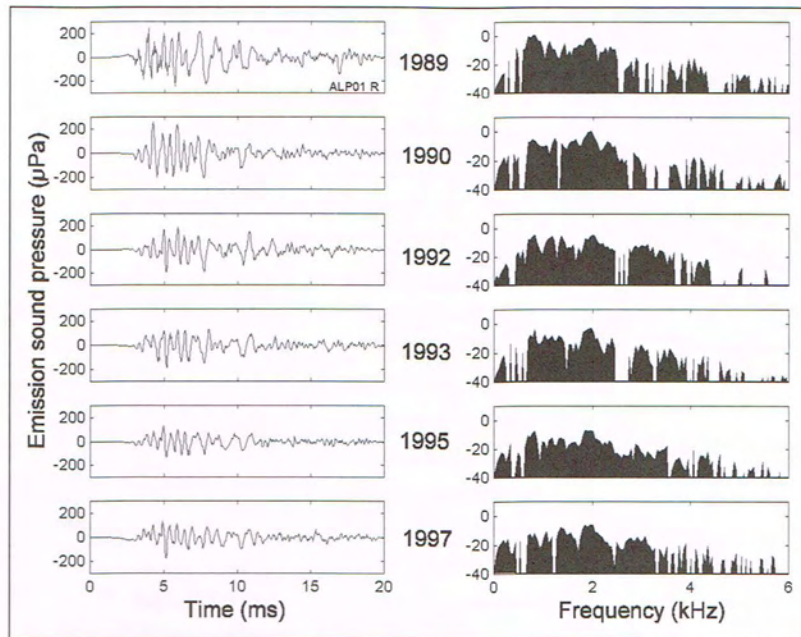
Background: Evoked Otoacoustic Emissions (EOAE) have opened up the field of audiology to provide a significant increase in the amount of diagnostic information. To limit its clinical application to determinations of emissions "present" or "absent", or test "pass" or "fail" for any particular client is merely to utilise a tiny fraction of the available information. The technique instead has enormous potential in evaluating firstly, the effects of noise exposure, both in the workplace and that due to leisure noise, and secondly, the state of function of the binaural processing of sound and the role of descending pathways. Many investigations have sought to understand the relationship between audiometric thresholds and emission spectra. At NAL we have shown that EOAEs complement the audiogram, providing a measure of outer hair cell motor performance which anticipates changes in the audiogram (LePage and Murray, 1993). This means that EOAEs also have the potential to offer new precision in the fitting of hearing aids, particularly in cases of uncertainty.

There exist two main types of EOAE test, transient evoked emissions (TEOAE) and distortion product emissions (DPOAE). Distortion products have the advantage that they are generally carried out at audiometric frequencies mostly only up to 8 kHz. The transient approach delivers the information as a spectrum across a range of frequencies. In young individuals this range extends to above 4 kHz, but in adult males emissions are present mostly only below 2 kHz. This presents a puzzle for audiologists because such people may be hearing frequencies which can still extend well above 4 kHz. Evidently the transient method is not so easily correlated with the audiogram. This is no inherent disadvantage, however, because the type and amount of information which TEOAEs offer is much larger. Our database contains around 13500 records and

many of these are from repeat measurements on the same people, over a ten year period. From observations of their emission spectra we have concluded that, just as the frequency range of normal sensitivities for the audiogram reduces over years, the bandwidth of the emissions steadily reduces, but earlier. This progressive reduction in bandwidth is directly related to the ski-slope feature in audiograms of aging ears and it is clear that OAEs can tell us much about the origin of the aging process in humans.

Australian Bureau of Statistics data shows that our population is living to a greater age. Since many of our youth are showing prematurely low emissions, this suggests our ears can age relatively much faster than the rest of the body. Taken together, the differential between chronological age and ear age would appear to be increasing. Our research is therefore directed at extending our declining lifetime of normal hearing with all the attendant costs. Part of effective hearing loss prevention must be tied to education. The other major component has to be the availability of early warning. Our research suggests that OAEs have the potential to provide early warning by utilizing the concept of "ear age" thereby bypassing all the complexities of communicating hearing awareness for the average young person prone to the damage due to noise exposure. It would be very useful in promoting hearing loss prevention to be able to say, "You may be 25 years old, but your ears are responding as if they are 45 years old". However, as things stand at present we have no simple error free way to measure the lifetime of normal hearing but we are making progress. We need to be able to refine our measures of OAE response to take into account normal aging (the process leading to presbycusis), gender, and changes which take place in the ear which are not immediately transparent from observing audiograms.

The emission responses change with time which are not a simple decline across all frequencies. The first figure shows the right ear of a male subject measured over an 8 year span. The series of six records, shows not just a general decline in the size of the emission. The size and shape of the time waveform is systematically changing, resulting in subtle changes in the shape of the emission cross-power spectrum. Examining the records shows that the relative



heights of the main peaks are changing, while other areas drop out completely and then return. The result illustrates the significant problem of deciding how much are changes due to measurement error or to the presence of noise on the emission signal, and how much is due to a real aging effect upon the ear. Secondly, are the changes in the peaks of the spectrum indicative of changes in the decline in outer hair cells? If so, do the changes represent a map of the degradation?

Whereas the early OAE literature reported how click emission waveforms are like a "fingerprint" for an ear, our data show that over time, as one would expect with progressive loss of OHC throughout life, the reproducibility over time degrades. It is clear therefore that to make better use of the OAE information we need to understand what are the underlying changes which bring about changes in the appearance of the transient emission.

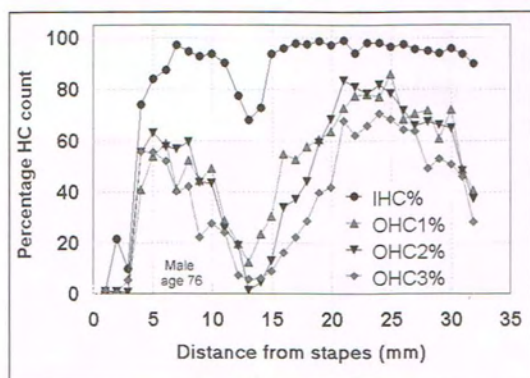
There are several approaches to this. One is to collect data over a long period (see our accompanying longitudinal study [Murray, *et al*]) and look for patterns which are common to very many people e.g. those with similar backgrounds or noise exposure profiles. The subsequent recognition of patterns may, however, provide little insight as to actual hair cell pathology. Another is to obtain otoacoustic emissions in live animal ears and correlate the cochlear histology post-mortem with previously measured emissions such as in the second

Transient otoacoustic emissions and their spectra for the right ear of subject ALP01 over an 8 year period.

It would be very useful in promoting hearing loss prevention to be able to say, "You may be 25 years old, but your ears are responding as if they are 45 years old".

Our research monitoring emissions in the same people over the past decade has shone light on the nature of the aging process...

figure; this is beyond the scope of in-house NAL research. A third way is to utilise mathematical models of cochlear micromechanics and, using a computer, simulate the effect of specific lesions of the OHC population by modelling decline in OHC motility and total loss of the cell bodies. The model allows us to do that as a function of their location by simply changing the size of the variable and observe the effect upon the emissions. The model does not necessarily allow us to create a map of damage because there may be an infinite number of configurations which could result in a particular emission picture. The model, does, however, allow us to simulate how a particular type of damage (e.g. tonal or impact noise) may affect the TEOAE. Hence we can at least use the model to build up a picture of classes of behaviour which occur with damage. In pictures depicting ear damage in animals exposed to loud noise, it is common to observe scattered loss of OHC, but for larger exposures significant patches of damage are seen which may show complete loss of OHC for



1 mm or more length of the cochlear epithelium containing the hair cells. So the first question posed of the model is to "create" small or simple lesions and vary their position along the basilar membrane just to

Rare insight into aging in the human cochlea: a postmortem determination of hair cell counts (inner hair cells IHC and three rows of OHC) in a patient aged 72 years who had had some noise exposure during his life (Re-drawn from Hawkins et al, 1976).

check that the frequency affected conforms to the expected inverse linear map connecting distance from the stapes to the log of frequency and to observe the effect upon the emission of size of lesion.

Procedures: There exist a variety of models for cochlear mechanics upon which such assumptions can be tested. The objective has been to use a basic single delay line model, which allows OHC activity to modify the vibration of the basilar membrane primarily by altering its stiffness but with the possibility of modifying mass and damping as well. For this purpose it has been found advantageous to adopt the discrete time domain model (Nilsson, 1977) which has been re-formulated using the wave-filter approach (Fettweiss, 1971), by Olofsson (1982) and Strube (1985). This has been reimplemented as a Mex (.dll) file which is

called from Matlab(tm). The result is a computationally efficient, flexible modelling environment which runs under Windows and which offers advanced displays of model properties.

For simplicity the first implementation models the OHC interaction as a scalar variable (roh) which varies from 0 to 1 representing the fraction of OHC functionality from a dead cell to a fully active cell, so that lesions may be modelled as changes in roh from one segment to the next, representing the long term effects of cochlear aging or noise exposure which make it applicable to the clinical research into prevention through early warning. The model is described as a wavefilter model made up of 350 segments, representing a 35 mm long basilar membrane. Each segment represents the combined activity of 0.1 mm or about 30 OHC. Sound "enters" the cochlea model by simulating a pressure waveform at segment 1. This progressively stimulates OHC activity as waves pass down the whole length of the cochlear and returning to the stapes, the reverse waves interacting with the forward waves at each point. The model is quite realistic in that OHC activity boosts the BM at each segment. While any stimulus type can be used, several parameters of the model can be measured at each segment (viz. displacement, velocity, pressure, even neural excitation). Most importantly the pressure wave applied to segment 1 is by definition the sound stimulus passing through the middle ear while the superimposed activity, which results from the OHC response, is the otoacoustic emission. If two tones are presented, a family of distortion products are produced by the model. Likewise by delivering a click sound stimulus the result is a realistic looking transient OAEs. (third figure). The beauty of the model is that just as a clinical otoacoustic emission is the result of signal averaging, such signal averaging can be applied to the model with the same procedures used in the clinic and the results may be analysed, complete with stimulus artifact removed, and presented in exactly the same way.

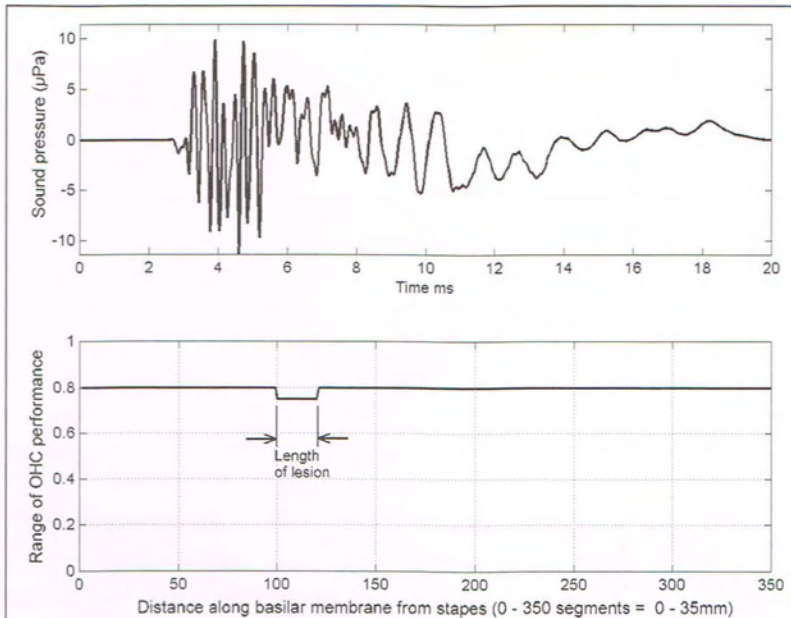
Results: Not unexpectedly, the presence of such lesions changes the frequency spectrum of the emissions significantly. The prime object of the current exercise is to observe what effect changing the position and length of single point lesions has upon the click emission spectrum. In recent times we have been utilising the short-term Fourier transform to produce time-

frequency plots of transient emissions. This particular representation allows the appreciation of both domains in the one figure. The output of the model is shown in the fourth figure. The three panels show the time-frequency representations of two lesions – one longer than the other by 50 segments, while the lowest panel shows the difference. It has been found that by changing the size of the lesions, there is a peak in the frequency response which appears to become accentuated and is actually tied in its frequency to the apical end of the lesion not the basal end (see fifth figure). The insight obtained in this way suggests that peaks in the TEOAE are due to sudden impedance changes in the cochlear partition which occur at the far end (distal to the stapes).

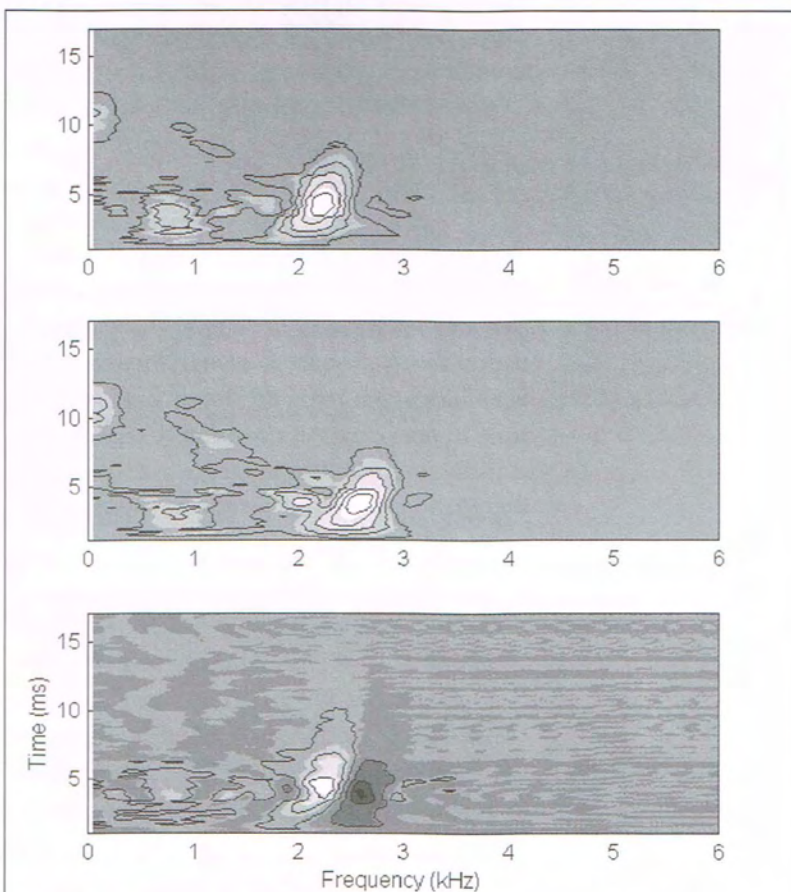
The computer model result seen in the fourth figure has been seen frequently in human OAE data without any definitive explanation. In the light of the model it can be suggested that the shifting peaks which are frequently seen and the resulting “light-dark” pigeon pairs which result in the difference plots, from one year to the next, have their origins in an increase in the size of damaged regions in their cochleas. It thus appears that following the approach which pairs real TEOAE data with model results may assist us in defining how far the idea of this type of result can be interpreted as a measure of physical damage which results from a particular kind of noise exposure.

Conclusion: In this our first application of the model artificial lesions of poorly functioning OHC are simulated and it has been found that for fixed length lesions, there is a peak in the spectrum which follows the map built into the model. In the case of variable length lesions the spectrum of the emissions exhibits a peak whose frequency varies systematically with the physical location of the apical end of the lesion with a small cumulative error. While such insight may be subject to the assumptions of the model, it was obtained with considerably less resources and ethical considerations than required to run a research program correlating hair cell cytochrome c oxidase with OAEs.

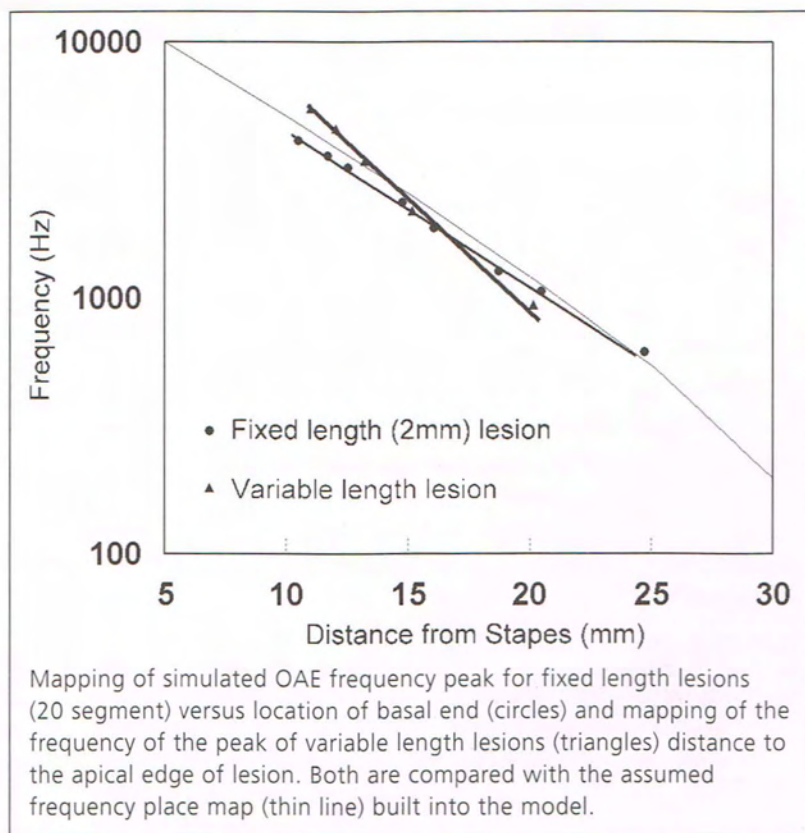
Significance: Our research monitoring emissions in the same people over the past decade shone light on the nature of the aging process and that has led to the need to confirm our descriptive model using a computational model. While such models are most typically



Simulated TEOAE for the standard non-linear stimulus paradigm for an ear with noise-type damage in which a loud pure tone has damaged the function of OHC over a short length (ca. 2 mm) of the cochlear partition. Remarkably, even a tiny change in rohc 0.8 to 0.75 produces a useful effect.



Time-frequency representations showing the time course upward of the frequency components of the emission. Two conditions of the model are shown, one with a 20 segment lesion at segment 150 and another instead at segment 200. The peak in the response is due to the presence of the apical edge of the lesion. The lowest panel shows the difference.



used to understand the normal cochlea, this is an application to the damaged cochlea and has provided insight as to how noise likely affects the ear for the first set of assumptions made. Such insight has never previously been available from human data. An extension of the idea that emission can help determine the rate of physical degradation in the cochlea is that the approach

may be useful in defining the growth of cochlear lesions due to noise or aging. Further investigation of the model in conjunction with our patient data may promote real effectiveness in prevention strategies by definitive descriptions of OHC degradation. Using the model may thus lead to a strategy for defining the effective age of any ear. In turn this may lead to a better determination of the effectiveness of hearing protection devices.

References

- Fettweiss, A. (1971). Digital filter structures related to classical filter networks. *Arch. Elek. Übertragungst.* 25, 79-89.
- Hawkins, J.E. Jr. and Johnsson, L.-G. (1976). Patterns of sensorineural degeneration in human ears exposed to noise. In "Effects of Noise on Hearing", edited by Henderson, Hamernik, Dosanjh and Mills. Raven Press, NY. 91-110.
- LePage, E.L. and Murray, N.M. (1993). Click-evoked otoacoustic emissions: comparing emissions strengths with pure tone audiometric thresholds. *Aust.J.Audiol.* 15:9-22.
- Nilsson, H.G. (1977). A model study of the auditory periphery. Dissertation from the Division of Physiological Acoustics, Department of Physiology, Karolinska Institutet, Stockholm, Sweden.
- Olofsson, M.Å., (1982). Distortion generation in a model for peripheral haircells. Nordic Acoustical Society meeting, Stockholm, Sweden, 407-410, (in Swedish).
- Strube, H.W. (1982). A computationally efficient basilar membrane model. *Acustica*, 58, 207-214.

HEARING LOSS PREVENTION

NAL-OAE1: NAL Transient evoked otoacoustic emission analysis software

Investigators: Eric LePage, Dan Zhou, Narelle Murray, John Seymour

Background: Over the last decade, NAL has developed a large database of over 15,000 transient evoked otoacoustic emission (TEOAE) records using the Otodynamics Ltd. ILO equipment. On the basis of these records a set of age/sex-normative statistics has been derived in terms of the parameters Waverepro% and the empirically derived Coherent Emission Strength (CES dB SPL) (Murray & LePage, 1993). By comparing any person's ear emission levels with those matched for age and sex in the Australian population it is possible to estimate a person's risk of hearing loss due to outer hair cell damage. A previous study (LePage & Murray, 1993) had also shown that values of CES are normally distributed and also that a critically low level of CES (~ 2.5 dB SPL), not strongly dependent upon age or sex, corresponds with the onset of a mild hearing loss (~ 25 dB HL).

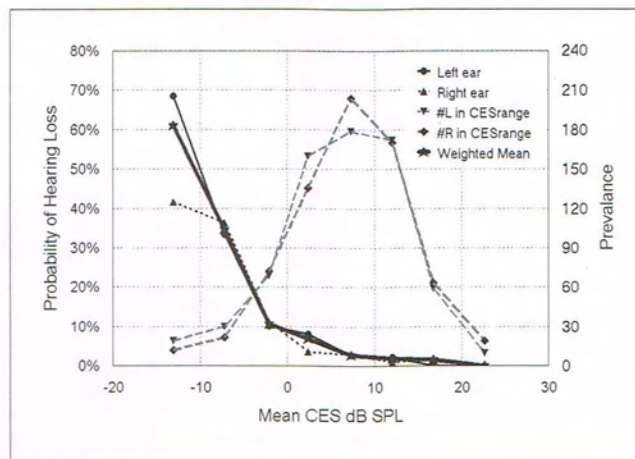


Eric LePage,
at a conference
presentation

Objectives: On the basis of our available normative Australian TEOAE data together with the associated pure tone audiometric database, to produce a Microsoft® Windows™-based software package to analyse this database and provide: 1) outcomes which allow comparison of any TEOAE record with these statistics, 2) an estimate of the probability of hearing loss assessed on the current record and, 3) an assessment of the risk of hearing loss expressed in terms of the proximity of the value of CES to the critical value.

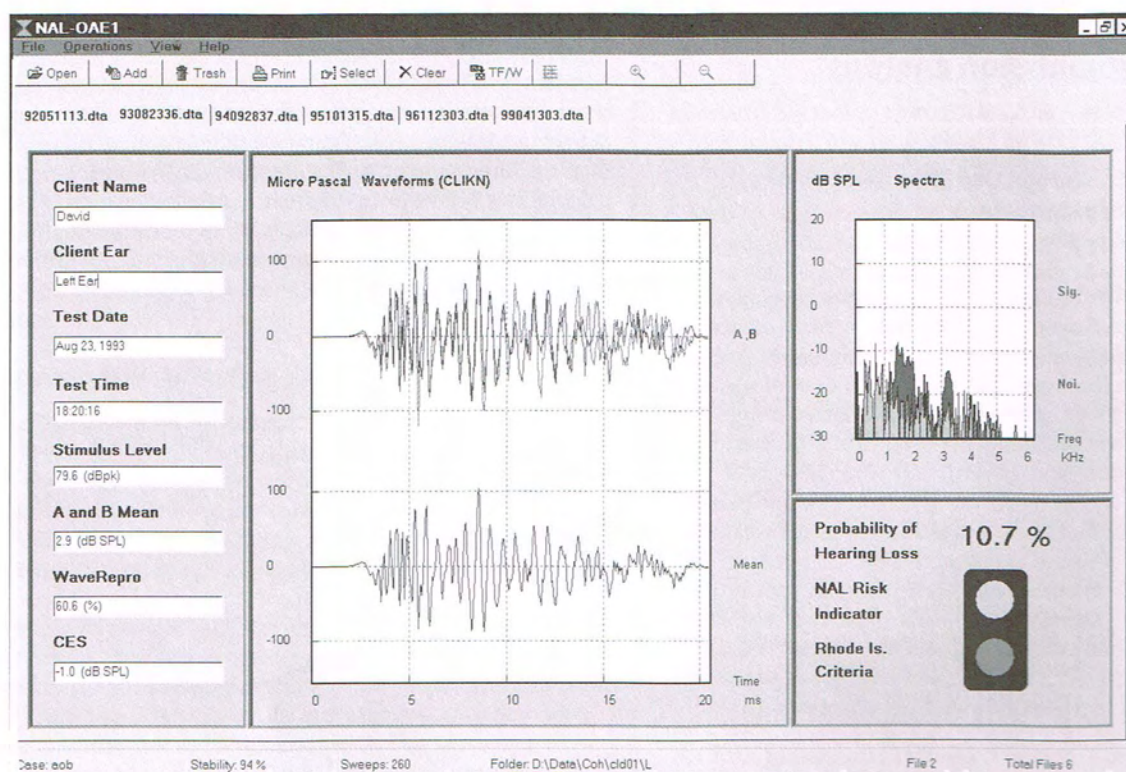
Procedures: Using the NAL database of subjects tested with both pure tone audiometry and TEOAEs (1396 ears) probabilities of hearing loss in seven equally spaced non-overlapping CES bands of 5 dB each were calculated. Within any particular band of CES the calculation is made for the probability of a hearing loss of 25 dB or greater occurring with the mean thresholds of frequencies 500 Hz, 1, 2 and 4 kHz. A "traffic light" indicator was then based on the broadband CES to give an indication of risk.

Results: The graph below shows the determination of the probability of hearing loss (%) for each of the seven CES bands. From the sample of the screen (next page), it can be seen that the essential elements from the ILO screen are retained (e.g. Response in terms of an FFT display, Timewaveform, Waverepro%, A&B Mean calculations). However, the main (or default screen) also includes CES calculation, probability of hearing loss (%) and a risk traffic light indicator, viz.: high (red), medium (yellow) or low (green).



The bell-shaped curves (upright and inverted triangles for left and right ears respectively) represent the distribution of the coherent emission strength (CES dB SPL) for the Australian population expressed as the number of cases within each of the 5dB groups (20 to 25, 15 to 20, ..., <-10 dB) from records for which we have accompanying audiometry (however, neonates have CES values extending upwards to +38 dB SPL). For each group the number of cases with a 3FA (1, 2 and 4kHz) hearing loss exceeding 25 dB HL is presented as a probability of hearing loss, again for left and right ears (open and filled circles respectively) together with a weighted mean (stars and thick solid line). The chart shows that it is possible to watch CES declining to a value near 0 dB SPL (critical value) below which the chance of hearing loss rises sharply. Hence the CES value for any record may be used as a measure of early warning, viz. proximity to the critical value.

Significance: This approach to TEOAE analysis is the first report of analysis showing the risk for hearing loss (an early warning measure) and the probability of the existence of an actual hearing loss. These dual measures of all the important features of the ILO record, provide a clear display of useful information for client education has been incorporated into a Windows(tm)-based software package. The reports from hundreds of subjects who have seen their ear data displayed in this way suggests the possibility of modification of attitude in those previously unaware of the significant hazard associated with noise exposure.



The figure shows the appearance of the main coloured display panel of the NAL-OAE1 analysis software. The program may open up an arbitrary number of standard ILO TEOAE data files using standard Windows [^](TM) operations. Each file is indicated with a tab at the top of the display panel which contains the file name. By selecting a particular file via its tab, the data are displayed as time waveforms and their spectral counterparts. The coherent part of the spectrum is displayed in blue and the "noise" signal is displayed in red (or black and white). The left panel shows the patient name entered at the time of recording, which ear, the test date, the time the file was saved, the peak stimulus level (dB SPL), the A&B mean value, the "WaveRepro" (Waveform Reproducibility %) and the NAL derived value of CES (Coherent Emission Strength dB SPL). The lower right panel displays the outcomes of the analysis, as the probability of hearing loss compared with the Australian population, the derived NAL risk indicator for people over 7 years of age (green, yellow and red, see text) and the Rhode Island criteria indicator for neonates and infants (green, yellow and red to indicate "pass", "partial pass" and "refer"). The status bar at the bottom shows other key indicators of record quality, the file source and display statistics. The program has many added features such as the ability to compare files, the ability to zoom parts of the time display and a direct comparison of the output variables for the displayed record with Australian population statistics for males and females, and for CES and Waveform Reproducibility. Any particular record display may be printed and the statistics for all the files on the NAL-OAE1 desktop may be saved in a log file for importing into a spreadsheet or database. Details of the availability of the software is available in the order form at the back of this volume, and on the Australian Hearing web page.

References:

- LePage, E.L., & Murray, N.M. (1993). Click-evoked otoacoustic emissions: comparing emission strengths with pure tone audiometric thresholds. *Aust. J. Audiol.* 15, 9-22.
- Murray, N.M. & LePage, E.L. (1993). Age dependence of otoacoustic emissions and apparent rates of ageing of the inner ear in an Australian population. *Aust. J. Audiol.* 15(2), 59-70.

Dan Zhou,
who writes the
software for
NAL-OAE1
analysis



To fit or not to fit: Preliminary Otoacoustic Emission assessment of children with severe mental and physical disabilities for consideration of hearing aid fitting

Investigators: Narelle Murray, Eric Le Page (NAL), Greg Birtles (Macquarie University, formerly of NAL), Donna Smith, Annette Smith, Lindsay Hamilton (Australian Hearing)

Background: Apart from very small infants, there are many children and adults in the community who, because of disabilities, are unable to respond in a meaningful way to behavioural tests of hearing acuity. These include those suffering from cerebral palsy, epilepsy and autism as well as developmentally delayed and multiply handicapped. Clinical audiologists regularly spend many hours behaviourally assessing the hearing requirements of these people often without a definitive result.

Research Question: Is there value in obtaining click-evoked otoacoustic emission records as a preliminary step in the process of evaluating the amplification needs for the physically and mentally handicapped?

Procedures: Over the past ten years Hearing Loss Prevention Research have been approached by Australian Hearing audiologists, the Deafness Center at the New Children's Hospital, Westmead, the Hearing Assessment Centre, the Spastic Centre of NSW and other organisations caring for those with physical and mental disabilities to assess the hearing of these people. We have now tested approximately 100 patients with the objective measurements of tympanometry and transiently evoked otoacoustic emissions (TEOAEs) in an endeavour to assess outer hair cell functionality in the cochlea. All patients were firstly given a visual otoscopic examination.

Individual Case Results

Case 1

History: Female aged 7 weeks whose parents were concerned as a result of a comment by their GP and crude "hearing" testing. She responded to noisemakers at levels of 55-75 dBA. Tympanometry results suggested normal middle ear mobility and pressure for both ears.

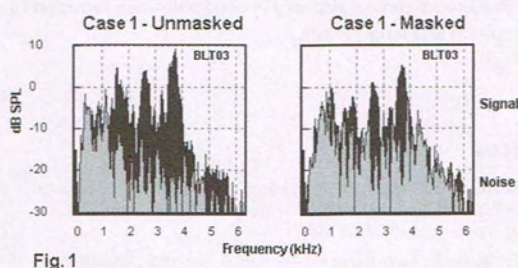


Fig. 1

Case 1: 7 week old female with outer hair cell activity within normal limits for her age; normal function of the medial efferent system. Hearing aids contraindicated. Right ear unmasked and Right ear tested with contralateral white noise of 90 dB.

Test Results: TEOAE results indicated high levels of cochlear outer hair cell activity within normal limits for her age. To test the possibility of a retrocochlear dysfunction, contralateral stimulation of 90dB (HL) white noise was applied to the left ear while testing the right ear with TEOAEs. This procedure produced a marked change (suppression) of the right ear response indicating normal function of the medial efferent (crossed) system.

Recommendation: Fitting of hearing aid/s was contraindicated.

Case 2

History: This male child was first seen in 1994 at the age of 17 months. Subsequently he was tested in 1995 and 1998. He presented with severe global brain damage, abnormal control of body temperature, no corneal reflexes, unable to cough; unable to cry, no gag reflex and unable to feed orally. He had been diagnosed as unable to hear. He was referred by the Spastic Centre for otoacoustic emission testing.

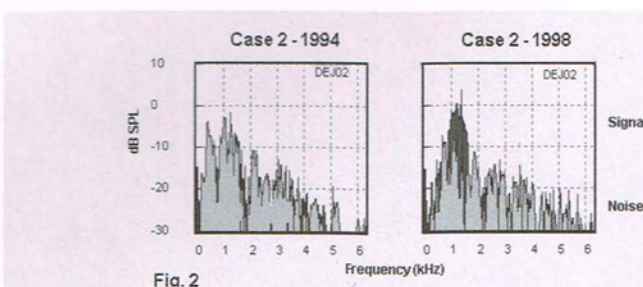


Fig. 2

Case 2: (1994) - 17 month old male with global brain damage. Outer hair cell activity well below normal limits for Australian normative males for his age. Hearing aids recommended.

Case 2: (1998) - same child now 4 years of age. Some low frequency outer hair cell activity now present. Continued use of hearing aids recommended.

Test Results: TEOAE testing in 1994 showed outer hair cell activity well below our normal limits for children of his age. While emissions were absent, there was some activity present and it was felt that sound stimulation should be encouraged.

Recommendation: It was recommended that hearing aids be fitted.

The subject was tested twice more, in 1995 and 1998. There was little change in 1995, but activity was still detected. In 1998 the right ear now showed some low frequency activity between 500 and 1500 Hz. However, since activity is absent above 2 kHz hearing aid use was continued.

Case 3

History: 14 month old female twin with cerebral palsy. There had been some parental concern re hearing. She could not be conditioned sufficiently for full Visual Response Orientation Audiometry (VROA) testing, but some behavioural responses were obtained at 80 dB SPL. Tympanometry showed normal middle ear pressure and compliance in the left ear with reduced compliance in the right.

Test Results: Results of TEOAE testing for both ears were below the normal limits for her age. The right ear showed lower emissions than the left, with sufficient outer hair cell activity in the left ear for hearing within the upper speech range.

Recommendation: Since significant emission peaks existed in the left ear, it was recommended that this child be retested within 6 months prior to any decision regarding fitting hearing aid/s.

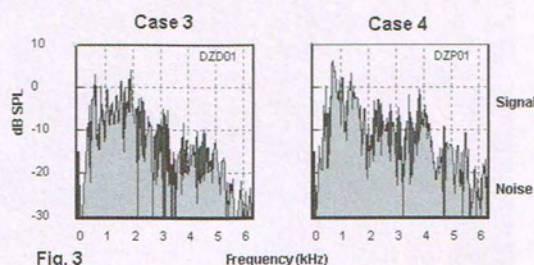


Fig. 3

Case 3: 14 month old female twin with cerebral palsy. Some outer hair cell activity in both ears - more in left than right. Recommendation for retesting within 6 months prior to hearing aid fitting.

Case 4: other 14 month old female twin with cerebral palsy, poor vision, asthmatic and developmentally delayed. Sufficient outer cell activity in both ears for high frequency hearing. Recommendation for retesting.

Case 4

History: 14 month old female twin of Case 3, developmentally delayed, with cerebral palsy and poor vision; asthmatic. There had been no parental concern re hearing and when tested she had exhibited some headturn responses to noisemakers.

Test Results: Results of TEOAE testing for both ears are below the average for her age. In this case, although the left ear shows more outer hair cell damage than the right, there appears to be sufficient outer hair cell activity in both ears for hearing in the speech range.

Recommendation: It was recommended that this child also be retested within 6 months when it is hoped that less noisy recordings (possibly due to asthmatic wheezing) could be obtained. Since partial passes were obtained for both ears the recommendation was made to wait six months and retest before considering fitting of hearing aids.

Case 5

History: 7 weeks old female infant. Gentamycin & possibly Vancomycin administered at approximately 1-2 weeks. No response to loud sounds.

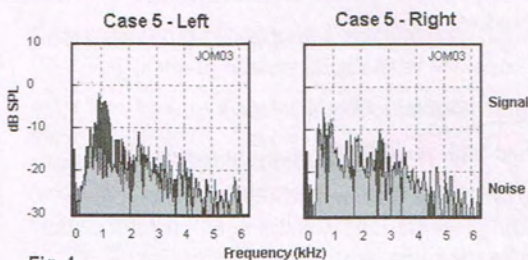


Fig. 4

Case 5: Left and Right ears of a 7 week old female who had had Gentamycin and possibly Vancomycin administered shortly after birth. Although results for both ears are significantly below normative limits there is scattered activity up to 5 kHz. Hearing aids recommended.

Test Results: TEOAE results for both ears were significantly below the average for neonates. However, both ears did show scattered activity up to 5 kHz. The main peak in the

left ear was at 1 kHz and in the right ear at 2.5 kHz. Contralateral stimulation was not carried out as the activity was considered too low. On the other hand, there seemed enough activity to suggest that the relatively high threshold Wave V in the ABR may have been an underestimate of her hearing acuity.

Recommendation: She may benefit from hearing aids set at a gain low enough to maximise preservation of existing outer hair cell activity and minimise discomfort. There should be no gain below 1.5 kHz in the left ear.

Case 6

History: 14 year old female with delayed development; resident of a children's home. Tympanometry within normal limits for both ears.

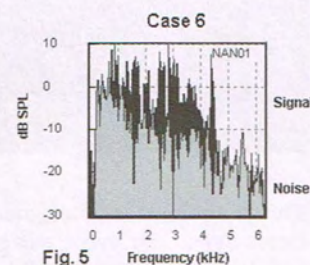


Fig. 5

Case 6: 14 year old female with delayed development. OAE results for her right ear within normative limits for Australian females of her age. Recommendation that hearing aids not be fitted.

Test Results: TEOAE results were well above average for her age for the right ear, with the left being below normal limits. That is, the ear performance suggested normal hearing should exist in the right ear, with the high risk of a mild hearing loss in the left ear.

Recommendation: Because of the unilateral nature of this lack of cochlear function, and because of the nature of this child's disability (constant movement of head as well as delayed development) it was recommended that a hearing aid not be fitted.

Significance: The key advantage of evoked otoacoustic emissions (EOAEs) in these clinical situations is a speedy means of ruling out the need for hearing aids if a partial pass is obtained. If emissions are low or absent in both ears trial fitting of hearing aids is suggested. However, if one ear is within normal limits or there are other difficulties, a further evaluation is suggested after a delay. If hearing aids are subsequently found to be ineffective the EOAE picture, particularly with contralateral testing may explain why.

However, each case is considered on an individual basis. In cases where there is unilateral, or even bilateral, low cochlear function, the patient may be suitable for fitting of a hearing aid. But the performance of daily tasks are complex and problematical for many of these children (particularly in a children's home) and the added burden of managing a hearing aid by both the children and their carers may be reason enough for not fitting hearing aids.

Reference:

Gorga, M.P., Stover, L., Bergman, B.M., Beauchaine, K.L., Kaminski, J.R. (1995). The application of otoacoustic emissions in the assessment of developmentally delayed patients. *Scand. Audiol.* 24, Suppl. 41.

Transforming hearing conservation into hearing loss prevention: Testing measures of early warning for cochlear hearing loss

Investigators: Eric LePage, Narelle Murray, John Seymour and Dan Zhou

Background: The hearing loss prevention group at NAL has been working for the last decade or so on the possibility that hearing loss in young people and adults can be anticipated in time to provide the possibility of postponing the onset of hearing loss, at least due to noise exposure, based upon estimates of redundant outer hair cell activity. Widespread research including our own (Murray and LePage, 1993; LePage and Murray, 1993) has shown that the onset of mild hearing loss is associated with a low net level of outer hair cell activity and that by the time emissions are considered "absent" the hearing level is between 30-50 dB, but there is some uncertainty as to this figure and to the frequencies or frequency ranges for which this applies. Two independent variables are supplied when click-evoked otoacoustic emissions data are collected in standard "nonlinear" mode at 80 dB pk stimulus level. These are the waveform reproducibility ("Waverepro%") and the emission sound level ("ABmean"). When the Waverepro is high, this is generally accompanied by high emission sound levels, but the reverse is not generally true, viz. the presence of external noise or significant spontaneous emission activity can result in a high emission level, composed primarily of noise. Neither of these variables can therefore be used as a single figure estimate of net residual outer hair cell activity and accordingly we defined a variable Coherent Emission Strength (CES dB SPL) which is a combination of the two raw variables¹ with the key advantages of each. In particular it is normally distributed for the NAL database population. The results of that research have been incorporated into the NAL software package known as NAL-OAE1 (LePage; Zhou; Murray, and Seymour, 2000). It has previously been established that when the Waverepro declines below about 30% and when the CES declines below about -2 to 0 dB SPL, the excess or redundant outer hair cell activity (LePage and Murray, 1993; LePage and Murray, 1996) is largely depleted resulting in a rising in hearing threshold. As the levels decline below these values there is a steep increase in the probability of there existing at least a mild hearing loss.

Research Question: The hypothesis that otoacoustic emissions provide early warning before hearing loss becomes evident is based upon the notion that significant changes in emission levels can be detected before significant changes in hearing levels can be measured. While we have clearly shown that normally hearing people in groups with differing noise-exposure profiles can be distinguished from each other purely on the basis of the click-evoked otoacoustic emissions (LePage and Murray, 1998) this is not the same as demonstrating by how much the emission decline preludes the onset of hearing loss, or by what fraction the two ranges overlap.

Research Procedures: The issue is whether the test-retest variability of one measure relative to its total range of variation is less than that of the other for its total range. Test-retest variability of pure tone thresholds is taken as ± 8 dB for all audiometric frequencies with the exception of 4 and 6 kHz where the variability is ± 15 dB. For click evoked otoacoustic emissions, test-retest variability is taken as $\pm 28\%$ for Waverepro and ± 5.5 dB for the CES (Murray; LePage, and Tran, 1997). The first step has been to determine what ranges are relevant to the comparison being carried out. Secondly, since the earliest warning for hearing loss is sometimes taken as the onset of a noise notch at around 4 - 6 kHz (McBride and Williams 2001), it is clearly necessary to compare the best otoacoustic emission measures with the best audiometric measure, even if it in fact represents the onset of a hearing loss and therefore the tail end of the subclinical period. Accordingly, from the NAL database we remove all individuals with known pathologies of the ear, but not individuals who are noise exposed (same criteria as used in LePage and Murray, 1998). One complication of dealing with Waverepro is that it is, itself, a measure of its own variability; if the Waverepro is high ($>90\%$) then the test-retest variability also tends to have high values. Conversely, as the Waverepro for any ear is found to be lower (ca 50%) then the repeat measure is likely to vary still more. And so, in order to compare CEOAE Waverepro for its early warning potential it is necessary to show the distribution of values seen in the population. When this is done (see again Figure 1) it is seen that whereas the sound level measures of the emissions are nearly normally distributed, the Waverepro (a correlation coefficient between two rapidly repeated recordings) is highly non-normal.

In order to carry out the comparison therefore it was necessary to arrive at a measure of the reproducibility which better represented its non-normal distribution. For this purpose the Waverepro measure used to compare with the audiometry was re-expressed in terms of the partitions containing equal numbers of recordings. The Waverepro values were ranked and equal numbers formed the graded categories used. The resulting decile categories 1 to 10 were found to have the following upper boundaries (+27.7, 50.7, 61.7, 70.7, 79.4, 85.2, 90.1, 93.9, 96.4, 99.9)% respectively. For the CES values, since this distribution was closely normal (see Figure 1) the categories 1 to 8 used are simply 5 dB wide ranges with upper values (-10, -5, 0, +5, 10, 15, 20, 25) dB SPL.

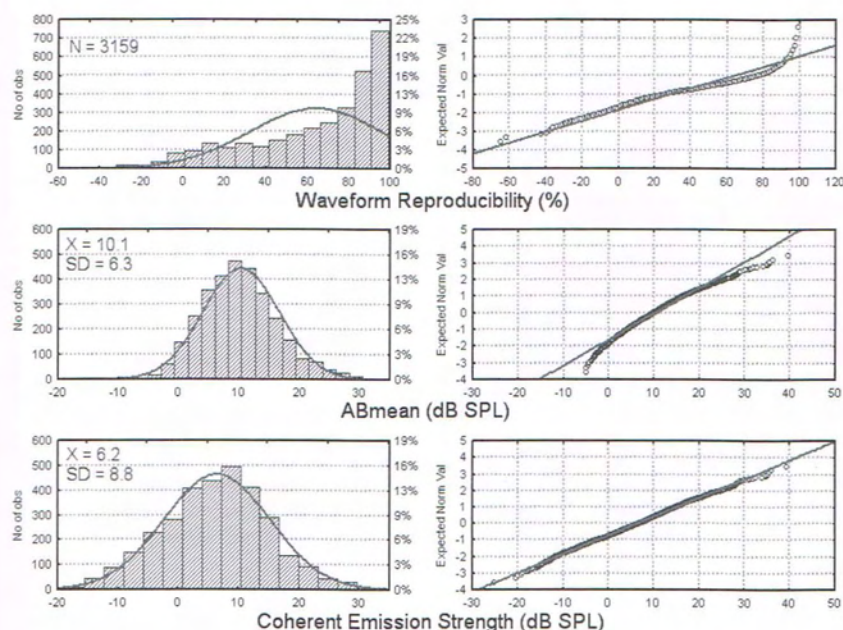


Figure 1 Statistical distributions for the three click-evoked otoacoustic emission variables for the NAL population. While the Waverepro (upper panels) has clear advantages for specifically indicating outer hair cell activity, its distribution is highly skewed as indicated by the shape of the normal distribution in the left panel and the deviation from normal in the right panel. The emission sound level (ABmean) is nearly normal but has the disadvantage that it is strongly influenced by noise. The best features are combined (CES dB SPL) yielding a variable which has practical value for estimating net levels of outer hair cell activity while possessing a normal distribution.

The audiometric values on 900 subjects were grouped in various frequency ranges or combinations to see which frequency best represents the onset of hearing loss. Accordingly, five curves are plotted, the linear averages (0.5, 1, 2) kHz, (0.5, 1, 2, 4) kHz, (1, 2, 4) kHz, (3, 4, 6, 8) kHz and max(4, 6) kHz respectively, the last being the maximum threshold observed at either 4 or 6 kHz. Figure 2 shows the results for these various measures of hearing level at the various frequencies, versus the non-frequency dependent measure of Waverepro and CES. The reasons for not carrying out filtering upon the CEOAE results are that a) we had previously shown early warning properties for the unfiltered CEOAE measures being tested here and hence we are really checking this against the audiometry, b) the issue of what frequency bands to use for such an exercise becomes highly complex when one considers that frequencies emitted in any one band can, and do, have many locations of origin along the cochlear partition (many schemes were tried and have led to new projects including the cochlear modelling project reported previously (LePage and Olofsson 1999)). The upper panel of Figure 2 is for the audiometry versus Waverepro category left ears, the lower panel is for the audiometry versus CES category. The central points are the mean pure tone values while the bars represent 95% confidence limits for all four charts.

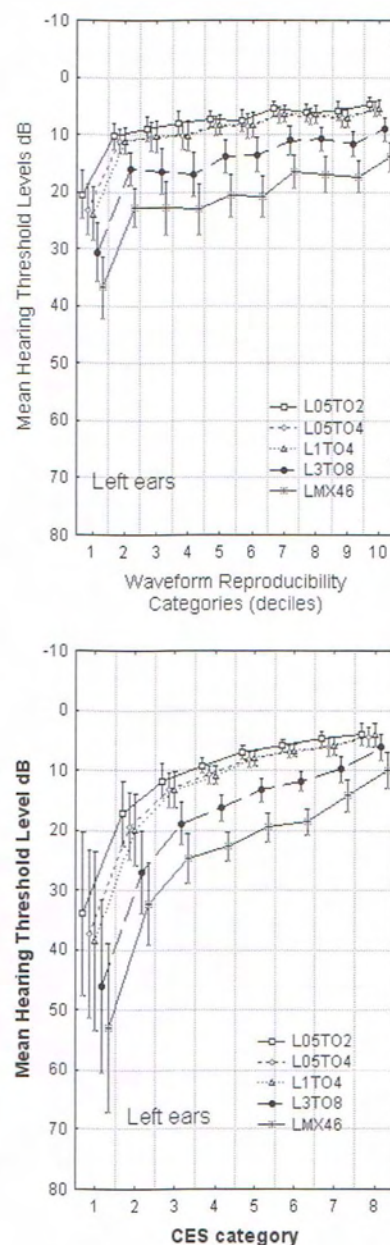


Figure 2 Four measures of pure tone thresholds are chosen to plot against the two useful OAE variables. Only left ears are shown. It is clear that for the subpopulation for which we have both OAE and audiometric results, there is not a huge number of individuals with raising hearing thresholds. However, it may be seen that those who do have hearing losses tend to group in the lowest category of Waverepro <27.7% (see text for category descriptions). The variable max(4,6) kHz threshold is clearly the most sensitive to early hearing changes. In the case of plotting the data versus CES it is seen that an early trend exists in both types of data, but that statistical variability is higher in the audiometric thresholds so that the CES registers a decline in redundancy before it would be registered by audiometry.

Findings: The upper panels of Figure 2 show that the various audiometric combinations all decline only by small amounts for most of the Waverepro values. Indeed a hearing threshold greater than 25 dB is only seen for two points in both the upper panels. Both of those mean values are for frequencies higher than 3 kHz. These two panels are consistent with the criteria chosen by Kemp of 50% representing the borderline between emission present and emission absent; in this case the value of 50.7% coincides with the onset of a mild hearing loss.

The finding of 50% reproducibility and the onset of a mild hearing loss (generally taken as 25 dB HL) coincidentally supports the notion that hearing loss starts to affect the upper speech frequencies by the time any person's ear reaches this value. The fact that the Waverepro has only reached this stage as part of the aging or damage process suggests that it is a very good measure of redundancy or excess capacity of outer hair cell activity. The fact that any coherent emission (Waverepro > 50%) is enough to allow retention of normal hearing levels suggests that there must be considerable flexibility in respect of coding at places where residual activity remains. The upper panel also suggests that the maximum threshold for 4 and 6 kHz is a considerably more sensitive measure than any other frequency combination. However, it still only begins to register a hearing loss in the very lowest Waverepro category, implying that Waverepro has considerable early warning potential by itself, i.e. it carries much more useful graded information than merely regarding an emission as present.

The lower panel of Figure 2 is for the same set of audiometric measures plotted versus our derived measure, the CES. Note that whereas the error bars are approximately the same height in the upper panels (each category having the same number of individuals), here they vary in height with the number of individuals in each category. Again clearly the best audiometric measure for early warning is the maximum value of the thresholds at 4 and 6 kHz. Considering that test-retest variability for these frequencies is up to 15 dB there will still be considerably uncertainty as to the actual value to assign to any particular test so that a notch at 4 or 6 kHz would need to stand out from frequencies either side to constitute a clear warning, but the CES value for this condition would be already lower than the previous critical value of 0 (category 3). For this number of subjects, however, the mean value greater than 25 dB HL coincides with the third category of 0 dB again confirming that this value of CES does indeed represent a critically low value of outer hair cell activity. Considering test-retest for CES is 5 dB this means that the CES declines well over half of its range by the time it is registered by regular audiometry. The degree of overlap between the emission and pure tone threshold ranges is therefore approximately 20 to 25 percent for each range (Figure 3).

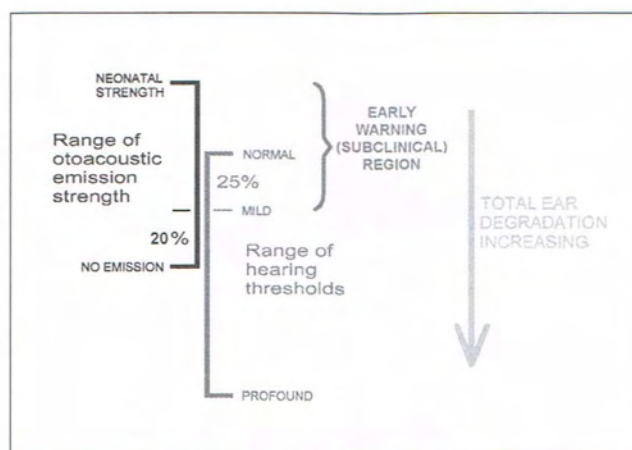


Figure 3 Schematic summarising the effective overlap between the objective and subjective data. As shown previously the highest 80% of the emission range is useful for early warning while the lowest 20% of the emission range is useful for estimating the risk for mild hearing loss.

Significance: Hearing conservation programs in the past have been based, as the name implies, upon conserving residual hearing. Such programs have been instigated by virtue of the number of workers in noisy occupations suffering a hearing loss. This study suggests that for such people the warning came only at the very end of the long-term damage process, at the end of the subclinical period. By contrast these results show that a very clear distinction can be made between a young person with normal hearing and with a high emission strength and a young person with normal hearing but a low emission strength. The higher the value of emission strength or Waverepro, the more confident we are that (aside from known exceptions) the ear has considerable redundancy and this means (barring accidents or disease processes) a person can look forward to a long lifetime of normal hearing. By contrast, a person with normal hearing who is identified through a screening or other program to have emission values much lower than is normal for their age may be at significantly greater risk for imminent hearing loss - in the range of 1 to 10 years rather than 40 to 60 years. Because of the property of emissions to exhibit higher variability as they decline in strength, there is less confidence in the low result, indicating that any low result should be repeated immediately, and probably again in a month's time. For these reasons, we feel confident to pursue the notion that otoacoustic emissions offers a new era of "hearing loss prevention" rather than conservation, because the subclinical condition may be recognised in plenty of time for preventive measures to slow the damage process. Our software package NAL-OAE1 was developed to graphically display the risk for hearing loss, extending the clinical usefulness of otoacoustic emissions considerably beyond the early detection of hearing loss in newborns.

References:

- LePage, E. L. and Murray, N. M. Click-evoked otoacoustic emissions: comparing emission strengths with pure tone audiometric thresholds. *Aust.J.Audiol.* 1993; 15 :9-22.
- LePage, E. L. and Murray, N. M. Latent cochlear damage in personal stereo users: a study based on click-evoked otoacoustic emissions. *Med. J. Aust.* 1998; 169:588-592.
- LePage, EL and Murray NM. A new approach to hearing loss prevention. In. Annual report 1995/1996. National Acoustic Laboratories; 1996: 25-30.
- LePage, EL and Olofsson, Å. Modelling transient evoked otoacoustic emissions in noise-induced hearing loss. Annual report 1998/1999: National Acoustic Laboratories; 1999: 26-30.
- LePage, EL; Zhou, D; Murray, NM, and Seymour, JD. NAL-OAE1: NAL transient evoked otoacoustic emission analysis software. Annual Report: 1999/2000. National Acoustic Laboratories; 2000: 3-4.
- McBride, D. I. and Williams, S. Audiometric notch as a sign of noise induced hearing loss. *Occup Environ Med.* 2001 Jan; 58(1):46-51.
- Murray, N. M. and LePage, E. L. Age dependence of otoacoustic emissions and apparent rates of ageing of the inner in an Australian population. *Aust.J.Audiol.* 1993; 15(2):59-70.
- Murray, N. M.; LePage, E. L., and Tran, K. Repeatability of click-evoked otoacoustic emissions. *Aust.J.Audiol.* 1997; 19(2):109-118.

Endnote:

¹ CES (dB SPL) is the emission sound pressure (ABmean), weighted by the Waverepro (rescaled to the interval [0,1]) squared.

The scope of non-noise factors influencing research aimed at the effects of noise: one example - blood cholesterol level.

Investigators: Eric LePage, Narelle Murray, Tony Butler¹

¹ NSW Corrections Health Service

Background: The prevention of hearing loss shares many aspects in common with other areas of preventive medicine and science - its success depends on individual behaviour up to the point where the condition starts to cost society in a significant way so that (1) we cannot fail to ignore it and (2) we seek means of reducing its effect. The disease may cost that individual significantly, and be seen to be very costly to society, but until the time onset of any disability is shown to be not just age-related but specifically related to any particular cause or set of causes, it is practically uneconomic to treat it otherwise than by rehabilitation, even if the number of cases with the condition approaches a third of the population.

Conditions which in the past have flagged hearing loss as being tied to occupational noise have demanded attention (see Figure 1) because clearly over a period of a working life, those working with average daily doses of 85 dB(A) or less suffer little hearing loss, whereas those working in 95 dB or 105 dB stand to suffer much more hearing loss. The figure shows three sets of curves. The heavy lines represent the mean loss over 25 years for 85, 95 and 105 dB(A) average daily doses. The thinner lines descending downwards show the standard deviation (16th percentile) and the 10th and 5th percentiles, respectively, indicating the varying susceptibility of individuals in terms of the hearing loss produced by the same exposure levels. These measures of susceptibility are determined after the hearing loss is already incurred. The most important fact contained in this plot, and the basis of the ISO1999 (1990) standard is that the best strategy to avoid noise-induced hearing loss is for the worker to avoid sound levels over 85 dB, because, according to the model, it doesn't matter how susceptible one turns out to be, only incidental increase in noise-induced permanent threshold shift (NIPTS) will occur. On the other hand if avoidance of loud sound levels is not possible, then the degree of loss depends hugely on whether one is more or less susceptible to noise exposure.

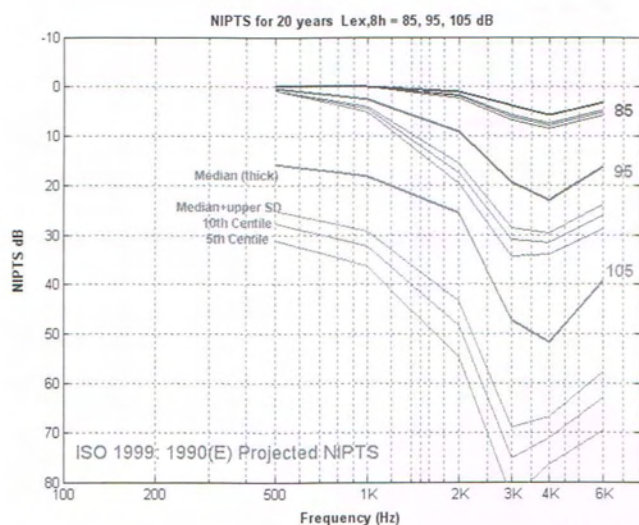


Figure 1 Projected hearing losses from ISO-1999 for occupational noise exposure for 25 years. The three sets of curves represent the hearing levels as a function of frequency for three increasing levels of noise Lex,8hr of 85, 95 and 105 dB respectively. Each set consists of four curves, the thick line is the mean hearing loss, the next below is the mean minus one standard deviation, the 10th percentile and the 5th percentile respectively, representing post-hoc measures of hearing susceptibility.

Traditionally, we have not known which people are most susceptible to noise induced loss until after they have received a hearing loss. An object of any modern prevention program therefore is to try to determine in advance who is more susceptible to suffering any particular condition to target the most efficient use of funds. Our approach is to obtain early warning measures of risk for noise-induced hearing loss, i.e. to work out who is more or less susceptible to noise before hearing loss occurs. This has been the topic of intense interest over recent years. However, it turns out there are very many causes of hearing loss.

There are now well-characterised genetic factors particularly associated with the Connexin and Pendrin genes. It does not make much sense to regard children identified as having genetic deafness as merely "being at risk" for hearing loss. The research into genetics of hearing loss is progressing to identify risk factors for parents who may be carrying such genetic risk factors. Figure 2 contains a schematic summary of the "not-obviously-genetic" causes of hearing loss, any one of which may increase risk for permanent hearing loss at some stage during life and in combination may pose increased risk. An area of intense interest is the combination of toxins and noise which may produce more rapid onset of hearing loss because of mutually potentiating factors (Henderson, 1999; Willott et al, 2001). For example, a worker in a noisy plant exposed to solvents or their volatile fumes may be far more at risk for hearing loss than the noise without the solvent. Into the general category of ototoxicity is also included prescription drugs which have hearing loss as major side effects, e.g. certain antibiotics

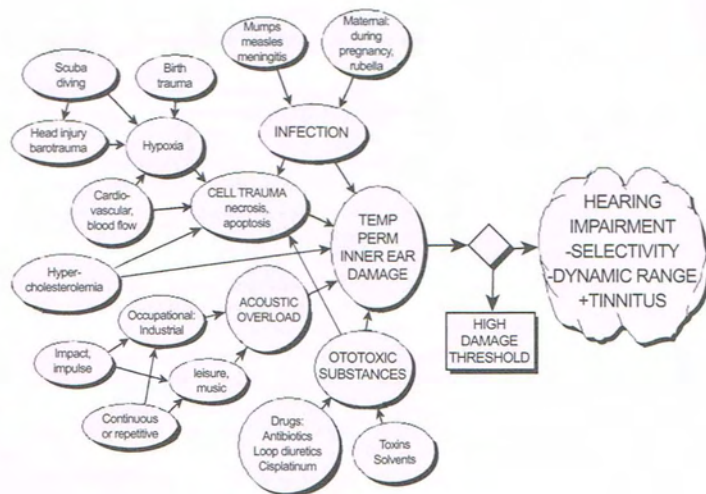


Figure 2 This schematic represents the main non-genetic, non-syndromic factors which may result in damage to the cochlea (infection, physical injury, hypoxia, acoustic overload and ototoxic substances) and some of the external factors which feed into these main factors. The result is an accumulation of damage to the inner ear for many years which is the result of temporary effects which are internally repaired and permanent damage for factors which are beyond repair. When the damage is extensive this results in the first signs of hearing impairment, initially tinnitus (temporary or permanent) and the inability to select speech particularly in noisy-background situations. Dynamic range is finally affected as sensitivity declines but this is often the last symptom to be exhibited. Sometimes the vestibular system is also affected. Note that age is not considered an explicit factor, but rather a net effect of the many other factors.

such as the aminoglycosides, and heavy-metal cancer treating drugs such as cisplatin. There are others which appear to have lesser ototoxicity in that they do not show rapid losses of hearing, but when taken over a moderate term may still result in hearing losses or may accelerate the normal aging processes by a reduction of the excess capacity of the outer hair cell system (LePage, 1998). Into this category come loop diuretics (such as furosemide and ethacrynic acid), while aspirin and quinine are regarded as only mildly toxic. Then there are the biochemical constituents of the body itself which when excessive may result in toxic reactions and secondary risk factors. Another example is the mutually potentiating factors of excessive noise and high blood cholesterol levels (Preyer et al, 2000).

Research question: A significant part of NAL prevention research has been the employment of otoacoustic emissions in addition to pure tone audiometry to serve as a more immediate gauge of levels of accumulated damage as a function of age and noise exposure. As we show elsewhere in this report, a ten-year study of a Sydney orchestra has failed to produce evidence that the population of orchestra players as a whole suffers any hearing loss or even shows any significant trend towards obtaining a hearing loss. Hearing losses are, however, found for some orchestra members. The click evoked otoacoustic emission data are characteristically highly variable from one year to the next, much more variable than shown in our test-retest study carried out over a ten day period (Murray et al, 1997), or is generally believed to be the case for otoacoustic emissions based upon limited studies. Our research for the past few years has been endeavouring to discover the source of variation, describe it, control it and/or be able to correct for it. To capitalise on the early-warning potential of otoacoustic emissions reported previously we need a better method of deciding how much of the variation we see from one record to the next is due to measurement noise and how much is due to inherent biological variation. Of the latter, how much of the variation reflects individual susceptibility (tougher ears appear more stable), how much of it amounts to a permanent decrease in emissions, how much is temporary and how much is due to dynamic mapping variation?

Research Procedures: At NAL we have two basic approaches to the problem. The first concerns changes over time in a cohort of individuals using click-evoked otoacoustic emissions looking for features which are common across the set for any person (each ear's "fingerprint") and looking at the types of variation which are seen on top of the common feature. Secondly, we are looking at the rapid variations that are seen in the process of collecting otoacoustic emission data.

The causes of permanent hearing loss shown in the figure are obviously also sources of variability in the data. Over a ten-year period our questionnaires revealed numerous individuals on drug therapies, which may have a temporary effect. Some individuals could have varying levels of blood cholesterol which may masquerade as an aging effect or be influenced by statin therapy to reduce blood cholesterol levels. To gauge whether for example, cholesterol level could be affecting variability in any longitudinal data, we participated in one study collecting otoacoustic emissions in which blood cholesterol level was collected.

This was the study run by the NSW Department of Health, on the general health of inmates in 26 NSW prisons. Some 850 inmates had a large number of health variables assessed either by questionnaire or other physical assessment including sensory testing and blood sampling. The total set of health variables was extensive. NAL staff were invited to participate in the study carried out in 1996 to obtain hearing data. Due to the size and nature and location of the population sample, pure tone audiometry was impractical, and click-evoked otoacoustic emissions were chosen to assess the ear performance.

Findings: Figure 3 shows the result of relating total blood cholesterol levels to emission Waveform reproducibility(%) in a one-way ANOVA. The upper panel in the figure shows the distribution of cholesterol levels found in the inmate population. Two vertical dashed lines are added to show the mean cholesterol values of two groups taken to represent normal and raised in the Preyer et al study. The effect has been explained by Oghalai et al (1999) in terms of the cholesterol level modifying the lipid levels in the walls of the outer hair cells, modifying their stiffness and the level of emissions they produce. A person (in this group) with total blood cholesterol of 8 (mMol/L) has a Waverepro of about 42% which is about ten percentage points below a person with a more acceptable value of 5. The lower panel shows that this difference is significant ($p=0.0014$). In terms of the distribution of Waverepro (elsewhere this report) this also represents a significant decrease in functional terms. In terms of our study comparing industrial exposure with personal stereos (LePage and Murray, 1998), this is an equivalent aging effect of more than ten years of industrial exposure, so that the risk for hearing loss can be expressed in terms of a reduction of the normal aging period before the onset of hearing loss.

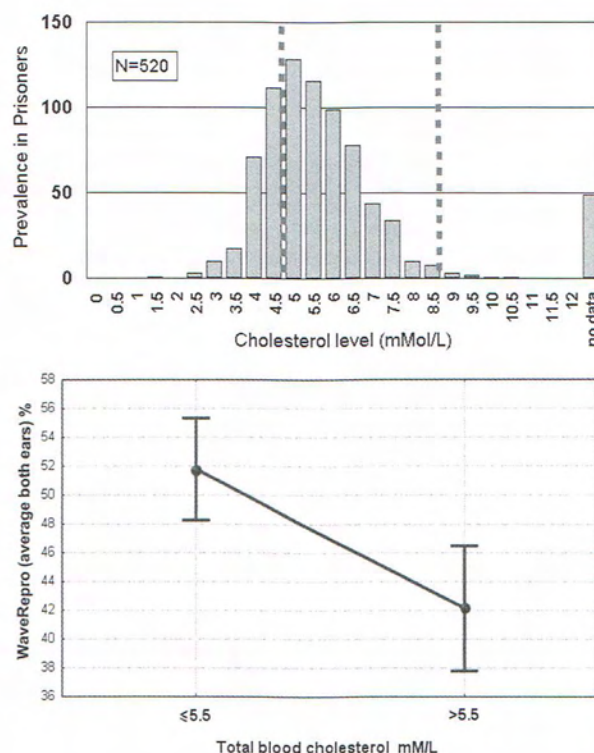


Figure 3 One of the non-noise factors affecting research into the effects of noise trauma upon hearing using otoacoustic emissions. Otoacoustic emission strength is seen to be dependent upon blood cholesterol level in a population of 682 inmates in NSW prisons. Error bars indicate the 95% confidence interval. The prevention message comes in many forms - one may be able to reduce risk for early hearing loss by usual measures of controlling blood cholesterol.

The figure therefore demonstrates that whether or not cholesterol level directly affects hearing, or susceptibility to hearing loss, it definitely affects the variable which is being used to gauge outer hair cell performance in such a way as to render difficult making conclusions from any longitudinal study that is trying to relate susceptibility to emissions.

Significance: Cholesterol level was not anticipated as a factor when the longitudinal study was established, but this outcome is used to underline the fact that many factors may have adverse affects on research into prevention of noise-induced hearing loss. It has been almost axiomatic in hearing conservation programs that the objective is to lower noise levels in line with equal energy considerations, so this result is an example of why such a strategy may in reality be effective, but not be seen to be so because other factors are not being controlled in the experimental design. The level of interest in biological factors affecting cochlear emissions is exemplified by the likely relevance of blood cholesterol to susceptibility to hearing loss. If susceptibility is tied to the way the ear ages (LePage, in preparation), and in turn this is tied to changes in mapping of sounds, which is in turn tied to outer hair cell stiffness, then it seems likely that, quite apart from affecting the otoacoustic emission measures of determining susceptibility, variation in cholesterol level could be an important factor underlying how the ear responds to noise exposure.

References:

- Henderson, D. (1999). Ototoxicity - Basic Science and Clinical Applications. Annals of the New York Academy of Sciences ISBN 1-57331-205-3, edited by D. Henderson, R.J. Salvi, A. Quaranta, S.L. McFadden and R.F. Burkard. November 28, 1999, Vol 884, 438pp
- ISO1999 (1990). Acoustics - Determination of occupational noise exposure and estimation of noise-induced hearing impairment. Second edition, 1990-01-15, Section 5.3
- LePage, E. L. Occupational Noise-Induced Hearing Loss: Origin, Characterisation and Prevention. *Acoust.Aust.* 1998; 26(2):57-61.
- LePage, E. L. and Murray, N. M. Latent cochlear damage in personal stereo users - a study based on click-evoked otoacoustic emissions. *Med. J. Aust.* 1998; 169:588-592.
- Murray, N. M.; LePage, E. L., and Tran, K. Repeatability of click-evoked otoacoustic emissions. *Aust.J.Audiol.* 1997; 19(2):109-118.
- Oghalai, J. S.; Tran, T. D.; Raphael, R. M.; Nakagawa, T., and Brownell, W. E. Transverse and lateral mobility in outer hair cell lateral wall membranes. *Hear Res.* 1999 Sep; 135(1-2):19-28.
- Preyer, S.; Baisch, A.; Bless, D., and Gummer, A. W. Distortion product otoacoustic emissions in human hypercholesterolemia. *Hear Res.* 2001 Feb; 152(1-2) 139-51.
- Willott, J.F.; Chisolm, T.H. and Lister, J.J. Modulation of Presbycusis: Current Status and Future Directions. *Audiology & Neuro-Otology* 2001, 6(5): 231-249.

Nine years' progress report of study of inner ear damage in an Orchestra.

Investigators: Narelle Murray and Eric LePage

Background: In 1992 WorkCover (NSW) invited NAL to be part of the Australian Opera and Ballet Orchestra Occupational Health and Safety Program as it related to the hearing of the orchestra. In particular it was felt important to measure any ear damage occurring in the orchestra with click-evoked otoacoustic emission testing as well as pure tone audiometry. This initial examination developed into the longest known ongoing longitudinal study of an orchestra employing click-evoked otoacoustic emissions.

Research Question: Can useful, definable parameters be derived from testing with either or both click-evoked otoacoustic emissions and pure tone audiometry to establish the basis of an early warning system for prevention of hearing loss, particularly for those working in noise?

Research Procedures: Thus far 140 members of the Australian Opera and Ballet Orchestra (78 males, 62 females) have been tested. Twenty four musicians have been in the study since 1992 and although not tested every year have been tested both in 1992 and 2001. Eleven of these have been in the program every year since its inception; a further 8 entered the program in 1992 and were last tested in 2000. Seven began in the program in 1993, 3 of whom were tested in 2001 and 4 in 2000. A total of 39 musicians have, therefore, effectively been involved in the whole study to this point. All Sections of the Orchestra and every instrument are represented with 11 members of the Brass section, 2 Percussion, 16 String players and 10 Wind instrumentalists participating. Each year musicians voluntarily have their ear damage assessed by the objective method of click-evoked otoacoustic emissions (OAEs). Pure tone audiometric thresholds (PTA) are also obtained. Every musician also completes a questionnaire on their aural health, length of time they have been a musician, length of time they have been in an orchestra and their average hours of music exposure per week including performance, rehearsal and private practice. Other factors associated with hearing loss, such as tinnitus, antibiotic and anti-inflammatory use, smoking and recreational noise exposure are also assessed.

Findings:

For the group of 39 musicians' pure tone audiometric results for the maximum values of either of the frequencies 4 and 6 kHz (deemed to be the established frequencies for noise induced hearing loss) there are no significant group differences from year to year between 1992 and 1999 for either left or right ears (Fig. 1A). The same applies for the calculated otoacoustic emission parameter of Coherent Emission Strength (CES) (Fig. 1B).

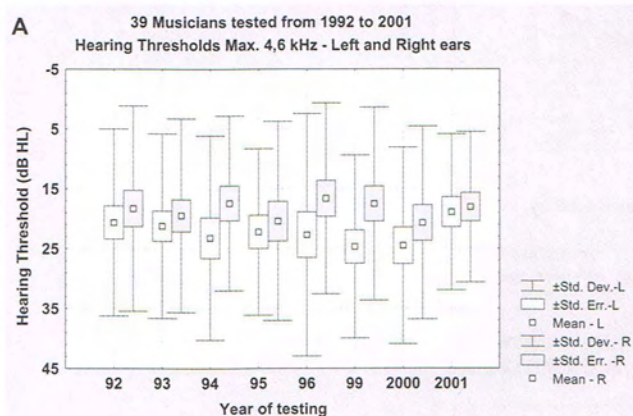
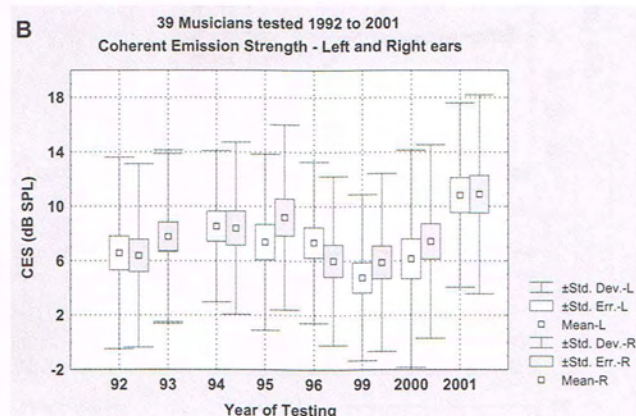


Fig.1.A. Mean, standard deviation and standard error of hearing threshold levels, taken as the maximum obtained at either 4 or 6 kHz in the left ears (open rectangles) and right ears (closed rectangles) for each of the years tested from 1992 to 2001. Only the results for those 39 musicians who entered the program in 1992 or 1993 and were still being tested in 2000 or 2001 are shown.



B. Mean, standard deviation and standard error of CES values for the same ears as in Fig. 1A. Left ears are open rectangles and right ears closed rectangles for each of the years tested from 1992 to 2001.

Individual results indicate a wide variation as evidenced by the large standard deviations in Figs. 1A and 1B. Group results for both parameters for the years 2000 and 2001 have shown an "improvement" particularly for CES values. Fig. 2 also demonstrates the "improvement" with the individual CES results for 2001 being above those for 1992, while the majority of PTA results for both years are within normal hearing limits (≤ 25 dB HL).

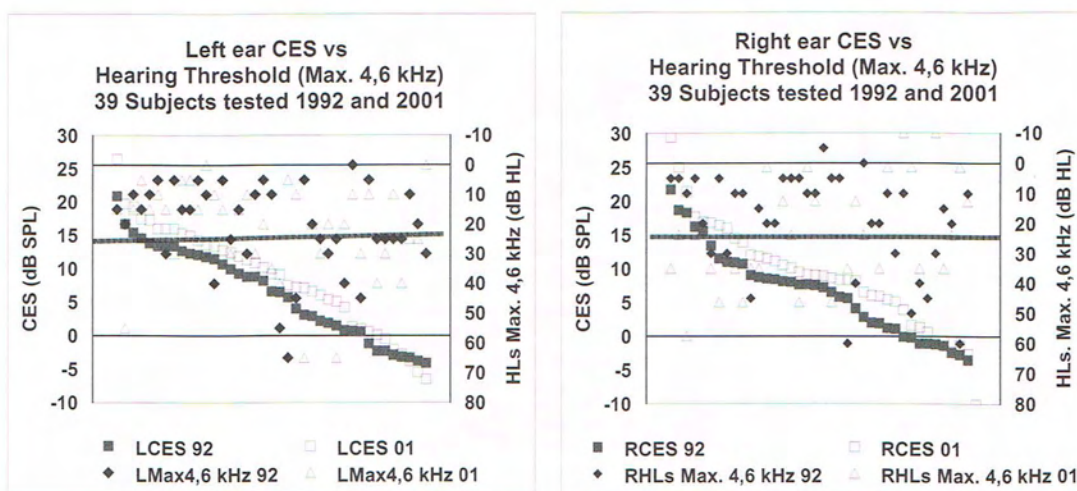


Fig. 2. Individual results in rank order of CES values (left ear shown in the left panel, right ear in the right panel) plotted against the hearing threshold values, again taken as the maximum values obtained at either 4 or 6 kHz. It can be seen that for both left and right ears the CES values for 2001 (open squares) appear above the 1992 values (closed squares) indicating an overall "improvement" in OAE results. It can also be seen that the majority of hearing thresholds have remained <25 dB HL (1992 values, closed diamonds; 2001 values open triangles).

Figs. 3 and 4 show some of the variations exhibited by individuals when their results from 1992 are compared with their latest results in 2001.

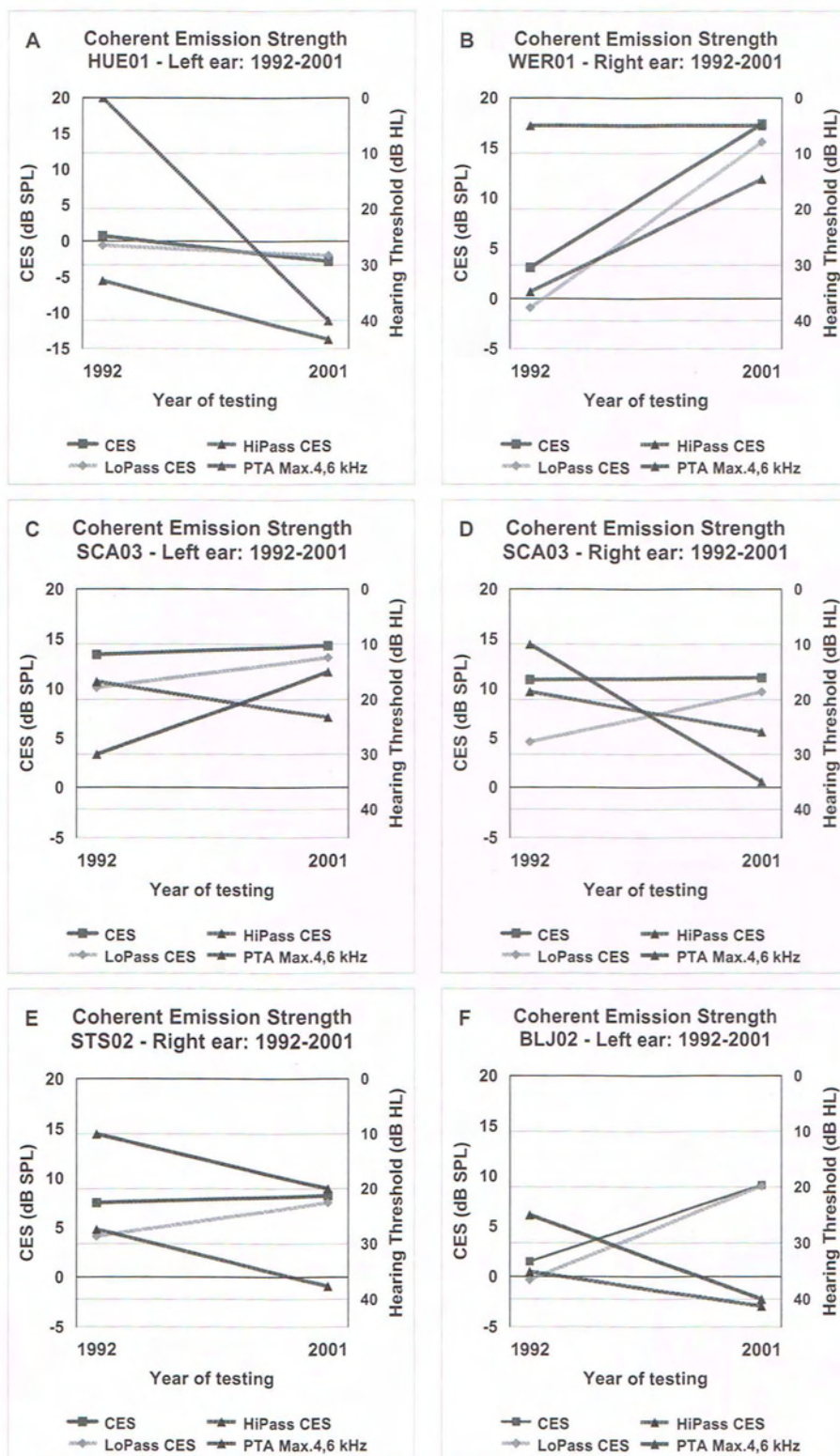


Fig. 3. Six panels demonstrate individual variations when results are compared between 1992 and 2001. In all panels, PTA is depicted by open circles read on the right hand axis. CES (closed squares) has been analysed as being the total value over all frequencies, LoPass (open diamonds) as being the CES value below 2 kHz and HiPass (closed triangles) as the value above 2 kHz. Panel A, HUE01's left ear, shows a marked decline in PTA (from 0 to 40 dB HL) and a lesser decline in OAE results. Panel B, WER01's right ear demonstrates PTA remaining at the same level, while the OAE results show a significant improvement. Panel C, SCA03, left ear shows a significant improvement in PTA, while the HiPass CES declines and the LoPass CES "improves". Panels D (SCA03, right ear), E (STS02, right ear) and F (BLJ02, left ear) all exhibit declining PTA and HiPass CES with LoPass CES "improvement".

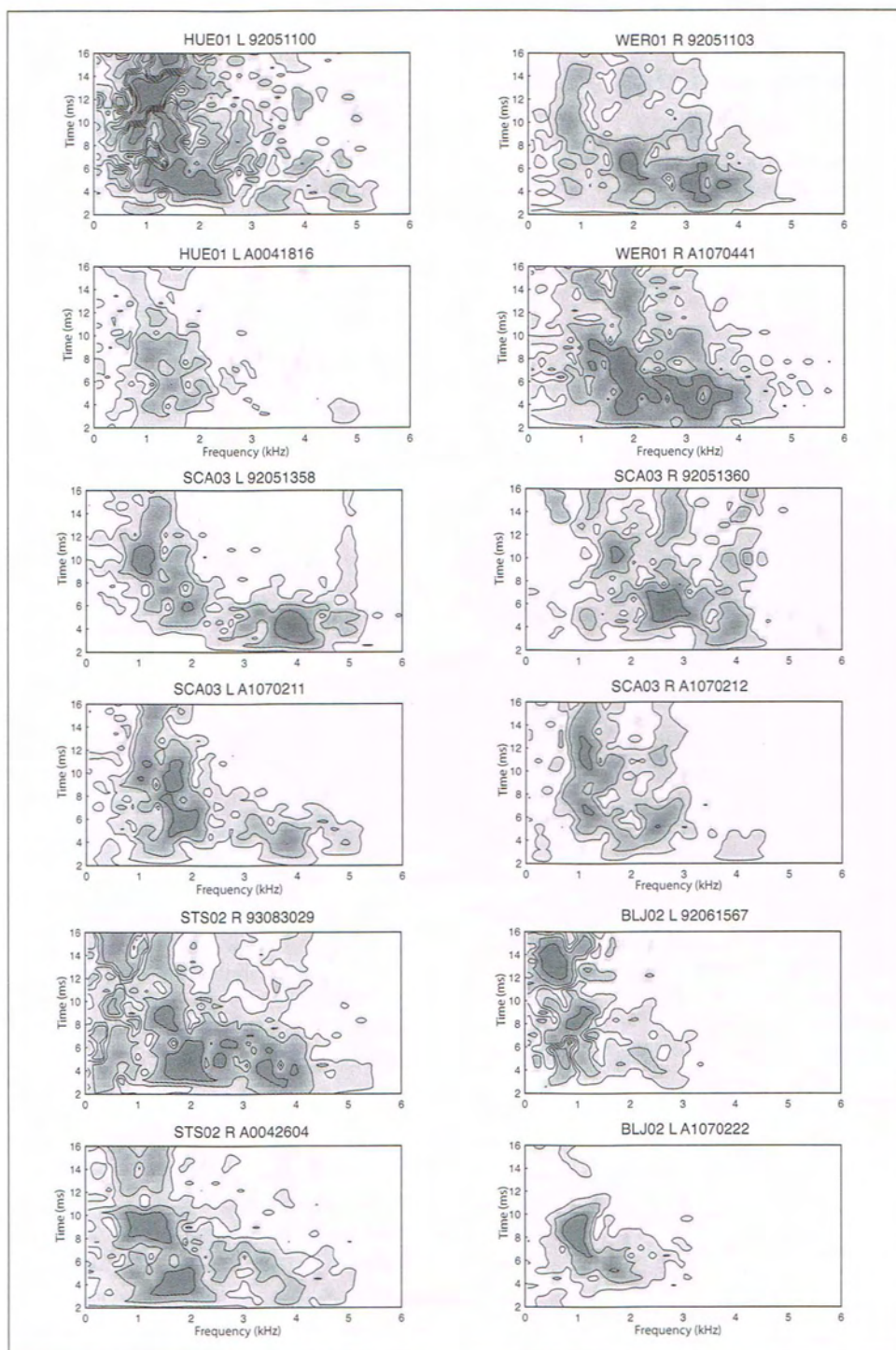


Fig. 4. Time-frequency plots depicting outer hair cell activity. The x-axis of each panel shows the frequencies from 0 to 6 kHz and the y-axis the latency of the response from 2 to 16 ms. The darker the area of the response the more intense the activity in that area. The top half of each panel represents the result from 1992, the lower half the result from 2001. The six panels in this figure show the same subjects as in Fig. 3 and should be read in conjunction with that figure by comparing them by codes (e.g. HUE01, WER01, etc.). HUE01 shows a general decline in activity over all frequencies from 1992 to 2001 - in line with PTA results also. WER01 shows a general intensifying of activity in areas from 1 to 4 kHz and at points of lower latency. SCA03, left and right ears, both show somewhat less intensity of activity in high frequency areas with a corresponding increase in activity at lower frequencies. Similar patterns of activity also occur for STS02 and BLJ02. It is surmised that these last four panels are indicative of some cochlear remapping whereby high frequency activity declines at the same time as lower frequency activity increases.

Significance: In previous studies (LePage & Murray, 1993, LePage & Murray, 1994) it has been demonstrated that there is a critical value of CES (~ 0 dB) at which point a mild hearing loss may be incurred. As in the previous studies, there are a number of individuals whose PTA results are within normal hearing ($= / < 25$ dB HL) limits

while their CES values are < 0 dB (SPL) (Fig. 2). This may be because OAEs are more immediately sensitive to both temporary and/or permanent cochlear change. If this is so then OAEs and in particular the CES parameter may be appropriate for use in hearing conservation programs as an early indicator of hearing loss. There are some ears for

which PTA results indicate a hearing loss while CES values are >0 dB (SPL). The explanation may lie in another project in which we are investigating the possibility that these ears exhibit a cochlear mapping change whereby decreased high frequency activity is exhibited as increased low frequency activity. This would present more clearly and rapidly in OAE testing than in PTA (see Figs.3 and 4). OAEs may very well prove to be a very helpful adjunct to all hearing conservation programs.

The Australian/New Zealand Standard AS/NZ1269.4 show projected pure tone hearing losses, on a population basis, for noise exposures between 70 and 100 dB(A) (LAeq,8h) for frequencies 0.5 to 6 kHz. These are based on equations set out in ISO 1999. The recommended daily limit for occupational noise exposure is set at 85 dB(A). In 1995, in an endeavour to lower the noise exposure to members of the orchestra, a perspex "cage" for the trumpet, trombone and tuba members of the Brass section was erected to restrict their effect on other members of the orchestra. Since 1998 orchestra management have also had a hearing conservation program in place which limits the noise exposure of all players to 85 dB(A)(LAeq8h). This would mean, according to the Standard, that 95% of players would only incur a 1 dB permanent threshold shift over 10 years. At most, 5% of the group would incur a 5 dB loss. At worst, had the orchestra been exposed to 95 dB(A)(LAeq8h) prior to 1995 the greatest permanent threshold shift would have been 20 dB for 5% of the musicians. As well, on the basis of a study on the repeatability of otoacoustic emissions (Murray et al, 1997), a significant change in Coherent Emission Strength (CES) values would need to be 5.5 dB or greater. On a group basis, this has not happened; neither of the aforementioned has occurred (see Figs. 1 and 2). This may indicate a) the orchestra, as a whole, have very tough ears, b) every player conscientiously cares for their hearing, or, c) the two implementations put into effect by Orchestra management have had a positive effect on the hearing of members of the orchestra. The last seems the most likely.

References:

- Standards Australia and Standards New Zealand1998. Australian/New Zealand Standard. Occupational noise management. Part 4: Auditory Assessment.
- LePage E. L., and Murray, N.M., 1993. Click-evoked otoacoustic emissions: comparing emission strengths with pure tone audiometric thresholds. *Aust. J. Audiol.* 15:1, 9-22.
- LePage, E.L., and Murray, N.M. 1994. Comparison of otoacoustic emission measures of cochlear damage in the Australian population with hearing loss in the Australian and British populations. *Proceedings of the Better Hearing Australia Conference, Adelaide, Aug 7-11, 1994.*
- Murray, N.M., LePage, E.L., and Tran, K. 1997. Repeatability of click-evoked otoacoustic emissions. *Aust. J. Audiol.* 19(2), 109-118.

Effective training methods for workplace noise reduction

Investigators: Suzanne Purdy and Warwick Williams

Background: This project is a direct result of the "Local Government Occupational Noise Management Strategy" project carried out by NAL under a grant from the *WorkCover New South Wales Injury Prevention, Education and Research Grants Scheme* (see *NAL Annual report 1999/2000*, p 5). That project indicated a need to more fully examine the methods used to train individuals in OHS issues.

Individuals are more involved with OHS in the workplace if they feel that they had some effect on the outcomes that affected them. It is hypothesised that this process of developing self-efficacy can be developed through a workshop learning process as opposed to the standard "chalk-and-talk" approach to OHS instruction.

Research has been carried out for many years with the object of improving OHS performance in the work place. Early work such as that by Zohar, Cohen and Azar (1980) showed questionable improvements in performance by providing feedback on the use of hearing protectors. More recently research has questioned the "hearing conservation paradigm" itself (Hetu, 1994) while contemporary approaches examine the effects of work place culture (Feyer and Williamson, 1998).

The self-efficacy approach to risk taking behaviour has been discussed extensively by Schwarzer and Reinhart (1995) and the possibility of developing this approach through the use of OHS workshops was demonstrated during the "Local Government Occupational Noise Management Strategy" project.

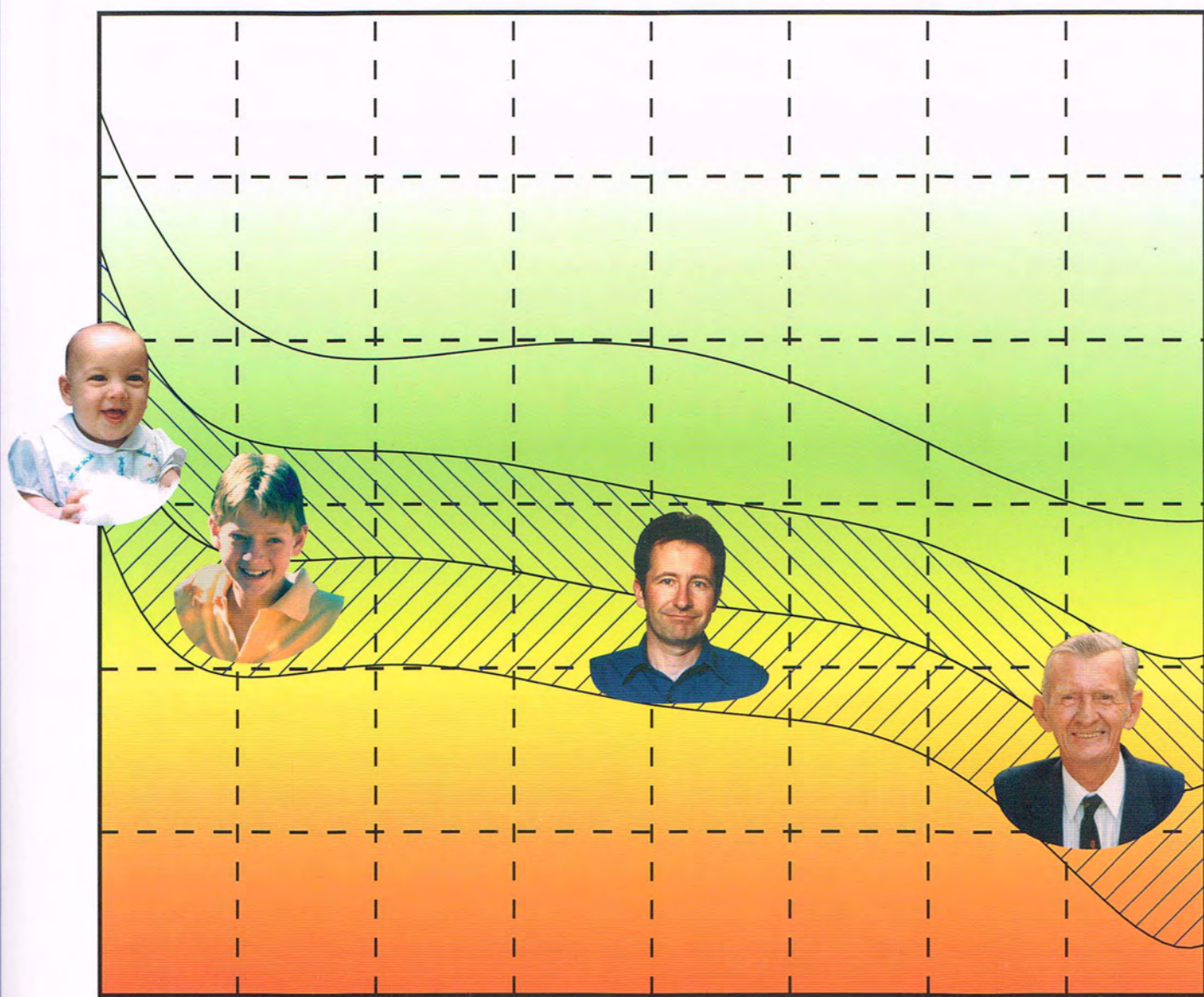
A significant influence on work-place safety culture is the 'safety climate' that can be measured through the use of a questionnaire such as that as developed by Williamson, Feyer, Cairns and Biancotti (1997). As safety culture has a direct relation to work place OHS, some of the parameters to be measured in the current project will relate to work-place safety climate.

Research questions: The three research questions that are addressed in this project are-

- 1) That training in hearing loss prevention that uses a workshop format, involves workers in generating their own solutions for hazard reduction, and discusses noise in the context of other workplace hazards will be effective in reducing workers' noise exposure levels;
- 2) That the effectiveness of hearing loss prevention training is affected by personal factors such as worker's perceived self-efficacy, noise annoyance, barriers, benefits and susceptibility to hearing loss; and
- 3) That the effectiveness of hearing loss prevention training is affected by the safety culture in the workplace.



National Acoustic Laboratories **Annual Report** 2001/2002





Secretary of the Department of Health and Ageing, Jane Halton, having her otoacoustic emissions tested and compared to the normative data generated from the NAL-OAE1 analysis software, with Dr Narelle Murray (left) operating the software and Dr Eric LePage (standing) explaining the results



Minister Andrews (right) is looking at how Dr Teresa Ching (left) fine-tunes the hearing aid of a child who also uses a cochlear implant in the opposite ear

Visitors

As always, NAL was visited by many local and overseas researchers and other professionals working in the area of hearing assessment, hearing loss prevention, and hearing rehabilitation loss (listed on page 51). This year we were also honoured by the visits of Ms Jane Halton, Secretary of the Department of Health and Ageing, and The Hon Kevin Andrews, MP, Minister for Ageing, on 15 March and 18 April 2002 respectively. They toured the test facilities and witnessed some of the research activities carried out at NAL.

New direction

One new direction for NAL this year was the commencement of a project proposed, commissioned, and funded by a hearing aid company. The results of any such research projects (there are now several more under discussion with other companies) are provided as a confidential report to the company commissioning the research. Projects of this type have many advantages. From NAL's perspective, the increased funding enables additional research to be done, and the knowledge generated allows us to more appropriately formulate successive projects (subject always to protecting information confidential to particular companies.) Some results will, by mutual agreement, also be published and be widely available. From the sponsoring company's perspective, NAL provides an efficient means to obtain independent research results as it has an established infrastructure of equipment, facilities, technical and audiological research expertise, and test subjects.



Dr Harvey Dillon explains to Minister Andrews the process of measuring cortical potentials (electrical brain activity) in infants and children to evaluate and fine-tune hearing aids.

Harvey Dillon

NAL-OAE analysis software – philosophy and a potted history

Eric LePage

Otoacoustic emissions became generally accepted at a scientific conference in London in 1980. At that stage two primary methods of generating otoacoustic emissions were being explored – distortion products (DPOAE) and transient- or click-evoked otoacoustic emissions (CEOAE). The advantages of either approach were recognized to have use in *early detection* of hearing loss. In the eighties, research showed how they could be classified as “present” or “absent”. This is particularly the case in neonatal emissions which are considered normally large. It was easy to develop an application for otoacoustic emissions for early detection of hearing loss in neonates and infants. These determinations had high values of sensitivity and specificity (~90%) indicating that emissions could be used reliably to decide which children should be referred for early intervention. By the early nineties, these considerations led to the development of the Rhode Island criteria (pass/partial-pass/refer), based on the strength of otoacoustic emissions at low frequencies.

The NAL experimental program on CEOAEs began when I arrived at NAL in 1989. My background was in cochlear mechanical measurements, particularly using transient methods, and I wished to apply this knowledge to better understand the changes in cochlear mechanics that underlie hearing loss, and to better prevent hearing loss. It was fully appreciated that although DPOAEs had the edge when it came to clinical acceptance, by virtue of plotting frequency responses as “DP-grams”, the click method had other advantages for understanding cochlear function, particularly in respect of timing information. The sounds (“echoes”) emitted from the cochlea have all but died away in 20 ms – the blink of an eye. This property means they have huge advantages in revealing fine changes in cochlear timing which occur due to aging or noise effects. Together with audiologist Narelle Murray, CEOAE and pure tone threshold data were accumulated and compared. Over the next decade it became apparent that CEOAEs had advantages for providing *early-warning* of gradual-onset hearing loss, which had not been shown for DPOAEs.

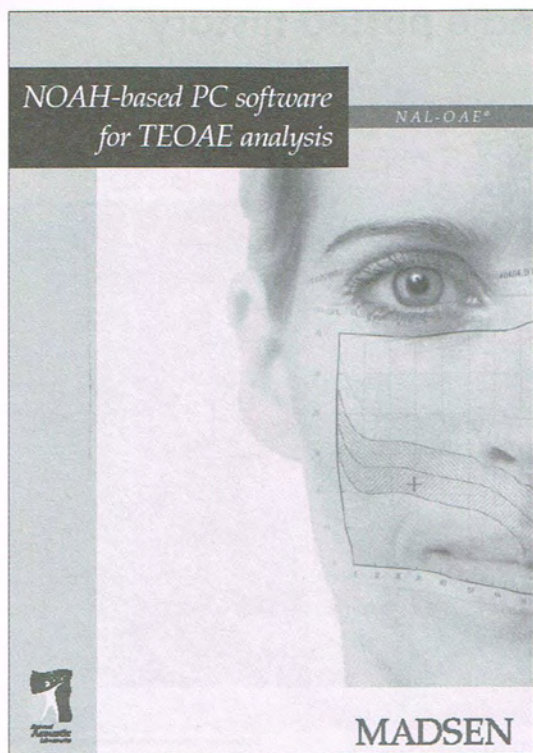
Australian population statistics for CEOAE emission strength and audiometry were generated, particularly paying attention to the frequency range of 1-4 kHz in both variables. It was clear that emission strengths declined by about 80% of their range before there was a significant risk for hearing loss. This is not to

assert, that the demonstration of audiometric notches are not also an early-warning sign, of sorts – because hearing disability does not necessarily accompany such notches when they first appear. Audiometric notches, however, indicate *existing* impairment, rather than predicted *future* impairment. Further, it was soon realised that the CEOAE test was faster than an audiometric test, and provided information that could not be obtained from a threshold test alone. CEOAEs may not provide inherently more information than DPOAEs, but for most applications so far, the latter has been constrained to producing audiometric-type information only for ready assimilation and comparison with audiograms.

In early 1999 Dan Zhou joined the NAL team to begin to develop software to make the NAL research results, particularly the population statistics, generally available and to develop the potential of the approach for early warning for hearing conservation application. Following the success of NAL-NL1 software for nonlinear hearing aid fitting, the NAL-OAE concept was developed, initially for streamlining analysis of transient otoacoustic emission records produced by the Otodynamics ILO-88/92 series data collection equipment. The important new feature of NAL-OAE was the ability to compare individual records with Australian population statistics we had built up over the decade for the variables (Waveform reproducibility and Coherent Emission Strength) we had used. Most important was the ability to compare any individual record with the emission strength versus age characteristics (see front cover), full knowing there is a 5 dB uncertainty associated with any particular result.

It is fair to say that the adoption of otoacoustic emissions by clinicians has been a slow process, possibly because the information was unfamiliar at first, to clinicians. To facilitate the fast acceptance of the NAL-OAE software, its capability was expanded to provide the most commonly accepted criteria for early detection – the Rhode Island criteria. In addition, just as hearing thresholds are usually categorised as “normal” or “raised”, so too has it been common to regard CEOAE emissions as “absent” or “present”. The essential progress that is incorporated within NAL-OAE has been to escape from this binary classification and recognise that CEOAE emissions vary over a continuum, and that “normal” emission strength varies smoothly with age. The NAL-OAE software thus indicates *normal limits* for CEOAE

“The important new feature of NAL-OAE was the ability to compare individual records with Australian population statistics...”



Flyer for promoting the NAL-OAE analysis software overseas. Courtesy of GN Otometrics.

at any age. By making this complexity of data available within an easy-to-use package, we hoped to accelerate the take-up of acoustic emission testing.

As a result of a visit to NAL by GN-Otometrics (then Madsen Electronics) in 1999, and subsequently one by Eric LePage to Denmark, preliminary discussions took place establishing a joint-venture with the view to developing and marketing the concept of early-warning of adventitious hearing loss, using the Madsen platform – firstly using

the Celesta OAE equipment, and subsequently the Capella equipment. A strong incentive to move in this direction was the audiometric database system (NOAH) integrated into the GN Otometrics system which the ILO equipment lacked.

The next two years resulted in more visits between Denmark and Australia, of Peter Johannessen and Dan Zhou with the view to resolving differences between the two primary commercial equipment models so that the Australian population statistics could also be applied to data obtained with the Capella. These visits also had the objective of preparing for the introduction of the new NOAH3 database system and to develop and provide NAL-OAE as

a NOAH module, and issues associated with HIMSA certification of the NAL-OAE module.

In parallel with the technical developments, legal and commercial issues associated with GN Otometrics marketing NAL-OAE Version 2 were considered and the resulting agreement was signed by GN Otometrics president Michael Brock on August 23rd, 2002 and Australian Hearing Acting Managing Director Neville Tomkins a week later.

This achievement has brought marketing activities, headed by Mr. Paul Morrison of GN Otometrics into full swing, drawing upon the expertise of all members of the combined team, including the graphics skills of John Seymour. October 10th, 2002 saw the official launch of NAL-OAE into the European market with other markets being planned. The desirability of catering for languages other than English has been the most recent activity at NAL, with Dan Zhou adapting his software for most European languages and GN Otometrics supplying the translations.

A bright future is anticipated for NAL-OAE, as new research results are incorporated. In general, NAL-OAE extends the previous scope of NAL research into noise-induced hearing loss and hearing conservation, by defining the new area of prevention of hearing loss based on the notion that, detected early enough, many of the widely-recognised toxic influences on the cochlear outer hair cells which generate the otoacoustic emissions may be more knowledgeably quantified and countered. The next few years should see NAL-OAE extended to include some promising developments in the area of brain-stem processing. It is hoped eventually to be able to distinguish between differences in CEOAEs due to environmental factors (noise, toxins) and those due to neuronal and genetic factors.

"A bright future is anticipated for NAL-OAE, as new research results are incorporated."



Signing of the licence agreement for marketing and distribution of the NAL OAE software for analysing otoacoustic emissions developed by Dr LePage and his team. Left: Mr Neville Tomkins, Managing Director, Australian Hearing. Above: Mr Michael Brock, President, GN Otometrics.

Effects of static ear pressure on click-evoked otoacoustic emissions

(The research is the experimental work required for completion of a Masters project of a student from Lund University)

Investigators: **Johannes Lantz and Eric LePage**

“... much effort has been put into determining whether otoacoustic emissions should be measured on people with deviant middle ear pressure”

Background: Ever since the otoacoustic emission measurement technique was developed, attempts have been made to describe how different middle ear configurations affect the otoacoustic emission measurements. Clinically, middle ear pressures different from zero are often encountered, and it is now well known that even relatively small tympanic pressure gradients alter the results from otoacoustic measurements in different ways. Since the signal pass through the middle ear system twice when measuring evoked otoacoustic emissions, once for the stimulus to reach the cochlea, and once for the response to return to the meatus, the accumulated middle ear effects may be substantial. Therefore, much effort has been put into determining whether otoacoustic emissions should be measured on people with deviant middle ear pressure, and what criteria to use for not doing so. Experiments have also been conducted to find ways of getting around the problem. Such “compliance maximisation” approaches are based on the theory that applying an ear canal pressure corresponding to the middle ear pressure achieves cancellation of middle ear stiffness offset.

Research Questions:

1. How much are click-evoked otoacoustic emissions (CEOAE) influenced by
 - variations of pressures within the middle ear?
 - variations of pressures within the external ear?
 - attempts to return compliance of the tympanic membrane to maximum to counteract its reduction by middle ear pressure?
2. Which CEOAE parameters are most influenced by these perturbations
 - waveform reproducibility, emission strength and emission latency?
 - which frequencies are most affected?
3. How completely is the perturbation produced by raised or lowered middle ear pressure removed by applying an equal (balancing) pressure in the external canal?
4. How much are the perturbations influenced by the middle ear and how much by cochlear processes?

Methods: Three non-invasive approaches have typically been used in otoacoustic emission research to manipulate the tympanic pressure gradients, pressure chamber, manual middle ear

pressure equalisation techniques – the middle ear pressure is manipulated by having the subject perform the standard Valsalva-, Frenzel- or Toynbee manoeuvres; and local application of external ear canal pressure – pressure gradients are achieved by locally inducing air pressure in the external ear canal. This is done by connecting the pressure vent of the probe to an air-pump and manometer. A huge benefit with this method is the high flexibility in pre-determining pressure levels. In terms of displacements and stiffness alterations of the tympanic membrane, positive middle ear pressure is considered satisfactorily simulated by applying negative ear canal pressure and vice versa. Maintaining a stable tympanic pressure gradient without leakage through the Eustachian tube is crucial since the collection of otoacoustic emissions does not allow for simultaneous middle ear pressure monitoring. Due to the anatomical characteristics of the Eustachian tube, relative middle ear over-pressure is particularly difficult to maintain and so carefully instructing the subject to avoid head movements and swallowing is advantageous.

For the experiments, thirteen healthy normal hearing volunteers, 22 to 38 years of age attended for testing. The included data was collected from a total of sixteen ears after a few exclusions of subjects. All ears included in the study had an overall otoacoustic emission waveform reproducibility exceeding 70 %. No subject included in this study had any known history of ear pathology. Five subjects were excluded due to (1) overall emission reproducibility being below 70 %, (2) inability to maintain the Eustachian tube closed or (3) highly compliant tympanic membranes.

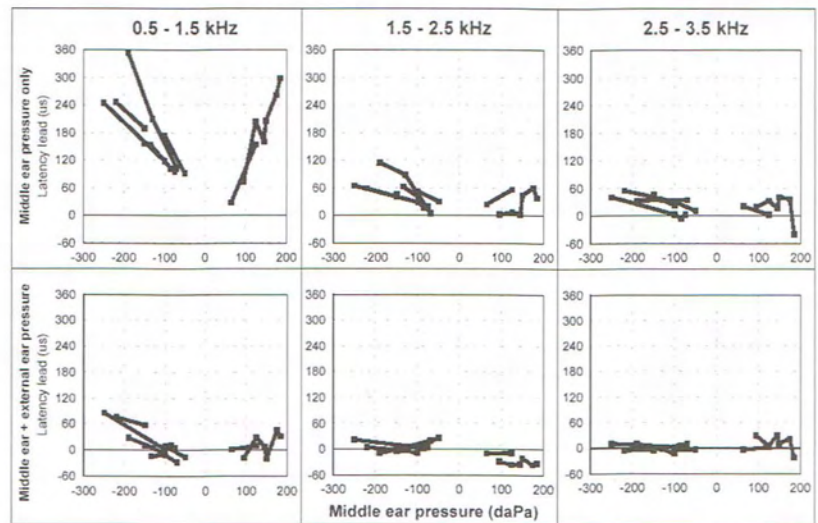
The GN Otometrics™ Capella™ unit was used with the Hortmann™ OAE-probe to collect the emission data using the standard “nonlinear” recording mode at 80 dB pk clicks. The Grason-Stadler™ TympStar™ Version 2 was used for tympanometry, as well as for pressurising the external ear canal. For the latter purpose, the tympanometer pressure tube was connected to the pressure vent of the Hortmann probe. The pressure was then applied using the manual mode of the tympanometer. Latency analysis was carried out in the PC-based otoacoustic emission analysis software NAL-OAE™ specially tailored by D. Zhou for this project. Tympanometry and occasionally equalisation of the middle ear pressure was carried out. A tympanometric peak pressure of ± 10 daPa was taken to be ambient. Two consecutive click-evoked otoacoustic emission measurements were made in ambient

pressure to serve as reference measurements. Then a sequence of click-evoked otoacoustic emission measurements was carried out with different positive and negative static ear canal pressures. The static ear canal pressure was varied positively or negatively in 30 daPa steps ranging from 0 daPa to a maximum of 180 daPa. Half the subjects were tested with negative pressure first and then positive pressure, and the other half were tested with positive pressure first and then negative pressure. The subjects were instructed to avoid any head-movements or swallowing, in order to keep the Eustachian tube closed. The middle ear pressure was assessed with tympanometry at some points during the test sequence to minimise the risk of additional influence of unknown middle ear pressure. Finally, a post-sequence otoacoustic emission reference measurement was made at ambient ear canal pressure. The recordings made with compliance maximisation were cross-correlated with the recordings made with net tympanic membrane pressure. For each of several frequency regions, latency shift was defined as the latency corresponding to the peak of the cross-correlation function.

Findings: The figure shows some representative CEOAE latency data. Increase in pressure, either negative or positive above ambient pressure has a marked effect upon CEOAE latency, particularly for the lowest frequency (1 kHz) band, the effect decreasing for the 2 kHz and 3 kHz bands. The change in latency (μ s) is in the direction of lead, the higher the pressure, the earlier the frequency component appears in the emission. The amount of latency lead decreases markedly with application of an external ear pressure set to be the same pressure (positive or negative) as indicated at the peak in the compliance curve obtained by tympanometry while the middle ear pressure is maintained.

An explanation of the latency shift phenomenon may be conceivable out of basic band-pass filter characteristics. When the stiffness of the middle ear system is increased by stretching the membranes with the appliance of static air pressure, the resonance frequency of this filter is shifted toward higher frequencies. Consequently, the phase shift associated with the resonance also shifts to a higher frequency. This means that the signal will pass through the system with less delay, and come out shifted in the time domain. A simple model shows that increasing the resonant frequency causes a time lead broadly centred on the original resonance, but extending down to low frequencies as well.

Significance: Audiometric measurements typically exhibit considerable variability and that variability often interferes with making clear conclusions as to cause and effect. An important feature of the CEOAE research carried out at NAL is the determination of latencies in



otoacoustic emissions, such as the latency changes registered with aging. Test-retest variability of otoacoustic emissions is up to approximately 5 dB on a day-to-day basis, however, much higher levels of variability have been seen in the longitudinal studies, including the orchestra project. The variability is greatest in the lowest frequency bands. Some of this variability could be due to variation of middle ear pressures in one measurement to the next influencing emission levels, even if the changes in latency due to middle ear pressures are relatively small compared with the latency changes due to aging (LePage and Murray, 2002). The recommendation which follows is at least the mandatory recording of tympanograms associated with each clinical otoacoustic emission measurement, and at most, the desirability of always carrying out a compliance maximisation procedure as part of the otoacoustic emission measurement, particularly when longitudinal data are obtained.

The upper panels show the effect, upon CEOAE latency, for three frequency bands, of varying middle ear pressure using Valsalva or other manoeuvres. The ordinate is the difference in latency from a pre-perturbation measurement and computed using modified NAL-OAE software. The lower panels show the partial success of cancelling the perturbation by application of an equal pressure in the external canal. Measurements from individual ears are joined by lines.

References:

- Avan, P., Buki, B., Maat, B., Dordain, M., & Wit, H. P. (2000). Middle ear influence on otoacoustic emissions. I: non-invasive investigation of the human transmission apparatus and comparison with model results. *Hear Res*, 140, 189-201.
- Hauser, R. (1992). The effect of systematic change in middle ear pressure on transiently evoked otoacoustic emissions – a pressure chamber study. *Laryngorhinootologie*, 71, 632-6.
- Konradsson, K. S., Svensson, O., Carlborg, B., & Grenner, J. (1999). Tympanic pressure gradients and otoacoustic emissions. *Ear Hear*, 20, 403-9.
- LePage, E.L. and Murray, N.M. (2002). Aging trends in human transient otoacoustic emission data: developing clinical estimates of declining outer hair cell redundancy. (submitted).
- Marshall, L., Heller, L. M., & Westhusin, L. J. (1997). Effect of negative middle-ear pressure on transient-evoked otoacoustic emissions. *Ear Hear*, 18, 218-26.
- Plinkert, P. K., Bootz, F., & Vossiek, T. (1994). Influence of static middle ear pressure on transiently evoked otoacoustic emissions and distortion products. *Eur. Arch. Otorhinolaryngol*, 251, 95-99.
- Trine, M. B., Hirsch, J. E., & Margolis, R. H. (1993). The effect of middle ear pressure on transient evoked otoacoustic emissions. *Ear Hear*, 14, 401-7.

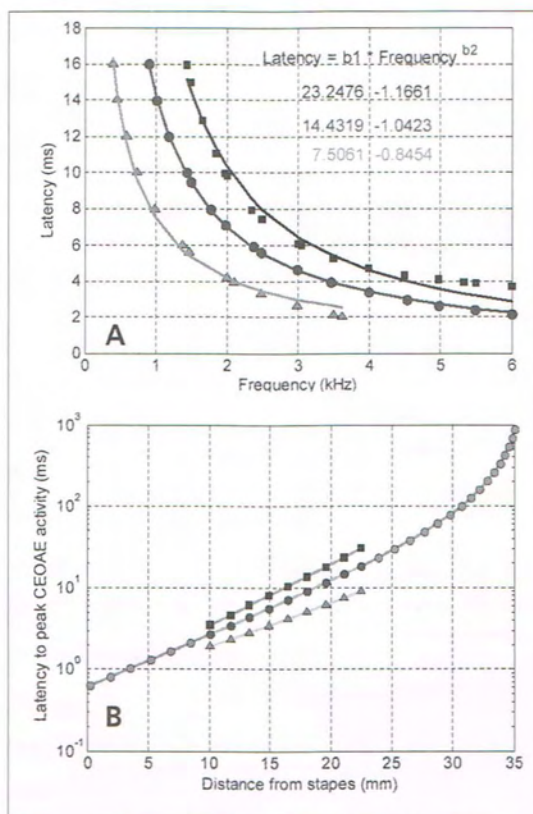


Figure 1. Panels A and B summarises how latency varies with age according to time-frequency plots prepared from human click-evoked otoacoustic emissions. In Panel A the centre curve joins the “peaks in the mountain range of activity” while the lines either side show where the mountain sides reach the plains. The inset numbers show the equation and the values of the coefficients b_1 and b_2 fit to the three curves. The lower panel B shows the same information plotted versus place along the cochlear partition, integrated with the human frequency-place map, providing an idea where different frequencies in the emission are generated.

unambiguous evidence of active behaviour in the cochlea and, except at very high sound levels, the passive mechanical action is regarded as linear. The “nonlinear test paradigm” most frequently used to obtain CEOAEs largely eliminates linear components of the response.

The otoacoustic emission is therefore thought to be the result of any nonlinear process of generation – and the only nonlinear process working at low levels is that due to outer hair cell (OHC) electromechanical activity and adaptation. This doesn’t mean that the otoacoustic emission is not strongly influenced by passive properties of the cochlear partition. The speed of propagation of the travelling wave set up by incoming sound to the place(s) of nonlinear interaction, and propagation of the reverse wave back to the ear canal is believed largely determined by the passive parameters. The timing primarily sets the latency of cochlear emissions which is thought to comprise equal times for forward and reverse propagation from each point. If it is true that the latency is largely controlled by the passive tapered properties of mass, stiffness and damping, then it is possible to think of the otoacoustic emission as some representation of the map of cochlear activity. This being the case, our longitudinal CEOAE data from our cohorts may potentially be used to derive a picture of how the cochlea ages in terms of changes to the distribution of activity as seen in our time-frequency plots (LePage and Murray, 2002).

Figure 1A shows the general “banana” shape relating latency (ms) to frequency (kHz) in the cochlear emission. Depicted are three curves which have been derived from time-frequency

Cochlear model for emissions

Investigators: Eric LePage, Åke Olofsson¹

¹ Dept. Technical Audiology, Karolinska Institute, Stockholm, Sweden

Background:

Click-evoked otoacoustic emissions (CEOAE) provide an important insight into the timing of cochlear mechanical activity and that timing is related to the place of origin of any frequency component in the emission. The mechanical performance of the cochlea is necessarily split up into a passive component and an active component. Otoacoustic emissions were the first

emission distributions averaged over 150 female subjects used as our estimate of the “least aged” set of data for establishing our map estimate. Figure 1B shows the effect of replacing the frequency abscissa with the human place-frequency map in terms of distance from the stapes. The human map extends for 35 mm which means that it extends both basally and apically from the frequency region which CEOAEs access (the three curves from Fig 1A in the centre region). Fig. 1B shows how CEOAEs provide a good measure of place estimation for the centre region of the cochlea. The curve in the centre extends beyond the mid-cochlear region, in both directions, since the human frequency-place map is defined for the whole 35 mm. If continuity is assumed the curve says that human CEOAEs mainly register activity in the centre regions of the cochlea, since the emissions with latencies less than 2.5ms are removed with the stimulus artifact. Note that the range of latencies is two orders of magnitude larger than the latency perturbations due to pressures in the middle ear (Lantz & LePage, 2002, this report).

Research questions: There is considerable interest in determining how much each of the linear and nonlinear components influence the emission. In a previous NAL annual report (1998/99) we investigated introducing artificial OHC lesions into this model. Continuing the exploration, we ask:

1. It is possible to reconcile the model with human responses?
2. What is the likely balance between passive and active contributions to explain the ageing curves previously shown to affect emission strength and latency?
3. What is the effect of varying the passive tapering properties of the model?
4. What procedure is necessary to systematically adjust the parameters of the model in order to find a set which most closely mimics the range of latencies observed in human CEOAE?

Procedures: Mathematical models of the cochlea are available in a impressive array of configurations from those which represent just the one-dimensional features of the cochlear partition, mass stiffness and damping, to three-dimensional finite element models which attempt to cope with the complex structure of the arches and cells taking into account mechanical strains created in other planes as the basilar membrane moves transversely. Since OHC activity was generally accepted in 1980, mathematical models have sought to describe, and been more

generally successful at describing, the displacement and velocity of the vibrating structures and how the level of activity modifies the vibration patterns, the neural excitation patterns, and of course otoacoustic emissions. Remarkably, the introduction of OHC activity into models, has resulted in significant improvement in the accuracy of simulating measured dynamic characteristics, particularly the timing and phase characteristics. The objective has been to use a basic single delay line model, with special provision made for OHC activity to modify the vibration of the basilar membrane.

For this purpose it has been found advantageous to adopt the discrete time domain model (Nilsson, 1977) which has been re-formulated using the wave-filter approach (Fettweis, 1971), by Olofsson (1982) and subsequently by Strube (1982). This has been reimplemented as a Mex (.dll) file which is compiled by and called from Matlab™. The result is a computationally efficient, flexible modelling environment which runs under Windows and which offers advanced displays of model properties. The single-dimensional delay-line model is made up of 350 segments, of the passive structure shown in Figure 2. Panel A shows the basic delay-line configuration for each segment. The values of R , L and C are all tapered with length along the cochlea, so as to provide tuning over audio-frequencies. Panel B shows the wave-filter representation of the same segment, using serial and parallel adaptors. The parallel adaptor contains branches for the reactive components, L and C . Not shown are the active elements in the model which are schematised in Figure 3. The output in Figure 3 is used as a local scaling of the stiffness constant which acts on the signal coming out of the R_c -port in Figure 2B. Each segment represents the combined activity of 0.1 mm or about 30 OHC. Segment 1 is located at the stapes and the pressure signals derived from this segment constitute sound signals (those arising from higher segments being taken as otoacoustic emissions). The complete model contains extra segments for middle and external ear, but these are omitted here.

In bare essentials, Figure 3 shows that the vibratory displacement of the basilar membrane, high-pass filtered to mimic the relative movements of the basilar and tectorial membranes, causing mechano-electric (forward) transduction in which the OHC depolarises (represented by the sigmoid shaped curve). This is a normal hair cell response. What makes the OHC unique is that they respond with electro-mechanical (reverse) transduction. Figure 3 indicates how this OHC “kick” is fed back to the motion in such a way that the net stiffness of the whole structure is slightly reduced.

Sound entering at segment 1 progressively stimulates OHC activity as waves pass down the whole length of the cochlear and, returning to

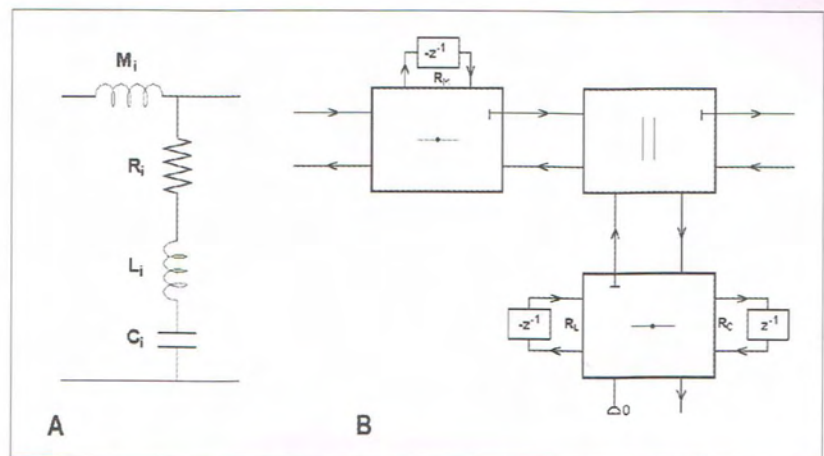


Figure 2. The basic configuration of the 1-dimensional delay line model – the passive components. The equivalent circuit of each segment contains two masses (L), one stiffness component (C) and one damping component (R). The second panel shows the wavefilter approach to the implementation of the model.

the stapes, the reverse waves interacting with the forward waves at each point. The model is quite realistic in that OHC activity boosts the BM displacement at each segment. While any stimulus type can be used, several parameters of the model can be measured at each segment (viz. displacement, velocity, pressure, even neural excitation). If two tones are presented, a family of distortion products are produced by nonlinear interactions in the model. Likewise, by delivering a click sound stimulus the result is “transient OAE” strongly reminiscent of human CEOAEs. The beauty of the model is that just as a clinical otoacoustic emission is the result of signal averaging, such signal averaging can be applied to the model with the same procedures used in the clinic and the results may be analysed, complete with stimulus artifact removal, and presented in exactly the same way with the same Matlab software.

The active response functions (not unlike nonlinear processing in hearing aids) use different charging and release times (ms). The active response is typically given a fast attack (charging) time and a slow release time, there is also a time constant for “dc-shift”, a limit “ c ” in $[0,1]$ ($=C_{lim}$) for controlling the amount of stiffness shift allowable, and a factor “ $Rohc$ ” in $[0,1]$ which controls the amount of active kick supplied by the OHC. In addition the model has very considerable flexibility in the kinds of tapering of the passive properties, e.g. how much the resistance R is tapered across segments.

The approach taken has been to explore the parameter space by systematically varying each of the key variables in nested loops for attack time, release time, dc-time constant, the limit “ c ” and of course the value of “ $Rohc$ ”. Each of these five parameters, respectively, was varied over three values according to the following sets:

Tchg = [.1 1 10] ms; Trel = [100 50 10] ms;
Tdc_disp = [10 5 1] ms; Clim = [.25 .5 .707];
Rohc = [0.7 0.8 0.9]. In addition these batch

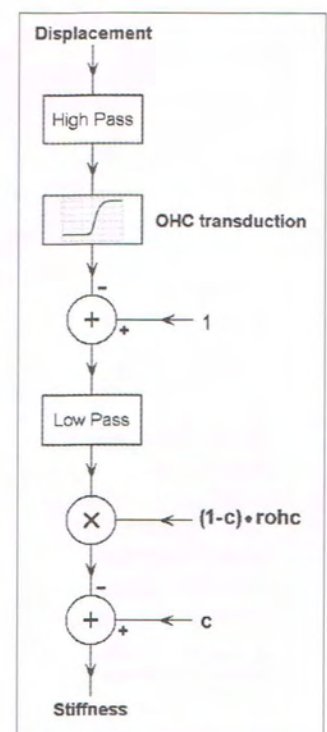


Figure 3. The model also contains an active component to mimic the response of the outer hair cells. This is depicted schematically as a vibratory displacement being forward-transduced, then the cell actively responds mechanically with a “kick”, the net result being to change the stiffness of the basilar membrane. The end result is cochlear amplification (not shown here).

procedures were executed for differing values of tapering cochlear resistance, but only one value of tapering is shown here. For each of these sets of parameters, the model was run for long enough (five repetitions of the “nonlinear” stimulus requiring billions of floating-point operations) for the initial transient response to settle down. The averaged CEOAE response (output from Segment 1) was analysed using our time-frequency analysis methods (LePage and Murray, 2002) to record the resulting latency vs frequency characteristics. The data were fitted to the equation

$$\text{latency (ms)} = b1 \cdot \text{frequency (kHz)}^{b2}$$

Results: The results were sorted in order of the nested loop parameters which give the latency versus frequency characteristics most like that of the “pristine” cochlea (Figure 1A) — i.e.

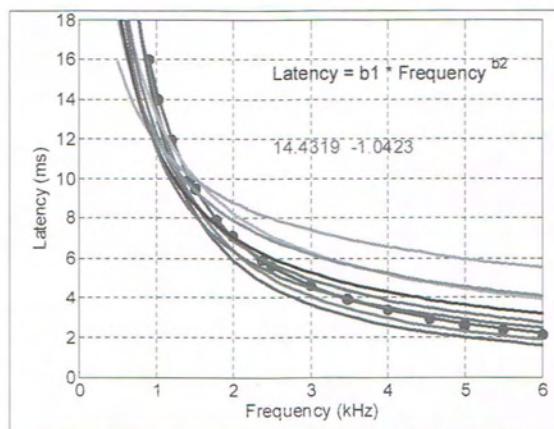


Figure 4. The curves for latency versus frequency which are the optimal result of the cochlear model. Only these eight best curves, corresponding to short attack and long release times can mimic the latency behaviour of the human otoacoustic emission, shown superimposed upon the central curve from Fig 1A.

the most positive values obtained of $b1$ and the most negative values of $b2$. The first eight rows in the table are shown in Table 1. Figure 4 shows the resulting curves for the latency specified by these near-optimal values of $b1$ and $b2$ superimposed over top of the central curve taken from Fig. 1A. In respect of Table 1, all the resulting curves provide some similarity to the curves of Fig. 1A. However, what is quite remarkable, is that after long-term exploration of the parameters of the model, the optimal results in the top eight rows are the only ones which approach similarity to the latency curve for what we believe is close to the pristine human cochlea. These top rows tend to have very short charging times (0.1ms) and very long release times (100ms). The model outcomes do not appear to be particularly sensitive to Rohc, or Tdc_displacement, but appeared to coincide with allowing more flexible conditions of “c” allowing stretching of the map. Evidently the

best possible result from adjusting the multiple dimensions of the parameter space cannot produce a greater spread of latencies from low frequency to high than the live case.

Conclusion: A simple one-dimensional delay line model for cochlear mechanics, to which is added a component designed to mimic outer hair cell activity with differing charge and release times is found to also mimic quite well the latency versus frequency curves applying to human click-evoked otoacoustic emissions. The bulk of solutions of the model are poor (i.e. they produce low latencies) at low frequencies. Indeed the very best performance which could be coaxed out of the model could not produce longer latencies than seen in human emissions at low frequencies. The result supports a recent assertion that the spatial layout of frequencies in the mammalian cochlea confirms to some form of stronger constraint (LePage, 2002).

Significance: The analysis of data as complex as click-evoked otoacoustic emissions can be performed in a host of ways, each of which may provide some insight. However, experimental work tends to find considerably better understanding in terms of mathematical models. The use of this simple model to mimic a very important characteristic of CEOAEs, its latency versus frequency, may also provide a very important insight in respect of the effect of aging upon latency and the result here fits in with the overall decline in latency with age (LePage and Murray, 2002).

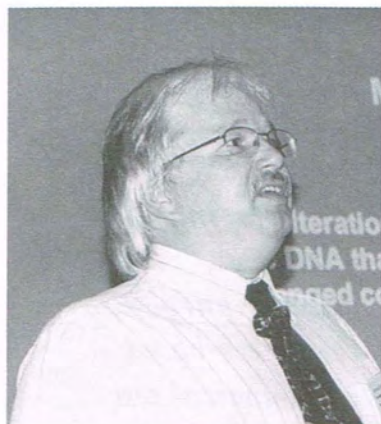
References:

- Fettweiss, A. (1971). Digital filter structures related to classical filter networks. *Arch. Elek. Übertragungst.* 25, 79-89.
- Hawkins, J.E. Jr. and Johnsson, L.-G. (1976). Patterns of sensorineural degeneration in human ears exposed to noise. In *Effects of Noise on Hearing*, edited by Henderson, Hamernik, Dosanjh and Mills. Raven Press, NY. 91-110.
- LePage, E.L. (2002). The mammalian cochlear map is optimally warped. (submitted).
- LePage, E.L. and Murray, N.M. (1993). Click-evoked otoacoustic emissions: comparing emissions strengths with pure tone audiometric thresholds. *Aust.J.Audiol.* 15:9-22.
- LePage, E.L. and Murray, N.M. (2002). Aging trends in human transient otoacoustic emission data: developing clinical estimates of declining outer hair cell redundancy. (submitted).
- Nilsson, H.G. (1977). *A model study of the auditory periphery*. Dissertation from the Division of Physiological Acoustics, Department of Physiology, Karolinska Institutet, Stockholm, Sweden.
- Olofsson, M.Å., (1982). Distortion generation in a model for peripheral haircells. Nordic Acoustical Society meeting, Stockholm, Sweden, 407-410, (in Swedish).
- Strube, H.W. (1982). A computationally efficient basilar membrane model. *Acustica*, 58, 207-214.

Tdc_disp	Trelease	Tcharge	Clim	Rohc	b1	b2
1	100	0.1	0.5	0.7	13.649	-1.1
10	10	10	0.25	0.8	13.215	-1.169
10	100	0.1	0.5	0.7	13.171	-0.675
1	100	0.1	0.25	0.9	12.256	-0.83
5	100	0.1	0.5	0.7	11.994	-0.603
5	100	1	0.25	0.8	11.823	-0.426
10	10	1	0.5	0.7	11.563	-0.717
1	10	10	0.25	0.7	11.457	-0.852
1	50	1	0.25	0.8	11.452	-0.577

Tables 1. This shows the eight best solutions (respectively) of the model after searching the whole parameter space ($3^5=243$ times the model was executed). Only the optimal value of these ($b1 = 13.6$ and $b2 = -1.1$) comes close to the latency-frequency curve of the “pristine” human cochlea ($b1 = 14.4$ and $b2 = -1.04$).

Causes and prevention of hearing loss: global trends in industrial and leisure noise, interactions, definitions and strategies – a one day colloquium



Dr Henrik Dahl



Dr Donald Henderson



Dr Ramesh Rajan

A one-day colloquium on the causes and prevention of hearing loss was held at NAL on 23 October, 2001. The Colloquium, which was organised by Dr Eric LePage, Senior Research Scientist in Hearing Loss Prevention Research, highlighted the complexity of hearing loss mechanisms in general, and noise-induced loss in particular. The colloquium was divided into four Sessions: 1. Causes of Hearing Loss; 2. Efferent/OHC-Mediated Protection Mechanisms; 3. Preventive Medicine Issues in Dealing with Noise-Induced Hearing Loss; and 4. A Panel Discussion between speakers and the audience. The following summary of the major points discussed is provided by Dr LePage and Dr Narelle Murray.

Session 1, entitled *Causes of Hearing Loss*, was moderated by Dr Pat Mutton of the Deafness Unit, the New Children's Hospital, Westmead. The first paper was by **Dr Henrik Dahl**, Principal NH&MRC Research Fellow at the Murdoch Children's Research Institute and Associate Professor in the Department of Paediatrics at Melbourne University. Dr Dahl's topic was *Genetic predisposition to hearing loss* and covered the underlying causes of deafness with special emphasis on the genetic aspects.

Of special interest is the recently identified connexin 26 gene which codes for a protein involved in communication between cells. Dr Dahl showed that research has demonstrated that connexin 26 mutations are the cause of deafness in nearly one-fifth of all children with prelingual hearing loss. He discussed the potential benefits and problems of work in genetics, including ethical aspects.

Dr Donald Henderson, Professor of Communication Disorders and Sciences and the Co-Director of the Center for Hearing and Deafness at the State University of New York at Buffalo was the second speaker, presenting two papers. The first was titled *Do our Noise Standards Reflect What we know about Noise?* and firstly, reviewed key scientific findings that have implications for noise standards. Specifically, he reviewed the validity of the A-weighting practice; the limitation of a simple energy metric that ignores the temporal variations during a noise exposure, and, the development of the concept of kurtosis as a factor when assessing the potential hazards associated with an exposure. Secondly Dr Henderson reviewed a series of noise interactions including the worker's use of therapeutic drugs such as aminoglycosides or

cancer drugs while they are exposed to noise as well as the positive interaction of some industrial solvents with noise.

Dr Henderson's second paper was titled *The Inner Ear's Response to Traumatic Noise: New Opportunities for Protection*. He stated that it has been known for many years that noise-induced hearing loss (NIHL) is associated with a constellation of pathological changes in the inner ear. However, while the outer hair cells appear to be the most vulnerable, noise also causes damage to the inner hair cells, VIII nerve fibers, supporting cells and blood supply. Dr Henderson reviewed new research showing that a major factor in these cochlear pathologies is the reactive oxygen and nitrogen species generated by high-level noise exposures. He also explained that the reactive oxygen species are associated with a process of active cell death – necrosis and apoptosis – and that the continued growth of hair cell lesions after an exposure are mediated by progressive apoptotic changes moving towards the base of the cochlea. This knowledge of hair cell death has opened the exciting possibility of preventing cochlear damage by antioxidants or intervention with anti-apoptotic drugs. A series of experiments was reviewed showing that bolstering the cochlea's level of glutathione reduces the effects of a noise exposure. Earlier research on protection used the round window as a point of treatment. New research with certain antioxidant drugs that are safe and easily tolerated when given systemically can also reduce the effects of noise. Since the period of cell death continues for days after the exposure, there are possible anti-apoptotic drug that can be used to limit the cochlear lesion. Dr Henderson discussed the results of this research in terms of their clinical implications.

Session 2 was moderated by Dr Suzanne Purdy, Senior Research Scientist at the National Acoustic Laboratories and was entitled *Efferent OHC-Mediated Protection Mechanisms*.

The first paper in this Session was presented by **Dr Ramesh Rajan** of the Department of Physiology, Monash University, Monash, Victoria. Its title was *Protective functions of the efferent pathways to outer hair cells*. Dr Rajan firstly reviewed the evidence that, in animal experiments at least, the olivocochlear efferent pathways from the brainstem to cochlear outer hair cells can protect hearing sensitivity from desensitizing effects of loud sounds and discussed the pathways and characteristics of the

protection. He presented data from experiments where loud pure tones were used to produce temporary threshold shifts (TTS). When presented by themselves, only the crossed olivocochlear bundle (consisting almost totally of that component known as the crossed medial olivocochlear system [CMOCS] and running to the outer hair cells) is involved in protection which is evoked by binaural but not monaural loud sound. Dr Rajan demonstrated that such CMOCS protection is graded to the amount of TTS that would otherwise occur, both at any one cochlear region at which hearing sensitivity is measured as well as across all affected cochlear regions. Such protection appears to be targeted to the most sensitive regions of the cochlea, and is most easily evoked by the tones most likely to cause TTS, but can be evoked by tones at all frequencies. This protection acts solely through lower brainstem pathways, but there are also possible modulatory influences from higher brain centres. Secondly, Dr Rajan discussed extensions of this protection to other more common conditions, when loud sounds occur in noisy backgrounds or when the loud sound is spectrally complex and demonstrated that both crossed and uncrossed olivocochlear pathways act under such conditions to protect hearing sensitivity. He also noted the variety of other types of trauma and conditions under which the olivocochlear pathways have now been shown to also protect hearing sensitivity. Thirdly, he presented recent data from other experimenters showing a functional way to measure the strength of olivocochlear effects on OHCs and how this appears to provide a mechanism that allows prediction of the vulnerability of an individual's ear to traumatic loud sound.

The second paper in this Session was presented by **Dr Eric LePage**, Senior Research Scientist, National Acoustic Laboratories and was titled *Protection of cochlea at the mechanical level, temporary and permanent shifts in evoked otoacoustic emissions*. Low hearing thresholds occur when the basilar membrane frequency response is sharply tuned: the nature of this nonlinear mechanical response may, in spatial terms, be regarded as the focussing of low level acoustic energy down to a few inner hair cells for maximum effect. The effect of loud sound is to cause the vibrations of the structures to approach the dimensions of the cells themselves, resulting in catastrophic shear forces. To internally protect the structures, the efferent/OHC response to

loud sound brings about a rapid defocussing of the energy so that it is effectively distributed over a long length of the basilar membrane. In the same process the mapping from frequency to place is shifted – dubbed the “half-octave shift”. Dr LePage showed that by direct mechanical measurements in guinea pigs the way the ear responds to loud sound is quite chaotic and highly dependent upon the history of the exposure. He stated that evoked otoacoustic emission measurements in humans have some advantages over animal models because they allow a better gauge of how rapidly the OHC respond to a tone burst. By looking at the latency of click-evoked emissions an overview the mapping changes can be gauged as they occur, using movies made of time-frequency displays. Not unsurprisingly the “Temporary Emission Shifts” (TES) vary widely in different individuals, some displaying a “tough” response little affected by the burst, others displaying a much more fragile response. Since these differences can be measured for much smaller doses than are required to produce a TTS, it can be shown how CEOAEs may become useful clinically to assist in the rapid identification of susceptible people.

Session 3 entitled *Preventive Medicine Issues in Dealing with Noise-Induced Hearing Loss*, was moderated Dr Jenny Rosen, Head, Audiology, Hornsby Kuring-gai Hospital.

The first paper in this Session was presented by **Dr Ross Dineen**, a clinical Audiologist for 23 years and partner in Milhinch & Dineen Audiology, Melbourne, Vic. He presented *Noise And Hearing In The Building And Construction Industry: A Study Of Workers' Views On Noise And Risk*.

Dr Dineen discussed the fact that there is ample evidence that noise constitutes a significant risk to the hearing health of workers in the building and construction industry, with demographic studies having shown the incidence of noise-induced hearing loss is as high as 60% in noisy workplaces. However, hearing conservation as currently practised appears to be having little impact on the level of hearing injury to workers. Studies of the construction industry in Australia have indicated that there is a low awareness of the risks posed by noise, with consequent minimal self-protective behaviours (Milhinch, Dineen & Doyle, 1997). Investigations into the extent of use of personal hearing protection indicate that workers' perceptions of risk, and



Dr Eric LePage



Dr Ross Dineen



Dr Narelle Murray

"Much of what provides incentive and excitement to life is loud sound – from racing cars, to boom boxes, to pop concerts."

their need for communication are important aspects of the decision to use or not to use hearing protection (Lusk & Keleman, 1993; Lusk et al., 1994).

Dr Dineen described research he had undertaken to identify some of the reasons why noise injury in the building and construction industry persists, despite the existence of Hearing Conservation Regulations in Victoria since 1978. More than 150 workers on Australia's largest construction site, the Crown Casino in Melbourne, participated in the study. Data were obtained in two ways: quantitative measures were recorded of sound levels in the work environment and the average daily noise dose to which workers were exposed; qualitative data were obtained from focus group discussions. In particular, workers' views of the relationship between noise, hearing and their own hearing safety behaviour were sought, as a means of gaining a better appreciation of their behaviour in the workplace.

The knowledge and experience of workers from within the construction industry was sought to help develop new practical noise management strategies, which might be willingly used, by both workers and management. An education program designed to address problems relating to workers' identification and management of noise hazards prevalent on large building and construction sites was developed (Dineen & Reid 1999). A subsequent study evaluating the efficacy of the education program in influencing workers' perception of noise hazards and their propensity to take protective behaviour was carried out.

The second paper of Session 3 was on *The effects of leisure noise in young people and a nine year longitudinal study of orchestral musicians* presented by **Dr Narelle Murray**, Research Scientist from the National Acoustic Laboratories

When examining the ISO1999 (1990) and the more recent ASNZ-1269 (1998) model it is clear that the primary components determining hearing threshold level over time are Noise-Induced Permanent Threshold Shift (NIPTS) and Hearing Threshold Level associated with Age (HTLA). Overwhelmingly, occupational noise is treated as the primary cause of NIPTS, while the aging component is determined to be due to other mostly non-noise-specific aging factors. Dr Murray showed the results of exploring the properties and possibilities of using

transient evoked otoacoustic emissions (TEOAE) to examine both decline in hearing acuity with age and noise. One of the chief findings is that otoacoustic emissions appear to be more sensitive to early stages of ear damage due, not just to noise, but all contributory factors leading to loss of outer hair cell activity. On a percentile population basis, otoacoustic emissions are seen to decline in strength long before the pure tone thresholds (three-frequency-average 1, 2 and 4kHz) enter the critical region deemed to correspond to a mild hearing impairment. Data were shown that indicated that leisure noise in young people was already having a significant impact on the distribution of emissions as a function of age. The implication of this is that leisure noise constitutes a modern complication in the assumption that NIPTS is primarily occupational. Dr Murray also presented data which showed that TEOAE clearly identify significant differences between young people with no hearing impairment but with differing noise profiles. These data indicate in particular that the damaging effect of typical usage of personal stereos by young people, was at least as marked as that due to working in industry. On the basis of just this one component of leisure noise exposure it is predicted there would be a steady increase in the number of young adults with NIPTS, a prediction which has now been confirmed. This added sensitivity of TEOAE to ear damage has been utilised in a longitudinal study of moderately noise-exposed individuals – players in the Australian Opera and Ballet Orchestra to look for evidence which might serve as the basis of a hearing conservation program based not just upon sound levels in that workplace, but by monitoring with TEOAE to provide early warning of ear damage before emissions decline to critical levels.

The final paper of the Colloquium was presented by **Dr Eric LePage**, Senior Research Scientist at the National Acoustic Laboratories and was entitled *Non-behavioural solutions to behavioural problems: how soon can we expect them? Limitations of current definitions and testing procedures: application of principles of preventive medicine*.

This talk was about the key factors impeding progress in promoting hearing loss prevention. Reports suggesting a rise hearing loss in young people were considered, together with a computer model developed in 1994 which predicted that the distribution of hearing loss as a function of age would change to a peak around

ages 30 to 40 rather than past age 60.

The consequences that this redistribution have for occupational noise standards and issues of compensation for hearing loss were discussed.

Much of what provides incentive and excitement to life is loud sound – from racing cars, to boom boxes, to pop concerts. Dr LePage posed the questions: How realistic is it therefore to get young males in particular, the segment of the community most at risk, to change their behaviour? Is introducing the kind of awareness education program lately used to limit the incidence of skin cancer going to be effective to stem any epidemic of hearing loss? If not, why not? Is there any evidence that the situation regarding the growth of leisure noise exposure is not static? Do we have a run-away situation where the now documented growth of hearing loss in young people is leading them to require higher sound levels still for comprehension? What should be our response to the apparent need to re-establish norms for normal hearing levels at 4 or 6 kHz? What would be the first manifestation of an epidemic of hearing problems? Can non-occupational behavioural issues be addressed by legislation, unless educational achievement is affected?

To make headway leads full circle to consider the nature of hearing impairment. Why is the onset so subtle? Does the answer have anything to do with physiological correlates of the equal-energy principle, e.g. the accumulation of cellular stress over an 8 hour day followed by recovery once exposure ceases? A group of potential drugs has been presented which may lessen the accumulation of cellular stress and the long-term conversion of TTS to PTS.

We live in an age where ready-fix pharmacological solutions to human health problems are highly accepted. It would be amazing if the issue turned out to be as simple as the introduction of glutathione or a precursor of glutathione into the diet or the water supply! Yet it may be decades before hearing can be protected by taking a drug without major side-effects. In the meantime we are obliged to consider why hearing conservation programs which are based upon monitoring of hearing threshold levels are not seen as strongly effective – we submit the reason is that the warning is not targeted at the individual, and the warning comes too late. Dr LePage advocated a two-pronged interim solution. Firstly, a continuation of urging the lessening of noise exposures

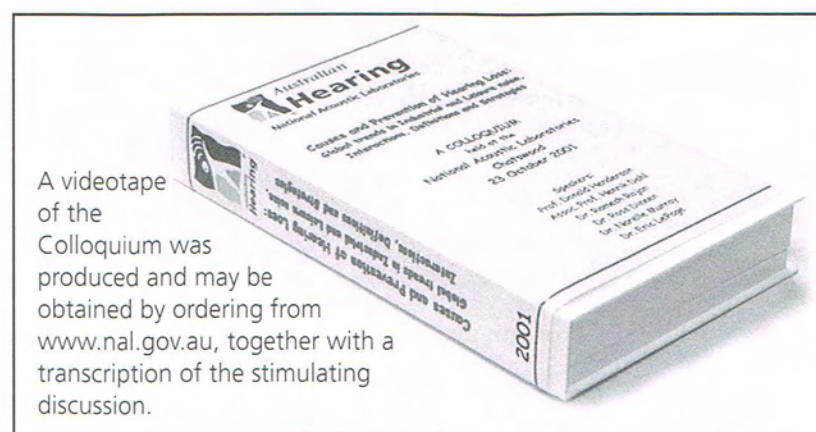
“... the damaging effect of typical usage of personal stereos by young people, was at least as marked as that due to working in industry.”

realising that this measure will still do very little to save the hearing of the most susceptible or genetically pre-disposed individuals. Secondly, an advocacy of the early-warning properties of otoacoustic emissions as having potential behaviour modification properties. Dr LePage proceeded to demonstrate an interpretation of TEOAES in terms of an effective fuel gauge measuring the decline in outer hair cell activity with age and long-term noise exposure. He also discussed work in progress to refine measures of susceptibility based upon measuring the effectiveness of efferent response.

A Panel Discussion between the audience and all the speakers followed. This was moderated by Dr Harvey Dillon, Research Director of the National Acoustic Laboratories.

Questions discussed were:

1. What should clinicians and hearing conservationists be doing in the light of recent developments?
2. What should society be doing to better prevent HL? How to stimulate behavioural change?
3. What research is needed to close the gap between knowledge of cochlear processes and the prevention of hearing loss?



A videotape of the Colloquium was produced and may be obtained by ordering from www.nal.gov.au, together with a transcription of the stimulating discussion.

Research Director's Overview

Once again I am pleased to present the Annual Report of the National Acoustic Laboratories. As in previous years, the report includes a representative sample of current projects, each report giving enough detail to be of interest to clinicians and researchers in our field, but hopefully expressed in a manner that is accessible to the interested lay reader. At the current time we have a total of 40 projects either just starting, in progress, or just completed. Each of these projects is helping to progress our central mission of either finding ways to prevent hearing loss, or improving the methods used to habilitate hearing in children or rehabilitate hearing in adults. Underpinning both of these goals is the need for informative and efficient methods to assess all aspects of hearing.

Hearing Assessment

There is a long and successful tradition of NAL procedures and findings being adopted by clinicians world-wide. The routes by which research outcomes are adopted can, however, be tortuous, slow, and uncertain, so an exciting development over the past year has been the development of the general purpose audiological test system currently referred to as HearLab. This apparatus consists of a general interface unit into which any of the usual audiological transducers can be connected, plus a high-speed connection to a personal computer. Progressively over the next decade, research projects leading to clinical procedures will go through a final software implementation phase in which the procedure is implemented in a software package that works in conjunction with HearLab. NAL would also be open to implementing the results of other research groups into software modules that work with HearLab. We anticipate that HearLab will be distributed by a major supplier of audiological test equipment. The first module will be a cortical evoked response test unit, specifically optimised to evaluate the effectiveness of hearing aids fitted to infants.



Hearing loss prevention

In most countries, the effectiveness of hearing protective devices is measured after the experimenter carefully places the device on the head or in the ear of the subject. The object is to ensure that the device is "properly" fitted. In Australia, the practice has been to have subjects fit the device themselves. The object of this is to have the device adjusted in the most realistic manner possible. An interesting analysis in this report shows that some subjects receive far less attenuation than the average attenuation, though none receive far more attenuation than average. Presumably this result reflects either poor placement of the device by some people on some occasions, or a design that is incompatible with achieving a reasonable attenuation on all people. Future work in this area will attempt to delineate what characteristics of hearing protectors are associated with reliable or unreliable protection when hearing protectors are used in their usual manner.

Of course, it does not matter how well hearing protectors are designed if they are not worn. One research study completed during the year investigated whether receiving an audiometric assessment and associated information about hearing status would cause people to take greater steps to protect their hearing. On the basis of self-report, there was a significant change in their willingness to protect their

hearing, which was sustained for the full 12 months of the study period. The two different methods of assessing hearing loss, and displaying this information to the subjects did not, however, have any differential effect on the extent to which subjects sought to protect their hearing in daily life.

It is common-place to hear music emerging from the headsets worn by people in public places and on public transport. It is reasonable to wonder whether such people are causing themselves to acquire a noise-induced hearing loss. A brief report in this Annual Report indicates that for many of these people, the daily noise dose received is sufficient to eventually cause permanent hearing damage.

Hearing rehabilitation procedures and devices

Our work on the benefits of sound-field FM amplification in classrooms concluded during the year. A paper in last year's report showed a marked and highly significant increase in the attainment of educational goals during periods when the system was installed in classrooms compared to periods when it was not installed. The companion paper in this report shows the effects of the system on classroom behaviour, as reported by the teachers, and the opinions of the children themselves about the system. Given the very positive results and opinions, it is a worthwhile goal to have sound-field amplification installed in every classroom.

A long-term study investigating how amplification should be altered in different background noises also concluded this past year. The study showed the clear need for different amplification characteristics in different environments, and showed how the amplification characteristics should vary with the sound spectrum of the noise, the overall level of the signal and the noise, and the goals of the hearing aid user. The results are relevant both to hearing aids with automatic adaptive noise reduction systems and to multi-memory hearing aids controlled by the aid wearer.

An intense effort was put into collecting data on the effects of hearing loss on the ability to extract useful information from audible speech. This very large experiment is collecting data from 80 subjects using speech filtered in a variety of ways, at different presentation levels, and in quiet and in noise. In addition, measures of frequency selectivity are being obtained to help interpret the findings. These results will be used to modify the Speech Intelligibility Index so that it can better predict intelligibility. Such modification is especially needed when hearing loss gets into the severe range at any frequency. This modification, when combined with the results of other recent experiments at NAL, will provide the data needed to derive the NAL-NL2 update of the current NAL-NL1 prescription procedure. Papers in this report give the background to the large experiment and indicate what other factors will be taken into account in the forthcoming revision.

Two other papers in this report reflect our approach of continuous (or at least cyclic) improvement. In one paper, our earlier procedure for assessing candidacy for multi-memory hearing aids is critically assessed by comparing predicted candidacy with actual candidacy experienced when the procedure was used in routine clinical practice. In the second paper, we investigate whether the amount of gain prescribed by NAL-NL1 matches the preferences of subjects. This is accomplished by noting the manner in which hearing aids are routinely fine-tuned by clinicians in response to comments by clients. These projects, like many before them, would not have been possible without access to practicing clinicians and their clients. We are grateful to Australian Hearing for kindly providing access to its clinical staff within its network of hearing centres for these two projects.

The special needs of children continued as a focus of our work. One paper in this report reviews the fitting and subjective evaluation methods we recommend when applying the NAL-NL1 prescription formula to children. Although the methods for achieving a desired

real-ear target in children of any age are now clear, there remains some uncertainty in what those real-ear targets should be. Research into this issue will therefore remain a priority for us for the next several years. Although not included in this report, our work continued on using evoked cortical responses to evaluate the effectiveness of hearing aid fitting in infants. Good progress has been made and we have finalised the statistical procedures behind the automatic interpretation of the results. This project will feature in next year's Annual Report.

Recognition

Dr Teresa Ching was guest of honour/keynote speaker at the Triennial Swedish Combined Audiological conference. This is a double honour for NAL as their last keynote speaker was also from NAL. Dr Ching also presented her research results on the combined use of hearing aids and cochlear implants in children and adults to clinicians in Japan. This Bimodal Hearing Seminar, which attracted participants from all four islands in Japan, was unusual in that Dr Ching was the only speaker. Various other NAL scientists were invited, expense-paid speakers at other overseas conferences during the year.

Recognition also occurred in the form that we value the most. A recent survey of clinical practice in the USA revealed that NAL prescription procedures are now so extensively adopted that they were the first *and second* most widely used procedures for prescribing hearing aids. Another clinician survey, also in the USA, revealed that the COSI procedure, devised by NAL in conjunction with Australian Hearing clinicians, was the most-used method for evaluating the outcomes of hearing aid fittings. These findings are particularly satisfying because NAL procedures are usually the practical culmination of a number of research projects. Many NAL researchers therefore find their work being used each time a hearing aid is prescribed or evaluated using a NAL procedure.

Contract research

As foreshadowed last year, several contract research projects were commenced or completed during the year. These projects all lead to a useful advance in knowledge, but as the research was paid for by a sponsor, the results were provided to the sponsor in confidence. Some of this work has, however, subsequently been put into the public domain through publication with the full agreement of the sponsor. Reports on two contract research projects also appear in this Annual Report, again with the agreement of the sponsors.

Overview of NAL work

The Research Committee and the Human Research Ethics Committee continued to give great service to NAL through their expertise and diligence. I am grateful to them for their generously donated time. I am particularly grateful to Dr Keith Joseph who chairs the Ethics Committee and to Professor Field Rickards who chairs the Research Committee and also serves on the Ethics Committee.

HARVEY DILLON

An examination of the apparent poor performance of some hearing protectors

Investigator: Warwick Williams

Introduction: Occasionally it happens that when hearing protectors are tested in accordance with the requirements of combined *Australian/New Zealand Standard AS/NZS 1270:2002 Acoustics – Hearing protectors* and its precursors, that in the view of the manufacturer/distributor/supplier, unexpected results are obtained.

Occasionally test results produce an unexpected high performance figure. However, the most common difficulty is low performance. Frequently this will be for a 'new' or innovative device on which great hopes and expectations were placed for competitive entry into a new market segment. The company who requested (and paid for) the testing wants an explanation from the testing laboratory as to why the device has not performed up to their expectations.

This 'low' performance is not limited to any particular device type or style. It occurs across the board with ear plugs, ear muffs, canal caps and helmet-mounted muffs. The precise reason for this 'underperformance' is currently unclear.

Background: In a recent paper Murphy and Franks (2002) suggested that modeling hearing protector attenuation test results assuming a normal distribution may be flawed and the traditional method of 'processing' the experimental results may be inappropriate.

Murphy and Franks analysed the ANSI (1997) and ISO (2002) test results from several sets of ear plugs

and one set of earmuffs using statistics for normal Gaussian distribution and for bimodal distribution. They found that in many cases bimodal data fit was much more appropriate than a normal distribution. Their conclusion was that "*standards could be based on empirical quantiles which do not assume any particular attenuation distribution*" (pg 2115) rather than specific assumptions and that perhaps a bimodal fit would be most appropriate.

In Australia and New Zealand, acoustic testing of hearing protectors utilises a "subject fit" methodology. The test subject is allowed to fit the hearing protector using only the instructions supplied by the manufacturer and the tester is not allowed to interfere in this fitting process¹. To assist the test subject to produce the maximum attenuation 'fitting noise' is supplied with an instruction from the tester to the subject "*so that you can adjust the protectors for good noise reduction*" (AS/NZS; 2002, P. 26).

The argument has been made (Berg 1996) that without the experimenter (tester) being directly involved in the hearing protector fitting, the results may be sub-maximal. Conversely others argue that the subject-fit method more realistically approaches what can be expected in the workplace when individuals are provided hearing protectors as part of an occupational noise management program. At the present time in Australia and New Zealand the second argument holds sway. The subject-fit procedure is gaining credence internationally with discussions underway for an International Standard (ISO; 2002) utilising a subject-fit protocol very similar to that of AS/NZS 1270.

Theory: Currently the suggestion of Murphy and Franks to use a bimodal model appears to fit the available data. Very simply, this model assumes that the measured test data arises from two separate and distinct causes that are indistinguishable during the course of testing.

¹ Contrary to some popular discussion 'naïve' test subjects are not used. The requirements of AS/NZS 1270 are for test subjects who have "*not had significant previous experience with hearing protectors*" (ref 1, p 21).

The two sets of data can be described by normal Gaussian distributions, N_1 and N_2 , respectively. Thus the overall distribution of test data can be described using a distribution function that is simply a linear combination of the two normal distributions. This combined distribution function N_{1+2} can be written as,

$$N_{1+2} = k N_1 + (1-k) N_2.$$

The constant k is directly related to the number of sample points from each cluster, ranging between 0 and 1. The more the two distributions overlap, i.e. the closer the two means and more similar the standard deviations, the more the combined distribution resembles a single normal distribution.

Analysis of some specific data: When a hearing protector is acoustically tested, attenuation is determined for each of seven test signals. These test signals consist of one-third octave bands of noise, filtered from a pink noise source and centered on octave band center frequencies. The seven separate attenuations, along with their respective standard deviations, are combined as described in AS/NZS1270, Appendix A, to give the SLC_{80} rating and subsequent Class of the hearing protector.

The data on which the statistical analysis is normally carried out is the attenuation and standard deviation at each one-third octave band. It is with this data that the test for bimodality is applied here. Several examples of octave band data have been chosen from tests recently carried out at NAL.

It should be noted that a hearing protector that

performs poorly in one particular test band does not necessarily perform poorly over all test bands. However, poor performance in one test band can markedly effect the overall rating of a device.

Consider the test results from ear plug A. The attenuation of this particular device in the 125 Hz band for each test subject is shown in Table 1.

These data have a mean of 19.5 dB and a standard deviation of 11.1 dB. This distribution of data is illustrated in Figure 1 along with the 'normal' curve with the same mean and standard deviation. However, as can be seen from the superimposed normal Gaussian curve the distribution of the data is far from normal showing two distinct peaks. The bimodal curve is illustrated by the double peaked curve in the figure.

However, if the data is regarded as being distributed bimodally the result is two independent, normal distributions, N_1 and N_2 , with means and standard deviations of 4.2, ± 3.1 dB and 26.1, ± 4.7 dB respectively, and $k = 0.30$. For this ear plug, a mean attenuation of 4.2 dB would be regarded as a 'poor fit' while 26.1 dB would be seen as an 'acceptable' value. For these results it is clearly demonstrated that the results from the 'poor fit' subjects draw down the results of the 'acceptable fit' subjects.

In the example cited there are approximately six test results that could be interpreted as being due to 'poor fit'. It would be tempting to put forward an argument that under the guidance of some declared criteria test subjects with a "low" and "high" attenuation results be respectively

No	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18	19	20
dB	24	5	10	27	28	36	24	18	25	25	19	30	2	29	26	23	2	3	3	31

Table 1: Individual attenuation in dB obtained by 20 test subjects for ear plug A at 125 Hz.

No	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18	19	20
dB	18	24	10	19	35	16	19	7	16	19	30	25	30	18	13	35	17	35	26	20

Table 2: Individual attenuation in dB, 20 test subjects, for ear plug B at 125 Hz.

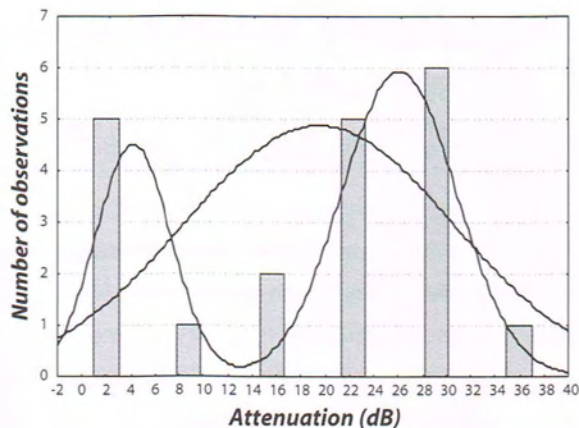


Figure 1: Attenuation results (dB) at 125 Hz for ear plug A, superimposed with possible normal and bimodal distribution.

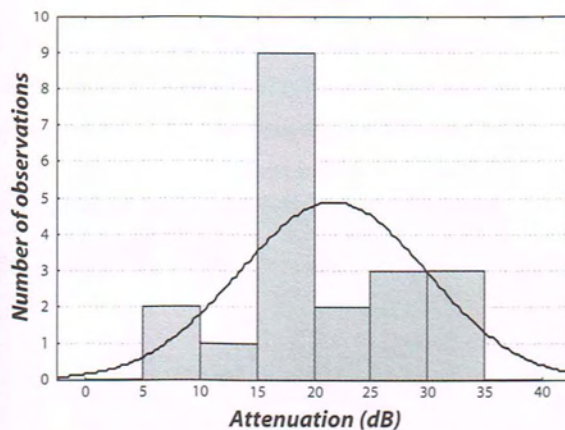


Figure 2: Attenuation results (dB) at 125 Hz for ear plug B, with superimposed normal distribution

divided into two groups and the data processed separately. However, it must be remembered that attenuation is tested at seven one-third octave bands and subjects that record a low attenuation in one particular one-third octave band do not necessarily record low attenuation results in other one-third octave bands.

Compare the above results for ear plug A with those for ear plug B tested at 125 Hz (**Table 2**).

Here the mean attenuation is 21.9 dB with a standard deviation of 8.1 dB. The distribution of the data is illustrated in **Figure 2** with the accompanying expected normal curve. It can be seen that this distribution is much better approximated by a normal Gaussian

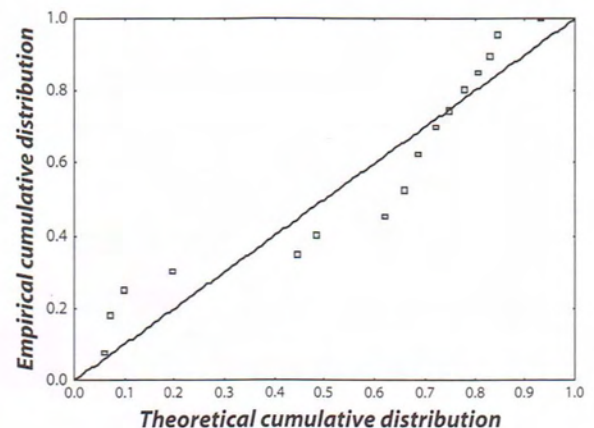


Figure 3: Probability – Probability plot for the attenuation of earplug A at 125 Hz

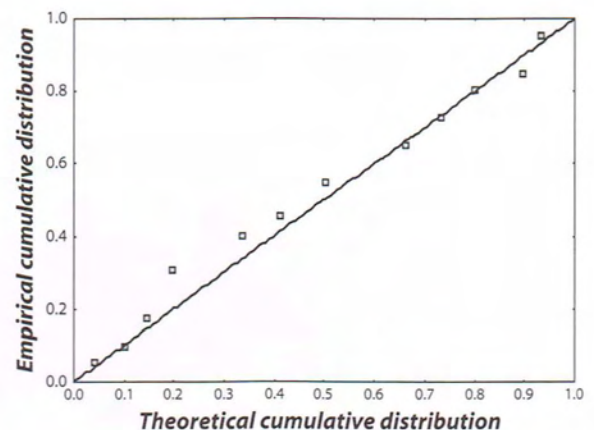


Figure 4: Probability – Probability plot for the attenuation of earplug B at 125 Hz

curve. Hence normal statistics can adequately describe the characteristics of this device.

The tendency to normal distribution is better described through the use of a *probability – probability* plot where, by definition, a normal Gaussian distribution is defined by a straight line. This is shown in **Figures 3 and 4** for earplugs A and B respectively where ear plug B conforms to the straight-line fit of a normal distribution as compared to ear plug A.

Consider now difficulties exhibited with the another hearing protector, a helmet-mounted ear muff, at adjacent test frequencies. The protector has not been removed or in anyway adjusted between these two test frequencies

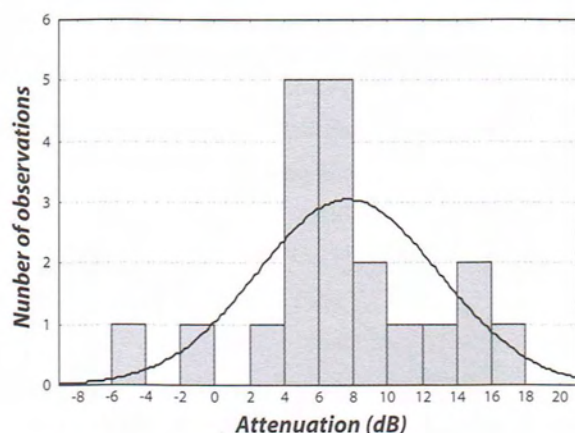


Figure 5: Attenuation results (dB) at 125 Hz for a helmet mounted ear muff, with superimposed normal distribution

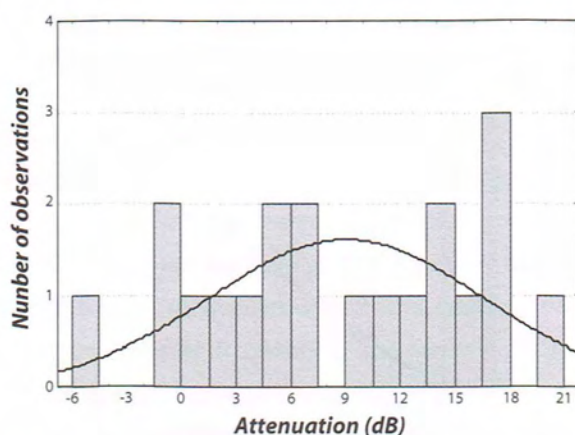


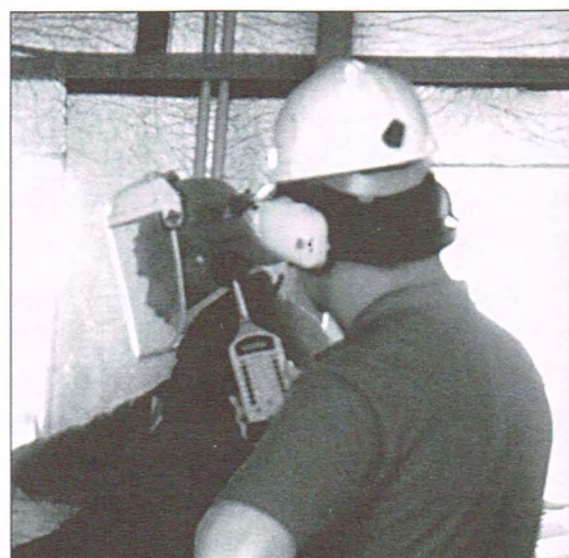
Figure 6: Attenuation results (dB) at 250 Hz for a helmet mounted ear muff, with superimposed normal distribution

and the resulting attenuation is an average of three measured thresholds out of five, the first two being discarded as they are considered to be practice runs at the particular one-third octave band.

Figures 5 and 6 show the distribution of attenuation test data for this protector at the two adjacent test frequencies of 125 Hz and 250 Hz and their respective suggested 'normal' distribution curves. The actual distribution of the data indicates that there is a great deal of difference in the both the spread and the concentration of the results. The degree of kurtosis (flatness) exhibited by both curves is very different with the kurtosis of Figure 5 being 0.27 and Figure 6 -0.90.

Discussion: As can be seen from the above analysis, the assumption that hearing protector test data is normally distributed may lead to conclusions that do not accurately represent the true performance of the device in question. Although analysis is only demonstrated on a limited number of data sets, the general principle of different possible distributions is clearly illustrated.

What statistics to apply may not be known until the mechanism of low attenuation is fully understood. Low attenuation caused by 'poor' fit could be caused by behavioural or educational difficulties such as individuals not following the fitting instructions; unclear fitting instructions; or intentional poor fitting for whatever reason. However, the poor fit could also be caused by physical constraints. These could include poor design, or anatomical features of the head, ear or ear canal yet to be considered.



An example of an incorrectly-worn hearing protector likely resulting in less than optimal attenuation.

References:

- Australian/New Zealand Standard AS/NZS 1270: 2002 Acoustics - Hearing protectors, fifth edition, Standards Australia, Sydney.
- WJ Murphy and JR Franks. Hearing protector attenuation: Models of attenuation distributions, *J Acoust. Soc. Am* 111 (5), Pt 1, May 2002, pp 2109 - 2116.
- ANSI S12.6 - 1997 American National Standard: Methods for measuring the Real-Ear Attenuation of Hearing Protectors. Acoustical Society of America, New York.
- ISO 4869 - Part 7 Subjective method for measurement of sound attenuation - Subject-fit method, third working draft, 2002.
- Berg, G. Attenuation and Protection - can we achieve both? *Proceedings of the Australian Institute of Occupational Health*, AIOH96, Perth 1996.

Increasing the effectiveness of OHS education for noise-exposed individuals

Investigators: W. Williams, S. Purdy, N. Murray, E. LePage, K. Challinor¹ (*Assistance in gathering data was also provided by S. Hynes, P. Foster, L. Smith and M. Sampson of New England Area Health, NSW*)

¹ New England Area Health, Tamworth, NSW

Background: Hearing loss due to noise exposure is an increasing problem throughout the industrial world (WHO: 1997). *"Hearing loss is one of the most pervasive occupational health problems in America today"* (US Dept of Health and Human Services: 1996) and in the US it is estimated that there are between 5 million and 30 million workers exposed to hazardous levels of noise on a daily basis (Berger: 2000). This has been translated into a figure of 10 million who have a hearing loss at least partially attributable to noise (Berger: 2000). In the UK it is estimated that *"at least 1.3 million employees are exposed to noise levels above 85 dB(A)"* during the regular course of their work (RNID: 1999).

The incidence of hearing loss in the Australian community due to noise exposure has been recognised for some time (AAS: 1976; NOHSC: 1989), while more recently the particularly high incidence in rural communities has been examined (Williams, Forby-Atkinson, Purdy and Gartshore: 2002). Estimates of hearing loss across the general Australian community lie between 5.7% (ABS: 1993) and 27% (Wilson, Walsh, Sanchez and Read: 1998). Noise levels in the workplace need to be reduced, and individuals and groups who work in noisy areas need to develop specific knowledge and techniques in order to protect themselves from excessive noise exposure (NOHSC: 2000).

Purdy and Williams (2002) found that simple audiometric testing was beneficial as it resulted in enhanced perceptions of the benefits of reducing workplace noise exposure.

Zohar, Cohen and Azar (1980) showed that ongoing feedback regarding hearing loss had a positive effect on workers' hearing protector use while Lusk et al (1999) recognised that "limited changes in behavior can be expected from a *"one-shot"* intervention". Unfortunately one-shot interventions are the reality for many organisations where access to workers for training purposes is limited.

Research Questions: This study had three main hypotheses that:

- 1) there would be a significant incidence of hearing problems in rural workers compared to an otologically normal population and that these problems would be revealed by audiometric testing and a self-report questionnaire;
- 2) having a hearing test performed by a nurse audiometrist and having the results explained would have a positive effect on perceptions about noise and hearing protector use, and these effects would be sustained over time; and
- 3) subjects who have both conventional pure tone audiometry and a more sophisticated presentation of the results from otoacoustic emissions (OAE) testing of inner ear performance (LePage, Zhou, Murray and Seymour: 2000) would be more likely to believe that noise exposure would further damage their hearing and thus, more likely to increase their use of hearing protectors and avoid noise. Also, as a specific corollary, that, all other things being equal, the presentation of individual OAE results to subjects would prompt subjects with low emission strengths to take greater preventative action providing their hearing thresholds were still 'normal'.

The approach was similar to that used by Zohar, Cohen and Azar (1980), differing only in that the feedback from the hearing test results was presented in the same session as the hearing test was carried out. There were no other formal or informal presentations to the participants other than the one

audiometric test session with accompanying feedback as described below.

Procedure: The project was carried out with assistance of nurse audiometrists who work in New England Area Health in northern NSW, part of the NSW Department of Health. Subjects were recruited when they presented for a routine audiometric test as part of their workplace occupational health program. Upon their agreement to be part of the program they were alternately assigned to one of two groups. Both groups completed a standard questionnaire before being divided for audiometric assessment. Group 1 received pure tone audiometry (PTA) only while Group 2 received PTA and otoacoustic emission (OAE) testing.

Subsequent to the initial questionnaire and audiometric assessment, individuals from both groups were followed up with repeat questionnaires at six week and twelve month intervals. The questionnaires were ones developed and published (Purdy and Williams; 2002) from a previous NAL study (*Effective training methods for workplace noise reduction*). This 20-item questionnaire has 5 sub-scales with items assessing:-

- (1) Benefits - the perceived benefits of reducing noise and noise exposure, for example through the use of hearing protectors;
- (2) Barriers - the perceived barriers to reducing noise exposure;

- (3) Self-efficacy - the perceived ability to reduce noise exposure and/or protect hearing (Bandura: 1986);
- (4) Attitude - attitudes to workplace noise and noise exposure; and
- (5) Susceptibility - an individual's perceived susceptibility to hearing loss, interpreted as whether they think noise exposure can/will damage their hearing.

The questionnaire also included information on demographics, hearing problems, conversational difficulties in noise, family reports of hearing problems and incidents of tinnitus.

Results: A total of 136 individuals were involved, 67 in Group 1 (PTA only) and 69 in Group 2 (PTA & OAE). The age range was 20 to 65 years with a mean age of 39.6 years and standard deviation 11.4 years. The subjects routinely worked in noisy environments and had been in their present position for 13.8 years ($\sigma=10.7$ years).

There was no significant difference in the PTA hearing levels between the two groups with a mean hearing loss of 24 dB HL ($\sigma=13.5$) at 3k, 4k and 6kHz. This is illustrated in **Figure 1** below.

For an otologically normal population at this age and mixture the expected average threshold would be 6.4 dB (ISO 7029). This result confirmed hypothesis 1.

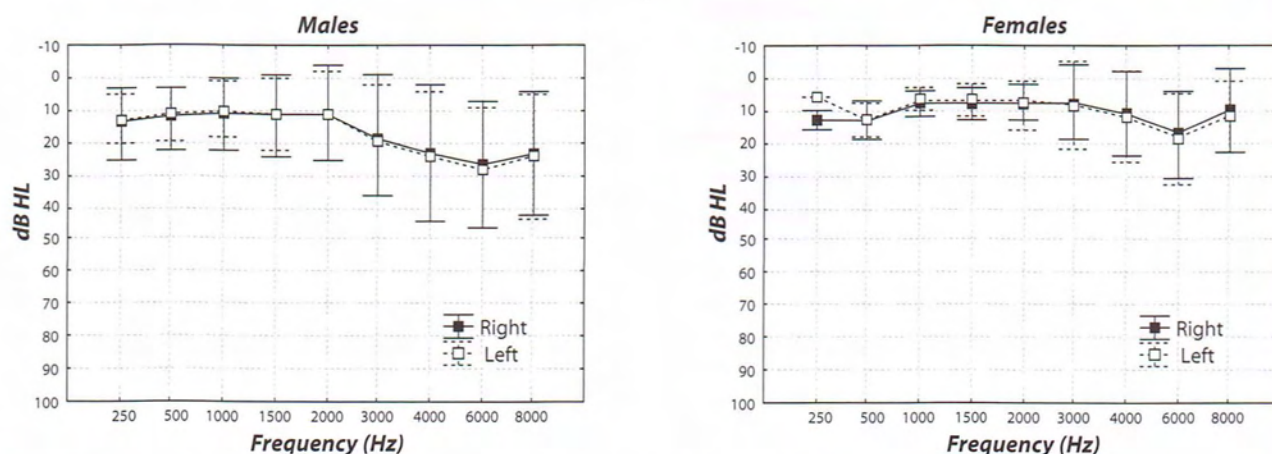


Figure 1: Right and left ear average pure tone audiograms for male (N=113) and female (N=23) subjects. Error bars show standard deviations.

Group comparison: Without the females, groups 1 and 2 were equivalent at the first visit, for all but one of the measures, self-rated noise exposure. Self-rated noise exposure was higher ($t = 2.2$, $df = 110$, $p = 0.033$) for Group 1 compared to Group 2. Noise exposure was therefore included as a continuous predictor (covariate) for a three-way analysis of variance using 'Group' as a between-group factor and sample time and sub-scale as repeated-measures factors. This analysis showed no significant differences between the two groups for questionnaire subscale scores ($F = 0.6$, $df = (1, 86)$, $p = 0.436$). There was, however, a significant effect of test occasion on questionnaire scores ($F = 8.1$, $df = (2, 172)$, $p < 0.001$). This is illustrated in **Figure 2**, which shows that subscale scores improved at the 6-week assessment except for the Attitudes subscale that showed no change across the three assessment occasions.

There were no group effects or interactions with group and hence the groups were combined prior to doing posthoc analyses to examine the repeat effect. Scheffe posthoc analyses for the individual sub-scales showed significant improvements between the Baseline and 6-week ratings for the Benefits ($p = 0.002$), Barriers ($p < 0.001$), Self-efficacy ($p < 0.001$) and Perceived Susceptibility ($p = 0.048$) subscales. For Benefits, Barriers and Perceived Susceptibility this improvement over the baseline was maintained at 12 months ($p \geq 0.862$). For Self-efficacy, however, ratings dropped significantly between 6 weeks and 12 months ($p = 0.005$) and consequently the final 12-month

result did not differ from the initial result ($p = 0.379$).

The questionnaire sub-scale test results are presented in **Figure 2** and can be summarised by saying Benefits, Barriers and Perceived Susceptibility ratings showed an improvement after hearing testing which was sustained, Self-efficacy showed an initial improvement but this was not sustained over time, and there was no change in Attitudes.

Outcome measures that might have changed as a result of the hearing testing include self-rated hearing loss, if subjects became more aware of hearing difficulties after the test. A Friedman non-parametric ANOVA of the responses to the questions about hearing problems, conversational difficulties in noise, family reports of hearing problems or tinnitus showed no significant changes across test occasions for either group ($p \geq 0.071$).

One anticipated outcome was that subjects would be predisposed to use hearing protectors more often after having hearing testing and becoming aware of hearing problems and risks to hearing. Although the figures showed that there was a weak trend towards increased use of hearing protectors, a repeated measures ANOVA of self-reported hearing protector use showed no significant change over the three test occasions ($F = 1.5$, $df = (2, 212)$, $p = 0.215$), and also shows no effect of group or interaction of group with test occasion.

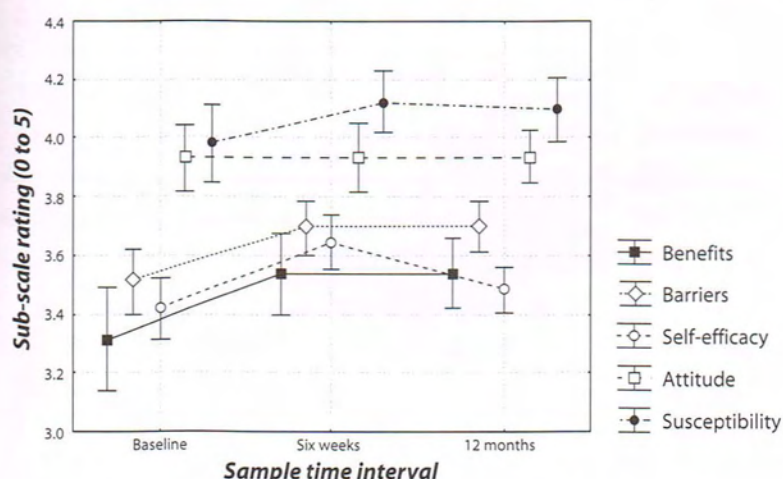


Figure 2: Mean sub-scale ratings for the male subjects ($N=113$, Groups 1 and 2 combined). The Baseline was obtained immediately prior to hearing testing. Results at six weeks and 12 months were obtained via telephone interview. The error bars show standard deviations. (High rating indicates better performance for all sub-scales.)

These results supported hypothesis (2) but not hypothesis (3).

Side note: As a result of the initial application of the questionnaire some interesting results arose before splitting the subjects into the two experimental groups. Hearing test results were compared with the individual's perception of their own hearing and their perceptions of noise annoyance, hearing damage risk and preventative action. Self-reported and family-reported hearing loss and conversational difficulties in noise correlated well with measured audiograms. The overall reported incidence of tinnitus quite high.

All subjects came from noisy work areas, however, the perceptions of workplace noise tended to be more positive (i.e. greater noise awareness) if people felt they had hearing problems. Those who reported no hearing loss were less concerned about noise exposure. Notably there was no difference in hearing protector use between those that did and those that did not feel they had hearing problems. Overall hearing protector use was limited to an average 29% of the time.

Better educated subjects amongst those who reported hearing problems were more likely to consider that noise reduction was beneficial. However, they also rated Barriers and Self-efficacy in the same way as subjects with less education and no self-reported hearing loss.

This implied that those with a hearing loss thought that action should be taken in order to reduce further hearing loss, but these feelings did not translate into any positive action. Sadly it was only those who worked in the management sub-group who felt that they could have some positive effects on outcomes. The disparity between managers and other work groups about noise is of some concern and indicates that there is a need for specific training to ensure that workers have the skills (and confidence) to take more positive action to reduce noise exposure in the workplace.

References

- Australian Academy of Science (1976). *Report of a Committee on the Problem of Noise. Report Number 20*, Australian Academy of Science, Canberra, March 1976.
- Australian Bureau of Statistics (1993). *Disability, Ageing & Community Services*, Hearing Impairment.
- Bandura, A (1986). *Social Foundations of Thought and Action: A Social Cognitive Theory*, Prentice Hall, New Jersey.
- Berger, EH, Royster, LH, Royster, JD, Driscoll, DP and Lippert, J (2000). *The Noise Manual*, fifth edition, American Industrial Hygiene Association, Fairfax, Va.
- ISO7029 Acoustics – Statistical distribution of hearing threshold as a function of age, International Organisation for Standardization, Geneva, 2000.
- LePage, EL, Zhou, D, Murray, NM and Seymour, J (2000). *NAL-QC Software Analysis*. Paper presented at the Australian Society of Audiology Conference, Adelaide, 2000.
- Lusk, SL, Honh, OS, Ronis, DL, Eakin, BL, Kerr, MJ and Early, J (1999). Effectiveness of an Intervention to Increase Construction Workers' Use of Hearing Protection. *Human Factors*, Vol 41, No 3, September 1999: 487 – 494.
- National Occupational Health and Safety Commission (1999). *National Strategy for the Prevention of Occupational Noise-Induced Hearing Loss*, Australian Government Publishing Service, Canberra.
- National Occupational Health and Safety Commission (2000). *Occupational Noise, National Standard for Occupational Noise [NOHSC: 1007(2000)] and National Code of Practice for Noise Management and Protection of Hearing at Work [NOHSC: 2009(2000)]*, 2nd Edition, Canberra, Australia, July 2000.
- Purdy, S and Williams, W (2002). Development of the Noise at Work Questionnaire to assess perceptions of noise in the workplace. *J Occup Health Safety – Aust NZ* 2002, 18(1): 77 – 83.
- Royal National Institute for Deaf People (1999). *Indecent Exposure – A joint report on noise at work by RNID and TUC*, TUC/RNID noise at work campaign, London.
- US Department of Health and Human Services (1996). *Preventing Occupational hearing Loss*, NIOSH, Cincinnati, Ohio.
- Williams, W, Forby-Atkinson, L, Purdy, S and Gartshore, G (2002). Hearing loss and the farming community. *J Occup Health safety – Aust NZ*, 2002, 18(2): 181 – 186.
- World Health Organization (1997). *Prevention of Noise-Induced Hearing Loss*, Report of an Informal Consultation held at the World Health Organization, Geneva, October 1997.
- Wilson, D, Walsh, PG, Sanchez, L and Read, P (1998). *Hearing impairment in an Australian population*, Department of Human Services Centre for Population Studies in Epidemiology, Adelaide.
- Zohar, D, Cohen, A and Azar, N (1980). Promoting Increased Use of Ear Protectors in Noise Through Information Feedback. *Human Factors* 22 (1), February 1980: 69 – 79.

Evaluation of the effects of high noise exposure: coal miners

Investigators: Eric LePage, Narelle Murray, Adrian O'Malley¹

¹ Australian Coal Industry Research Laboratories

Background: A pilot study was commissioned by the Australian Coal Industry Research Laboratories Limited (ACIRL) to conduct a research hearing loss prevention project involving miners operating at a NSW deep coal mine. In many studies carried out at NAL, the status of any ear has been assessed using pure-tone thresholds, tympanometry and evoked otoacoustic emissions. The clinical status of any ear measured in this way has mostly taken to be the permanent condition of the ear. However the possibility exists that any record has been influenced to an unknown extent by the effects of recent noise trauma causing temporary shifts.

This study concerns our first attempt to assess the influence of recent noise exposure upon click-evoked otoacoustic emissions (CEOAE) (LePage, Murray and Macrae, 1992). It is well known that loud noise exposure results in a Temporary Threshold Shift (TTS) by which is meant a decrease in hearing sensitivity which reverts to normal hearing exponentially following cessation of the noise producing it. Whereas we might have expected by analogy, that emission strengths decline (as occurs with ageing and tends to occur with hearing levels), the resulting Temporary Emission Shifts (TES) have appeared complex, more so than in a recent report by Marshall and Heller (1998). This may be due to 1) emissions giving a much more dynamic picture of cochlear activity; and 2) the miners each have different occupations within each team and/or are wearing their ear-protection devices differently. The within-subject data variability from one shift day to the next is larger than that obtained using sound-booth test-retest conditions (Murray et al, 1997). Since

the data were obtained, the insights and techniques developed make it possible to assess the effects of extreme noise exposure contained in these data.

Research Questions: Through the use of CEOAE:

- 1) To compare long-term cochlear damage in a highly noise-exposed group with a normative male Australian population of the same age range; and
- 2) To quantify in individuals measures of cochlear trauma accumulated in a high noise environment.

Research Procedures: Twenty-four deep coal miners working in a variety of jobs were voluntarily tested with CEOAEs before and after shift (either morning or afternoon) every day for over a ten-day period beginning on a Friday (day 0) with retesting occurring on days 3, 4, 7 and 10 thereafter. Workers did not necessarily have any days of quiet before the testing began. Further follow-up CEOAE testing was carried out on one day before and after shifts four months later. Questionnaires regarding aural health, wearing of hearing protection, type of job, recreational noise exposure, hearing difficulty, changes to hearing ability (from the spouse's viewpoint) and smoking were completed by all participants. ACIRL staff provided the estimated daily noise dose for each miner. Mine management provided pure tone audiometric results for both initial testing on employment and the most recent audiogram.

Findings:

Comparison of miner and normative Australian data

Figures 1A and B show the audiometric and otoacoustic emission comparisons between the group of coal miners and our normative male Australian data (LePage and Murray 1993) which excluded ears with otological pathology but included all noise-exposed people excepting the miners in this study. The normative CEOAE and PTT threshold data were for the pooled data from 439 subjects while the coal miners numbered 24, (two teams of 12 workers). All data are partitioned by age into four decade groups.

These are compared with the means of the normative population. In Panel A, the mean, across subjects and ears of the poorer of hearing levels at either 4 or 6 kHz are plotted because this measure is most sensitive to noise-induced hearing loss (ISO-1999: 1990). A comparison of the groups reveals that, for the two central age ranges there is no significant difference between the Australian normative male population and the coal miners. Both groups approach a mild hearing loss (at 25 dB HL) when they are in the 40-49 year age range. Panel B shows CES values for Australian normative males compared with those for coal miners in the same age ranges. The values of the CES (dB SPL) for the two groups are significantly different ($p < 0.01$), even when differences in age and hearing level are controlled for via their inclusion as co-variables.

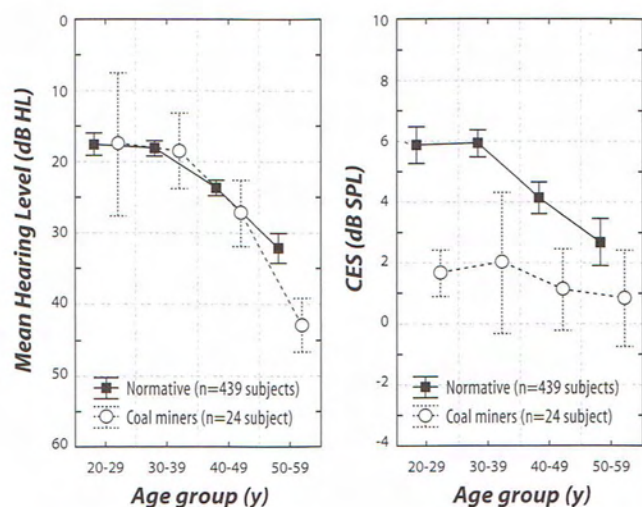


Figure 1: The left panel shows comparison of Australian normative males (filled squares and solid lines) and coal miners (open circles and dashed lines) for pure tone audiometric thresholds taken as the mean of the poorer threshold at either 4 or 6 kHz. Whiskers denote mean \pm standard error of the mean. There is only one subject in the 20-29 year age range amongst the miners ($n=2$ ears). The right panel shows CES values for Australian normative males (filled squares and solid lines) from the subset of NAL subjects used to generate the left panel. These CES values are compared with those for coal miners (open circles and dashed lines) in the same age ranges ($p < 0.01$). Whiskers represent standard errors. Values for coal miners are the mean values for both ears for first and last test.

Effect of the extent of heavy noise exposure on mean emission and hearing levels

Figure 2 shows the dependence of pure tone thresholds and mean emission strength (CES dB SPL) for left and right ears over all measurements upon level of noise exposure. The two groups are based upon mean noise dose of the individual worker, obtained from the ACIRL estimates of the noise level in terms of the percentage of maximum permissible daily dose (i.e. energy dose as a fraction of 90 dBA over an 8 hour day). This estimation relates to occupation type, not the use of hearing protection devices (HPDs). The values are reclassified into "Low" and "High" rated noise exposure according to whether the percentage lies below or above 60%. The left panel shows the means of the pure tone threshold obtained at either 4 or 6 kHz whichever is the poorer threshold. There is no significant difference between the threshold results for the two groups. The right panel shows the

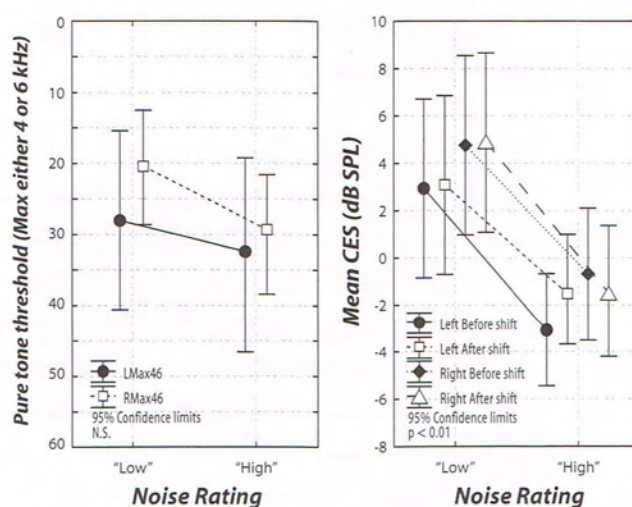


Figure 2: Left and right panels show dependence of pure tone thresholds and click-evoked otoacoustic emission strength (CES dB SPL) upon estimates of noise exposure. The emission dependencies are significant ($p < 0.01$) while the difference in pure tone thresholds is not significant.

otoacoustic emission measure CES plotted for the same two groups for the data on all six days. The four sets of data are for CES values before and after daily work shifts for left and right ears. The differences in CES for the two groups are significant ($p < 0.01$) showing that those with high exposure have lower emission strengths, and by inference, more accumulated cochlear damage. Since 6 days of data have been used to carry out the ANOVA on the otoacoustic emissions, to make the comparison of the two methods more comparable, the ANOVA was performed on the data for just the first day ($p < 0.05$) and on just the before-shift data $p < 0.05$.

Figure 2 suggests that CES values decline with increases in the estimation of the noisiness of worker occupations. The mean of ears in the high noise category are below our previously established critically-low level of 0 dB SPL, considered to be approaching a mild hearing loss (LePage & Murray 1993). It is consistent that the pure tone threshold values in the left panel are around a 25 dB hearing level.

Significance: Traditional thinking about noise-induced hearing loss is strongly couched in terms of the "Equal Energy Principle" and what energy absorbed by the ear constitutes a reasonable "Daily Dose" of noise. The principle was developed on the temporary shifts that result from quite high levels of noise exposure, such as coal miners encounter on a daily basis. These data show that the pure tone thresholds in the miners as a group are not significantly different from the normative population data we have collected at NAL. On the other hand, the mean values of emission strength for the group of miners are significantly lower than the normative values suggesting that otoacoustic emissions are more sensitive to noise-trauma. In addition, it was seen that the amount of depression of hearing levels with the degree of noise exposure is not significantly different. By contrast, otoacoustic emissions appear to be more sensitive ($p < 0.01$) to the different levels of trauma encountered by the low and high noise groups. In many instances, temporary

click-evoked emission shifts may be positive- as well as negative-going as seen in the higher noise group and the characterisation and cause of this effect is the subject of ongoing investigation.

References

- ISO-1999 (1990). Acoustics: Determination of occupational noise exposure and estimation of noise-induced hearing impairment. Geneva.
- LePage, E. L. and Murray, N. M. Click-evoked otoacoustic emissions - comparing emission strengths with pure tone audiometric thresholds. *Aust.J.Audiol.* 15, 9-22, 1993.
- LePage, E. and Murray, N. Measures of susceptibility to hearing loss: otoacoustic emissions and noise exposure. *NAL Research & Development Annual Report 1992/93*, 19-20. 1993.
- LePage, E.L. & Murray, N.M. (1998). Latent cochlear damage in personal stereo users: a study based on click-evoked otoacoustic emissions. *Med. J. Aust.*, 169, 588-592.
- LePage, E. L., Murray, N. M., and Macrae, J. H. *Otoacoustic emission assessment of ear damage in coal mine workers: Pilot study May - October 1992*. National Acoustic Laboratories Commissioned Report No. 75, March, 1993. Sydney, Australia.
- Marshall, L. and Heller, L. M. Transient-evoked otoacoustic emissions as a measure of noise-induced threshold shift. *J Speech Lang Hear Res.* 1998 Dec; 41(6), 1319-34.
- Murray, N. and LePage, E. Tracking the growth of cochlear damage in a cohort study. *NAL Research & Development Annual Report 1994/95*, 28-30. 1995.
- Murray, N.M. & LePage, E.L. (1993). Age dependence of otoacoustic emissions and apparent rates of ageing of the inner ear in an Australian population. *Aust.J.Audiol.*, 15(2), 59-70.
- Murray, N. M.; LePage, E. L., and Tran, K. Repeatability of click-evoked otoacoustic emissions. *Aust.J.Audiol.* 1997; 19(2)109-118.
- NAL Annual report 2001/2002*. Causes and prevention of hearing loss: global trends in industrial and leisure noise, interactions, definitions and strategies - a one day colloquium. 18-21. National Acoustic Laboratories, Sydney, Australia.

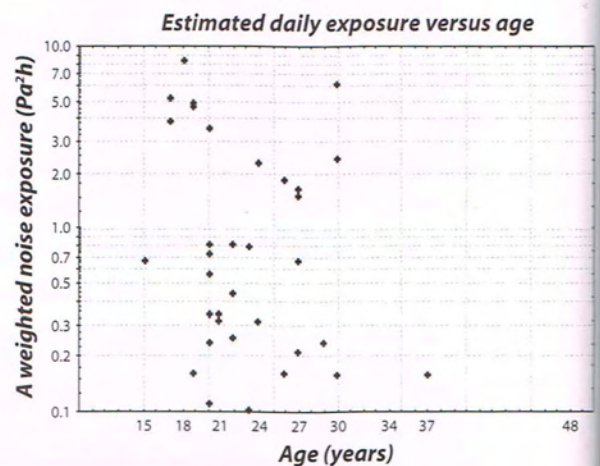
Personal Stereo Noise Exposure

Investigator: W Williams

Brief report: This research aims to survey the levels to which personal stereos are used in every day circumstances by individuals going about their daily activities. Potential subjects for this project are those who are observed using a personal stereo. Personal stereos could be any of a number of devices for which earphones are worn while doing other activities such as commuting or exercising. There is a lot of anecdotal evidence concerning the noise levels experienced by personal stereo users but to date, while several laboratory studies have been carried out, there have been no published studies of measurements carried out in the "real world". Similar work was carried out in France by Christian Meyer-Bisch in 2002 but the results are yet to be published. The NAL protocol is similar to the French study.

To date subjects have been approached outside Flinder's Street Station in Melbourne and the Town Hall in Sydney's CBD. Many potential subjects are reluctant to participate as they seem to fear that the experimenter is either begging or 'carrying out a survey' to sell something!! However, a respectable sample size has so far been collected and an initial examination of the exposure levels shows that around 25% of users receive an exposure that places them 'at risk' of possible noise injury. This is not taking into account any other noise exposure they may receive at work or among other leisure activities.

At the suggestion of the Research Committee, the project has been extended to gather more data from other locations, possibly away from areas of noisy public transport. This will open the opportunity to gain measurements in public areas where background noise is not so prevalent.



The figure shows the distribution of noise exposure for personal stereo uses versus age. The *A-weighted noise exposure* is the estimated daily exposure from personal stereo use only. A continuous, equivalent A-weighted sound exposure level, $L_{Aeq, 8h}$ of 85 dB is equal to one Pascal squared hour, $1 \text{ Pa}^2\text{h}$.

Percentage Loss of Hearing Tables

Narelle Murray, Dan Zhou

For many years (since 1974) the NAL Procedure for calculating Percentage Loss of Hearing has been used by the Commonwealth and most States of Australia for compensation of hearing loss purposes. An Improved Procedure was introduced in 1988 and since 2002 all States and the Commonwealth now use this system. To modernize the procedure, a Windows™ based program was developed during 2002-2003. This program allows the user to simply enter the hearing levels of the client which are then automatically converted to both monaural and binaural Percentage Loss of Hearing. If required, extensions to 6000 and 8000 Hz may be added and an optional correction for presbycusis is also included. The results may be printed out in a very neat format. This is a simple, easy-to-use program which means that the user does not have to access the written Tables for every calculation. The program and the *Improved Procedure for Determining Percentage Loss of Hearing* (NAL Report No. 118) may be ordered via the NAL website: www.nal.gov.au.



Factors affecting speech intelligibility of hearing impaired people

Investigators: Teresa YC Ching, Harvey Dillon, Frances Lockhart, Emma van Wanrooy, Christopher Brew, Lydia Lai¹, John Newall², Jonas Brännström³

¹ University of Texas

² Macquarie University

³ Lund University

This study is supported by CRC Hear.

Background: Speech cannot be understood if it cannot be heard. Therefore, a major goal for rehabilitation of hearing impaired people is to amplify all speech frequencies to be audible at a comfortable level. In doing so, speech intelligibility is maximised for people with mild or moderate hearing losses. However, the same cannot be said for people with severe or profound hearing losses. Research has shown that providing high sensation levels at frequencies where the hearing loss was severe or profound did not always improve speech intelligibility, and might even be detrimental (Murray & Byrne, 1986; Ching et al, 1998; Hogan and Turner, 1998). This reduced usefulness of audibility with increased hearing loss is commonly referred to as "hearing loss desensitization" (Pavlovic et al, 1986; Studebaker et al, 1997), and is known to produce a more adverse effect at the high than at the lower frequencies.

The Articulation Index (ANSI, 1969), now known as the Speech Intelligibility Index (ANSI, R1997), provides a theoretical framework for relating audibility to speech intelligibility. Using the Speech Intelligibility Index (SII), the performance of people with normal hearing or with milder hearing losses could be predicted adequately from the amount of audible signal above the listeners' hearing thresholds (Kamm et al, 1985; Dirks et al, 1986; Dubno et al, 1989), but the performance of people with more severe