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Effects of Noise on Hearing V:th International Symposium

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**THE EFFECTS OF ACOUSTIC TRAUMA, OTHER COCHLEAR INJURY AND DEATH
ON BASILAR-MEMBRANE RESPONSES TO SOUND**

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Rhode's (1971, 1973) discovery that basilar-membrane responses to characteristic-frequency (CF) tones grow nonlinearly with stimulus intensity was accompanied by some evidence that the nonlinear behavior was linked to the sensitivity and sharpness of frequency tuning of the mechanical responses. Recent extensions of Rhode's pioneering work show that, indeed, normal basilar-membrane responses to sound are characterized by a triad of features: high sensitivity, sharp frequency tuning and nonlinearity. These features manifest themselves only for near-CF stimulus frequencies and appear to be inextricably interlinked: none has been observed in isolation and they probably constitute three aspects of a single mechanism. The present paper surveys the effects of acoustic overstimulation, furosemide, quinine, death and surgically-induced cochlear trauma upon basilar-membrane responses to single tones, clicks and pairs of tones. The mechanical effects of death or cochlear trauma - abolition or reduction of nonlinearities, decreased sensitivity and broadened frequency tuning - are consistent with the idea that the alterations of basilar-membrane vibration causally determine similar CF-specific effects in auditory-nerve responses. The effects of cochlear insults also imply that the organ of Corti and the basilar membrane sustain a mechanical feedback relationship. Thus, the second filter once posited to account for auditory-nerve frequency selectivity has been rendered unnecessary, being replaced by a cochlear amplifier that is tightly coupled to basilar-membrane vibrations.

NOISE INDUCED EXPRESSION OF HEAT SHOCK PROTEINS IN THE COCHLEA

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The expression of heat shock proteins was originally shown in *Drosophila* in response to heat, but has now been shown in almost all cells, in all species, in response to a wide variety of stresses. Several different families of heat shock proteins have been found, differentiated by their molecular weight, with HSP 20s, 60s, 70s and 90s families the most widely studied. Their function is believed to be protective, through different mechanisms ranging from protein folding to interaction in various ways with cytoskeleton. We have examined if they may have a general protective role in the auditory system as well as a specific role in protection from noise induced hearing loss. We first studied HSP 72 in the rat cochlea and found that it was induced in response to hypoxia and noise overstimulation (110 dB SPL, broad band, for 90 mins), in stria vascularis and in outer hair cells (Myers et al 1992; Lim et al 1993). The constitutive form in the HSP 70 family is also present in cochlea and its level of expression may also be influenced by noise. We have recently found that noise overstimulation induces the expression of other families of heat shock proteins. There is a noise induced expression of HSP 27 in outer hair cells. HSP 90 was expressed in inner and outer hair cells without stress (constitutive) and noise overstimulation appeared to influence the level of expression.

It is interesting to consider what role heat shock proteins might play in protection from noise induced hearing loss. Since expression of induced forms can take several hours, the induced forms are not likely to be involved in initial events and constitutive expression would be most useful for immediate protection. Induced expression might be involved in recovery processes. Heat shock proteins could therefore have a role both in the production of and recovery from temporary threshold shift. One mechanism by which this could be achieved is through influencing the cytoskeleton. HSP 27, in particular, is believed to have interactions with actin. Heat shock proteins may also be involved in "toughening" of the auditory system. Pre-exposure stresses which elevate heat shock protein levels have been shown to decrease the effects of a second stress. For example hyperthermia protects against subsequent light damage in retina (Barbe et al, 1988) or infarct after ischemia (Hutter et al, 1994). In addition to further examining expression of heat shock proteins in the cochlea we will also examine their functional role through training, cross-training and 'knock-out' experiments.

CHANGES IN GENE EXPRESSION IN THE COCHLEA FOLLOWING NOISE-INDUCED HEARING LOSS.

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The expression of several molecules either known or suspected to be involved in the transduction process was studied in the cochlea of noise-exposed rats using in situ mRNA hybridization. The animals were exposed to a two-octave band of noise (1.4-5.6 kHz) at 110 dB SPL for one hour, an exposure which results in substantial TTS but little or no PTS. They were sacrificed after a recovery period of 0, 1, 2, 4, 8, 16 or 14 hours. The expression of mRNA was evaluated in the inner ear using in situ hybridization with radiolabeled riboprobes. Probes complementary to mRNA encoding non-NMDA glutamate receptors, acidic fibroblast growth factor, c-fos, c-jun, β -actin, fibronectin, and five subunit isoforms of Na,K-ATPase. Most genes showed no change in expression. However, immediately after exposure, mRNA encoding the $\beta 2$ isoform of the β subunit of Na,K-ATPase in the stria vascularis showed a marked decrease in expression. Expression increased gradually over the next few hours. In contrast, no change in the expression of mRNA encoding the $\alpha 1$ isoform of the α subunit was seen in the stria, and expression of the various Na,K-ATPase isoforms in other tissues was not affected. Since the β subunit is required for membrane insertion and function of the enzyme complex, the results suggest that traumatic noise exposure results in a temporary decrease in Na,K-ATPase activity in the stria vascularis.

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MELANIN IN THE NORMAL AND NOISE-DAMAGED COCHLEA

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Melanin is an interesting molecule which probably has a dual function. At moderate energy stimulation levels such as ultraviolet radiation or sound energy melanin could have a protective effect basically in diminishing the effects of surplus energy such as heat or free radicals produced by the oxygen metabolism. This is illustrated by the protective effect of melanin in the skin of people with dark skin in comparison with people with fair skin (less melanin). In the inner ear melanin is abundant in the intermediate cells in the stria vascularis. These cochlear melanocytes might have a similar function here in reducing the damaging effect of moderate noise levels. However, both in the skin and in the inner ear at a certain level, or under certain circumstances, the melanin may have a toxic effect on the surrounding tissues emitting harmful radicals. This can be illustrated by use of chloroquine using an experimental TTS model. The dual function of melanin makes it particularly difficult to assess experimentally for instance with noise.

GENETIC SUSCEPTIBILITY TO NOISE-INDUCED HEARING LOSS IN MICE

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The C57BL/6 inbred mice have a gene that results in age-related hearing loss (AHL) and in an increased susceptibility to noise-induced hearing loss (NHL). Shone et al. (Hearing Res., 1991) showed that six-month-old C57BL/6 mice exhibited high-frequency hearing loss, loss of hair-cells and greater susceptibility to NHL, in contrast to the normal hearing CBA/Ca mice. Borg and colleagues have also shown that the C57BL/6 mice are differentially sensitive to traumatic noise exposures compared to the CBA mice. Erway and colleagues have shown that 3-4-month-old C57BL/6J mice, before they exhibit AHL, are more susceptible to 110 dB SPL noise (broadband 5-32 kHz) exposure for 1 hr., than are the CBA/CaJ mice. Two F1 hybrid strains of mice indicate that the difference in susceptibility to NHL may be due to the AHL gene (*ahl*). The CBAxC57 F1 hybrid mice (putatively *+/ahl*) do not differ from the CBA/CaJ mice (putatively *+/+*) in regard to TTS and recovery from exposure to 110 dB for 1 hr. By contrast, the C57xDBA F1 hybrid mice (putatively *ahl/ahl*) are as susceptible to NHL as are the C57BL/6J inbred mice (also *ahl/ahl*).

The postulate that the *ahl* gene contributes to the susceptibility to NHL has been tested genetically in mice. The CBAxC57 F1 hybrid mice (*+/ahl*) were backcrossed to C57BL/6J mice (*ahl/ahl*), yielding progeny of two different genotypes and phenotypes: 1/2 expected to be *+/ahl* with little/no PTS; 1/2 expected to be *ahl/ahl* with PTS, especially at 16 kHz. The backcross progeny were exposed to 110 dB noise for 8 hrs. to maximize PTS in the *ahl/ahl* mice. Half of the progeny (*+/ahl*) exhibited TTS and rapid recovery within 3 days. The other half of the progeny (*ahl/ahl*) exhibited large TTS and slow recovery rates with PTS; however, about 25% of these mice recovered to normal (no PTS) by two weeks post-exposure. Re-exposure of these putatively *ahl/ahl* mice to 110 dB noise for 4 hrs. resulted in PTS to two weeks, whereas re-exposure of a comparable group of the putatively *+/ahl* mice exhibited no PTS.

The backcross mice were chromosomally typed independently of the noise-exposure and hearing tests. Genetic mapping results indicate close linkage between two markers on Chromosome 10 and a genetic factor which affects the level of susceptibility of these mice to noise-induced hearing loss. The one and same *ahl* gene appears to make mice more susceptible to AHL and to NHL. Reported variabilities for NHL among outbred strains of guinea pigs and chinchillas may be related to genetic variability, as shown among these inbred, hybrid and backcross strains of mice.

The effect of the *ahl* gene on increased susceptibility to NHL or AHL may be related to impaired homeostatic and/or repair mechanisms within the organ of Corti. The decreased rate of recovery of the *ahl/ahl* mice from noise exposure, with potentially full recovery, may implicate repair mechanisms.

**SENSORY CELL REGENERATION AND FUNCTIONAL RECOVERY FOLLOWING
ACOUSTIC TRAUMA AND AMINOGLYCOSIDE OTOTOXICITY: A REVIEW.**

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In the inner ears of birds, hair cells that have been lost as consequence of exposure to noise trauma or ototoxic aminoglycosides are replaced by new hair cells. These arise through trauma-induced proliferative activity in the undamaged supporting cells in the area of the lesion. Proliferation begins about 18 hours after the onset of a traumatising noise regardless of the total time of exposure, and will continue even in the presence of damaging sound. New hair cells can be recognised 90-100 hours after the start of noise exposure. Likewise, with aminoglycoside induced-damage, new hair cells may appear during a chronic dosing regime and whilst hair cell loss is still progressing.

The new hair cells become innervated and studies using electrophysiological, biomechanical and behavioural techniques have shown that functional recovery occurs. After noise exposure, functional recovery appears to progress faster than the rate of hair cell regeneration. This has been ascribed to: a) recovery of function of the tall hair cells, which possess afferent innervation and which survive noise exposure; b) regrowth of the tectorial membrane which is disrupted over the lesion area but quite rapidly repairs; and /or c) recovery of the tegmentum vasculosum to restore the ionic profile and potential of endolymph. After aminoglycoside-induced damage, which affects both tall and short hair cells, functional recovery takes much longer, ca. 20 weeks, and may correspond to the appearance of the new hair cells and subsequent re-establishment of appropriate innervation.

Recently, evidence has been presented suggesting a regenerative capacity exists in the mammalian inner ear also. After aminoglycoside-induced damage, spontaneous, but incomplete, replacement of lost hair cells occurs in the mature vestibular system of guinea pigs, and stimulated regeneration of hair cells in the immature organ of Corti of rats has been reported. So far, however, there is no evidence for regeneration in the mature mammalian cochlea. Studies of organotypic cultures of avian and mammalian inner ear tissues are being used at a number of centres in an effort to identify factors that may influence hair cell regeneration. Some possible molecular characteristics associated with the process have been suggested.

PHYSIOLOGICAL CORRELATES OF SPONTANEOUS OTOACOUSTIC EMISSIONS INDUCED BY ACOUSTIC TRAUMA.

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Spontaneous otoacoustic emissions (SOAE) are rarely seen in chinchillas unless the animals have been exposed to intense sounds. Here we report on the acoustic and physiological properties of a SOAE that was observed in a chinchilla following a 105 dB SPL exposure at 2.8 kHz for 2 hours. The frequency of the SOAE varied from 4200 to 5100 Hz and the SPL range from 25-35 dB. The SOAE was suppressed by external tones, and the suppression contour had a sharply tuned tip near 5800 Hz. Distortion product otoacoustic emissions (DPOAE, 2F1-F2) were relatively normal; however, the levels of the primary tones increased in a highly nonlinear fashion as a function of input level at frequencies near the SOAE. Sodium salicylate (300 mg/kg, i.p.) completely abolished the SOAE, but the SOAE completely recovered after 24 h. Salicylates had no effect on the DPOAE. Nimodipine, a calcium channel antagonist had no effect on the SOAE or the DPOAE.

The cochlear microphonic (CM) potential, recorded from the round window, contained a prominent spontaneous CM at a frequency equal to the SOAE. In order to generate a sound evoked CM equal in amplitude to the spontaneous CM, the SPL of the external tone had to be approximately 65 dB SPL, i.e. 25-30 dB higher than the SOAE measured in the ear canal. This suggests that only a small percentage of the energy in the SOAE is transmitted back out of the cochlea into the ear canal. The spontaneous CM could be suppressed by an external tone and the suppression contours resembled those of the SOAE.

Many patients with SOAE fail to perceive their emissions. Thus, one of the important questions we wished to address is whether the SOAE is transmitted from the cochlea into the central nervous system. To answer this question, recordings were made from over one hundred auditory nerve fibers. The spontaneous discharge rates of normal auditory nerve fibers seldom exceed 100 spikes/s; however, the spontaneous discharge rates of the units in the animal with SOAE were distinctly bimodal with an abnormally high proportion of units with spontaneous rates between 90 and 140 spikes/s. Single tone suppression of spontaneous activity is rarely if ever observed in normal auditory nerve fibers; however, driven activity can be suppressed by a second tone. In the animal with SOAE, spontaneous activity could be dramatically suppressed in units with characteristic frequencies (CF) near the SOAE. The suppression areas were located above and below CF. These results suggest that the spontaneous activity was actually driven by the SOAE and that the SOAE is transmitted to the central nervous system.

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EFFECTS OF ACOUSTIC OVERSTIMULATION ON DISTORTION-PRODUCT AND TRANSIENT-EVOKED OTOACOUSTIC EMISSIONS.

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It is widely held that the various classes of evoked otoacoustic emissions (EOE) depend on micromechanical processes in the outer hair cells (OHC) of the organ of Corti. As the so-called active mechanisms are highly tuned, it has been suggested that EOE might provide frequency-specific indexes of cochlear condition, perhaps even in the case of infraclinical alterations. Although direct studies have confirmed that extensive loss of OHC is correlated with absence of EOE, several issues remain unsolved, as to whether or not quantitative relationships exist between EOE characteristics and cochlear status.

Acoustic overstimulation of the cochlea producing OHC damage and auditory fatigue or noise-induced hearing loss (NIHL) provides a good model for studying EOE physiopathology. Disclosing systematic EOE changes after noise exposure would open possibilities for early NIHL diagnosis by EOE monitoring, which seems at best premature thus far.

Evidence will be presented from animal experiments, showing that several properties of transient-EOE depend on the whole cochlear status including the high-frequency part in a fairly systematic way; it may account for a significant part of the large residual variance found when these EOE are tentatively correlated with audiometric data only at the same frequency. The range of interest of transient-EOEs might be extended using such properties. On the other hand, there is a number of lines of evidence that distortion-product otoacoustic emissions (DPOE) provide a more frequency-specific evaluation of cochlear mechanisms when appropriate stimulus parameters are chosen. However, several difficulties in using DPOEs as predictors of hearing sensitivity will be presented. For example, moderate NIHL of 20 to 40 dB can be associated with normal DPOE amplitudes, although they tend to exhibit abnormal growth functions. These results will be discussed in relation to the hypothetical sources of these emissions.

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THE EFFECTS OF NOISE ON COCHLEAR BLOOD FLOW

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While a variety of specific mechanisms have been proposed to account for the changes observed in the inner ear following high intensity sound exposure, they basically fall into two categories. Loud sound induces damage by: 1) direct mechanical trauma of the delicate organ of Corti structures, or 2) as a result of overdriving the metabolically dependent structures of the inner ear. Regardless of the mechanisms underlying noise induced hearing loss (NIHL) they are subject to the dynamic responsiveness of those functions of inner ear variables that stabilize the homeostatic environment. Changes in the homeostatic variables are required to prevent damage or effect repair as required to optimize the sensitivity and other receptor properties of the inner ear. Issues of the role of homeostatic mechanisms in NIHL come together in consideration of the effects of noise exposure on cochlear blood flow (CBF). Noise as the adequate stimulus for hearing requires a metabolic response of the inner ear, and this will depend on a change in CBF. Current data indicates that noise may decrease, increase, and/or have no effect on CBF. The changes reported in CBF can be described as variable and depend on the measurement technique. The changes are frequently inconsistent across studies and with other related measures, such as oxygen tension or metabolic activity.

On the basis of the literature and our recent studies employing laser Doppler flowmetry and intravital microscopy (IVM) of the responsiveness of the vasculature to noise, all of these changes in CBF may in fact occur. Our studies are based upon quantitative measures of vascular changes provided by IVM and the development of new techniques for of averaged laser Doppler measures of CBF. These techniques allow us to measure the phase and amplitude of pulsed flow through the inner ear and analyze the spectrum of the frequency components of flow. Our recent studies indicate that there is a dynamic responsiveness of blood flow in the inner ear that varies with the intensity of stimulation. Indeed both increases and decreases of CBF do occur during sound exposures. These appear to be largely driven by systemic factors during short sound exposure (i.e. <5 minutes). They may be influenced by neurally mediated changes, and perhaps by the direct mechanical effects of high intensity sound on local vascular elements. The latter lead to local constrictions and the disruption of regional flow in subsets of the lateral wall vessels. These changes and their implications will be discussed.

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EFFERENT AND PRIMING MODULATION OF NOISE-INDUCED HEARING LOSS

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A significant modulator of noise-induced hearing loss (NIHL) is the activity of the efferent pathways from the superior olivary complex to the cochlea. The role of these efferent pathways in reducing NIHL has been well established in studies in the guinea pig. In these studies it has been shown that a variety of manipulations can activate these pathways to protect the cochlea from loud sound exposures at a frequency in the mid-range of the guinea pig audiogram. The protective effects of the efferents could be attributed specifically to the crossed olivocochlear pathways, with the effects being mediated via the nicotinic cholinergic synapses of the crossed pathways. The protective effects of all manipulations were similar across exposures of varying intensity and duration, as might be expected from the fact that they were all mediated by the same pathways. The two most pertinent effects to the issue of the modulation of NIHL were that (a) protection was optimal with simultaneous application of the test manipulation and the loud sound exposure but did have long-lasting tonic effects, and (b) protection was graded to the amount of desensitization that would otherwise occur, with protection saturating at about 17 dB reduction in the desensitization caused by a loud sound. Finally, studies of descending influences on protection suggested that only one of the nuclei contributing fibres to the crossed efferent pathways was responsible for the protection.

Although an earlier study suggested that similar olivocochlear protection from loud sound did not occur in cats, more recent experiments have shown that such protection mediated by this pathway can be elicited in cats. Studies by others have also found similar crossed olivocochlear protective effects in rats, extending the generality of this role for a specific component of the olivocochlear pathways across species. In both species, as in the guinea pig, this protection is robust and elicited even when other efferent pathways to the auditory periphery are inactive. In detailed studies in the cat, there appeared to be, at least for relatively-loud sounds, a frequency-dependency to the protection. This dependency may reflect the susceptibility of the cat cochlea to different frequencies of loud sound exposure. These experiments also show that the amount of protection obtained in the cat was generally similar to, though larger, than that obtained in the guinea pig.

Finally, in earlier studies we demonstrated that modulation of NIHL could also be obtained by "priming" a cochlea prior to a high-level loud sound exposure. Here, an initial priming low-level exposure could significantly reduce the NIHL caused by a subsequent high-level loud sound exposure, even when there was no residual NIHL to the priming sound. Studies of this priming effect have since been extended by others into more detailed studies of "toughening" effects in NIHL. We have examined whether the two sets of effects modulating NIHL were independent by testing whether the priming effects were mediated by the olivocochlear pathways.

THE COMBINED EFFECTS OF HEARING LOSS AND HEARING PROTECTION: IMPLICATIONS FOR WORKER SAFETY

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This research assessed the effect of hearing protection on the localization of free-field sound sources by individuals with moderate bilateral sensorineural hearing loss. The results of twenty-four subjects with screened normal hearing and twenty-three subjects with high-tone thresholds of 40 to 60 dB HL, all over the age of 40 years, were compared. Individuals were tested with the ears unoccluded and fitted binaurally with conventional level-independent E-A-R foam plugs and E-A-R 3000 earmuffs, and level-dependent Bilsom 2392 muffs with limited dichotic amplification. Sound localization was studied using a horizontal array of six loudspeakers, positioned 60 deg apart. Within each of the four protector conditions, two one-third octave noise bands centred at 500 Hz and 4000 Hz were presented, both in quiet and in a continuous 65 dB SPL-white noise background. The intensity of the 300 ms stimulus was 80 dB SPL. One block of 120 forced-choice speaker identification trials was presented for each of the 16 listening conditions defined by protector condition, stimulus frequency and background. The results indicated that both groups achieved higher scores in the unoccluded than the protected conditions. For the 500 Hz stimulus, there was no difference due to the type of protector for either group. Right/left discrimination was close to 100% and front/back discrimination was 40%. Masking noise had no effect except for the normal subjects, unoccluded. For the 4000 Hz stimulus, in the normal subjects, high-frequency right/left discrimination was adversely affected, equally by muffs and plugs, but more so by the amplifying device. Front/back discrimination was affected more by muffs than plugs likely because of interference with pinna cues. A high proportion of the impaired group were unable to localize with the conventional protectors. In contrast, their performance with the amplifying device was similar to that of the normal group, even though their scores were significantly lower in the unoccluded condition. For both groups, a rearward bias in the perception of direction was evident for the Bilsom 2392 muff at the high frequency. Finally, hearing loss resulted in relatively longer decision response times in the unoccluded condition, and shorter response times in the protected conditions.

EFFECTS OF NOISE-INDUCED HEARING LOSS ON TEMPORAL RESOLUTION

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Temporal resolution can be modelled by an array of filters, with each filter followed by a nonlinearity and a sliding temporal integrator. The filtering and the nonlinearity are probably peripheral in origin, whereas the temporal integrator is more central. To account for the way that forward and backward masking combine in normally hearing subjects, the nonlinearity has to be compressive in form; the compression probably arises in the cochlea and reflects the operation of an active mechanism dependent on the integrity of the outer hair cells. This chapter considers temporal resolution in impaired hearing in the context of this model.

Noise-induced hearing loss can have adverse effects on temporal resolution because it often reduces the audible bandwidth of the stimuli, and because it results in a reduced sensation level of the stimuli. The sliding temporal integrator appears to be unaffected by hearing loss, although the nonlinearity preceding the integrator may be less compressive, and this can lead to reduced temporal resolution for sounds with slowly fluctuating envelopes. The reduction of compressive nonlinearity can also account for the reduced temporal integration that is often associated with cochlear damage.

MASKING BY LOW-PASS NOISE

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Persons with sensorineural hearing loss report difficulty hearing speech and other signals in the presence of background noise. A psychoacoustic correlate of this report is the masking of speech and other signals by maskers that vary in their spectral characteristics. For the past several years as a part of a large scale study of presbycusis, we have examined masking produced by a low-pass filtered noise in normal and hearing-impaired young and older adults, and in normal and hearing-impaired young and older gerbils. Results are straightforward in many respects, but contradictory and paradoxical in others.

In a psychophysical task with human subjects masked thresholds within the frequency region of the low-pass (1 kHz) masker were essentially the same for all subjects, regardless of age or hearing level. Off-frequency masked thresholds (1.5-2 kHz) were much less predictable, ranging over 40 dB. Neither excessively high nor excessively low masked thresholds were related to aging per se nor strongly related to elevated auditory thresholds.

In normal and aged gerbils, the identical low-pass masker was used to mask auditory brainstem potentials (ABR) that were produced by 1.8 msec tone bursts. In young normal hearing animals on- and off-frequency masked thresholds were predictable whereas in older animals both on- and off-frequency masked ABR thresholds were larger than predicted. In particular, the off-frequency masked ABR thresholds at 2 and 4 kHz were 10-20 dB higher than predicted. In order to identify the locus of the overmasking effect observed in aged gerbils, the identical masking paradigm was completed using the compound action potential (CAP) recorded by a round window electrode. Results observed previously in the ABR were confirmed at the level of the auditory nerve. Thus, the overmasking effect observed in the ABR of aged gerbils is present in the auditory nerve and, therefore, has its locus in the auditory periphery.

Inasmuch as overmasking masking is reflective of a decrease in the frequency selective properties of the ear, one might expect that other measures of frequency selectivity in the auditory periphery would be similarly decreased. However, tuning curves of single fibers had elevated tips indicative of a loss of sensitivity but otherwise normal characteristics. Moreover, two-tone rate suppression was measured both below and above the characteristic frequency of the unit. These are paradoxical results. That is, in quiet-aged animals frequency selectivity of the auditory periphery is normal when assessed by single fiber tunings curves and the presence of two-tone rate suppression, but abnormal when assessed by the upward spread of masking reflected in the CAP and ABR. These results will be discussed in light of additional single fiber, CAP, and ABR data.

**EFFECTS OF COCHLEAR HEARING LOSS ON
SPECTRO-TEMPORAL PROCESSING**

J.W. Hall, USA

SOUND CONDITIONING PROTECTS THE OUTER HAIR CELLS FROM NOISE TRAUMA

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Sound conditioning with a continuous low-level, long-term acoustic stimulation provides protection against a subsequent noise exposure known to induce either a temporary or a permanent threshold shift. The sound conditioning, or "training" investigations originated when it became apparent that the outer hair cells had a motile response to electrical, chemical, and mechanical stimulation. The hypothesis was to test whether the outer hair cells could be "trained" or sound conditioned in attempt to afford protection against a subsequent noise trauma. The aim of the present study was to determine if sound conditioning results in an altered morphology to the organ of Corti or an altered pattern of damage after subsequent noise exposure. In addition, to test whether the outer hair cells are directly affected by sound conditioning, measures of the distortion product otoacoustic emission both during the sound conditioning and after the subsequent traumatic exposure will be compared to a control, unconditioned group.

The results suggest that low-level, long-term conditioning does not cause any significant functional or morphological alteration to the guinea pig cochlea. In comparison to the control, unconditioned group, the consequence of a subsequent traumatic exposure on the sound conditioned group, 1) reduces the degree of outer hair cell loss; 2) causes an altered pattern of damage; and 3) maintains the amplitude of the distortion product otoacoustic emission over a wide frequency range.

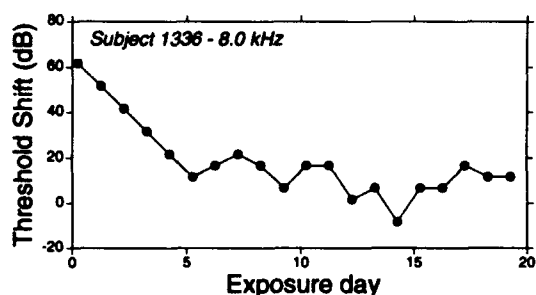
While the mechanisms underlying the protection against noise trauma by sound conditioning are not yet known, there are several likely candidates responsible for this phenomenon. These include the middle ear muscles, the efferent system, as well as a modulation of cochlea blood flow, or metabolism.

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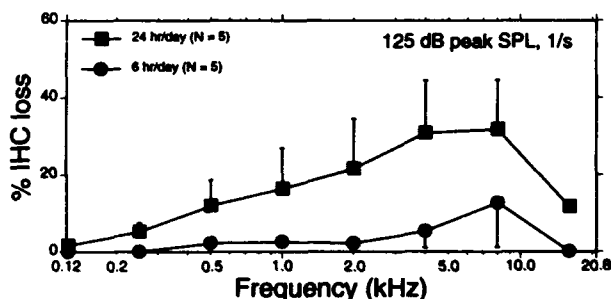
THRESHOLD SHIFT DYNAMICS FOLLOWING INTERRUPTED IMPACT OR CONTINUOUS NOISE EXPOSURE: A REVIEW

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A resistance to the development of noise-induced threshold shifts (TS) has been found in experimental animals following two very different noise exposure paradigms. (1) Priming subjects, with relatively low-level exposures has been shown to alter the sensitivity of the auditory system to TSs from subsequent high-level exposures, and (2) Interrupted noise exposures repeated on a daily schedule have shown that the auditory system can gradually develop a resistance to the noise such that, on subsequent days, thresholds improve despite the continuing exposure. These two effects have been collectively termed the "toughening effect" and can be elicited by continuous or impact noises. Toughening can manifest itself in the transient behavior of TSs, in reduced permanent TSs and in reduced sensory cell losses. Correlates have also been found in VIII-nerve physiology and in distortion product cochlear emissions. The magnitude, time course and frequency specificity of the toughening effect are related to stimulus parameters. Explanations of the origins of this effect usually focus on the outer sensory cell system but correlations with anatomical data are not consistent. This paper will review the accumulating data base on this interesting aspect of noise-induced hearing loss. [Research sponsored by NIOSH Grant R01 OH02317.]



Example of the more than 40 dB TS recovery at 8 kHz in an individual animal exposed to a daily six-hour 113 dB peak SPL impact noise for 20 days.



Mean percent inner hair cell loss (IHC) for interrupted (6 h/day for 20 days) and non-interrupted (24 h/day for 5 days) exposures to 125 dB peak SPL impacts. The error bars represent one standard error of the mean.

STIMULATION INDUCED RESISTANCE TO NOISE INDUCED HEARING LOSS

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Acquired resistance to noise induced hearing loss is reflected in both decreasing temporary threshold shift (TTS) with repeated exposures (10-50 dB) and, with exposure to higher level traumatic noise, increased resistance (10-25 dB) to permanent threshold shift (PTS). This paper reviews the studies on increased resistance to PTS. Several trends emerge from this research. First, low frequency, intermittent exposures, render the auditory system more resistant to future higher level low frequency exposures, as well as, impulse and impact noise exposures. The increased resistance to PTS can be established with as few as two six-hour exposures and the effect appears to last for at least thirty days. By contrast, high frequency or low frequency intermittent exposures do not render the auditory system more resistant to future high frequency exposures.

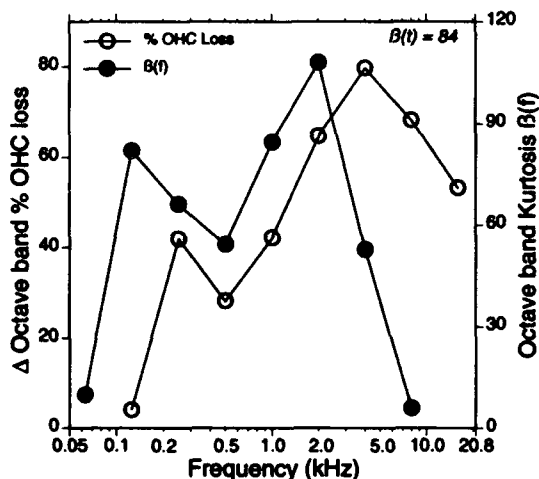
The magnitude of the acquired resistance effect suggests fundamental changes in auditory physiology. In addition, the strength and persistence of the effect suggests that it may be possible to develop active prophylactic exposures for individuals unable to use conventional hearing protective devices.

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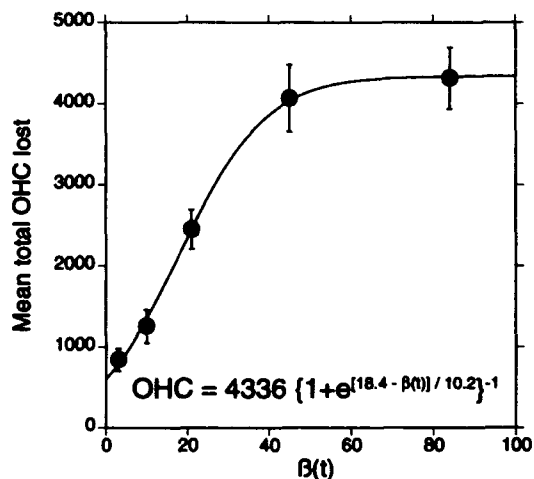
THE APPLICATION OF FREQUENCY AND TIME DOMAIN KURTOSIS TO THE ASSESSMENT OF COMPLEX, TIME VARYING NOISE EXPOSURES.

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Industrial noise is typically characterized by high-level noise transients that are superimposed on a continuous noise background producing a complex (non-Gaussian) temporal signal. High kurtosis exposures have been shown to pose a higher risk of hearing loss to the repeatedly exposed individual than an equivalent energy Gaussian noise. Current measurement practice relies on a time averaged energy metric, the L_{eq} , to characterize noise exposures. The L_{eq} will be inadequate for any exposures in which temporal variables are important in determining the extent of trauma. This paper shows that, for continuous noise exposures having the same L_{eq} and spectra, the statistical properties of the signal are important in the prediction of acoustic trauma. The results indicate that the kurtosis computed on both the time domain signal, $\beta(t)$, and the frequency domain signal, $\beta(f)$ in conjunction with the L_{eq} can order the magnitude of acoustic trauma and reflect its frequency specificity. These conclusions are based upon the results obtained from 114 chinchillas exposed to one of twelve different noises in a five-day, asymptotic threshold shift producing exposure paradigm. Hearing thresholds were obtained using brainstem evoked auditory responses and sensory cell populations were quantified using surface preparation histology. [Research sponsored by NIOSH Grant R01 OH02317.]



Distribution of outer hair cell loss (OHC) compared to $\beta(f)$ for a 100 dB L_{eq} non-Gaussian noise exposure



Relation between $\beta(t)$ and total OHC lost for five equal-energy exposures having the same spectrum.

**EXCITOTOXICITY AND PLASTICITY OF THE IHC-AUDITORY
NERVE SYNAPSE CONTRIBUTE TO BOTH TTS AND PTS.**

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**INDIVIDUAL DIFFERENCES IN PERIPHERAL SOUND TRANSFER
FUNCTION: RELATION TO NIHL.**

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The peak-frequency of sound transfer function (STF) from free-field to the tympanic membrane vary one octave from 2 to 4 kHz between individuals. The major reason for these variations are differences in the size of ear canals. The influence of the ear canal size on noise induced hearing loss (NIHL) was studied in two experiments. The STFs were measured with a probe-provided miniature microphone in an anechoic chamber.

In the first experiment temporary threshold shift (TTS) was studied in 36 subjects exposed to 2 and 4 kHz narrow band noise in free field at two different test-occasions. The subjects were divided into three groups depending on their STFs. The average TTS differed significantly between groups.

In the second experiment 100 male subjects' left and right ears' STFs were measured with the same technic described above. Further, the left and right ears' hearing thresholds were registered with Békésy-audiometry and the ear canal length and volume measured. The high-frequency hearing thresholds and the ear canal volume were significantly correlated to the frequency of maximum STF-magnitude.

These two experiments indicate that individual differences in STFs could influence the sensitivity to NIHL. The STFs of subjects with large ear canals are more low-frequency dominated than those with small ear canals. Further, subjects with low-frequency dominated STFs are more sensitive to NIHL than other subjects.

EXTENDED HIGH-FREQUENCY HEARING LOSS FROM NOISE EXPOSURE

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Noise characteristically leads to hearing loss with a maximum around 4000 or 6000 Hz, but may also lead to hearing loss above the conventional audiometric frequency (CF) range. A review of extended high-frequency (EHF) audiometry and EHF hearing loss from steady-state and impulse noise exposure is given.

Noise-induced hearing loss (NIHL) is then compared in the CF and EHF range for around 175 males aged 18-59 years with known NIHL in the CF range from occupational exposure to steady-state and impulse (weapon) noise. Following an interview on noise exposure history, air conduction and bone conduction thresholds were determined in the CF range, and supplemented by air conduction thresholds in the EHF range, 9-18 kHz, in 1 kHz steps, using the Interacoustic ASHF10 tone generator, Koss HV/1A headset, and 1/3 octave band contralateral masking by Bruel & Kjaer random noise generator. The subjects were grouped according to age. The CF hearing losses were classified as Grades I through IV according to Man & al. (1981).

EHF thresholds were throughout higher than in age-matched subjects not exposed to noise. For slight CF acoustic trauma, NIHL Grade I, the EHF threshold elevation was age-related, as if additive to presbycusis. For severe CF acoustic trauma, NIHL Grades III and IV, the EHF thresholds were poor and largely overlapping for the different age groups above 2 kHz. Supported by case studies this indicated that single events of impulse noise exposure exceeding a critical level may cause extensive hearing loss from 2-3 kHz and up. Group data for slight acoustic trauma Grades I+II for the younger subjects showed a tendency towards EHF hearing loss about one octave above the characteristic 3-6 kHz "dip". This extra notch was more evident in some individual audiograms. The findings are discussed related to asymptotic threshold shift, and aspects of animal data, basilar membrane mechanics, and pathophysiological mechanisms at the haircell level.

OTOTOXIC EFFECTS OF CHEMICALS ALONE OR
IN CONCERT WITH NOISE; A REVIEW OF HUMAN STUDIES

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Attention in occupational health to the prevention of hearing loss has focussed almost entirely on workplace noise. The literature on the effects of noise exposure on workers' hearing is extensive. Conversely, the effects of other factors such as general worker health, hand-arm or whole-body vibration, physical work load, and chemical exposure have received comparatively little attention in terms of their consequence for hearing loss. There is a growing body of evidence suggesting that many other factors and exposures commonly observed at the workplace can contribute to the occurrence of occupational hearing loss. Because current regulations consider only high-intensity noise as ototraumatic, it is conceivable, in the light of research results, that large numbers of workers have unmet needs regarding prevention of occupational hearing loss. The hearing-protection and noise-reduction strategies currently in use may be insufficient. Current exposure limits for both noise and other agents may not be appropriate for protecting workers in environments where simultaneous exposures occur. Evidence from human studies on the ototoxicity of environmental chemicals will be reviewed. This paper will also address the need for a better understanding of the risk posed by combined exposures and consider various aspects of effective hearing loss prevention programs.

INTERACTIONS BETWEEN AGE-RELATED AND NOISE-INDUCED HEARING LOSS

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The most common causes of sensorineural hearing loss are exposure to noise, aging, and the interactive effects of noise and aging. Exposure to noise is oftentimes separated into occupational vs non-occupational (socioacusis) categories. Sometimes aging effects are separated into "pure aging effects" and thus differentiated from nosoacusis (hearing loss produced by disease factors, trauma). Even pure aging effects are sub-divided (arguably) into four distinct categories based upon the pathologic anatomy (sensory, neural, metabolic, and mechanical) and specific audiometric details. The coexistence of multiple causes of sensorineural hearing loss as well as many forms of age-related hearing loss presents several necessarily challenging problems. For example, one may be asked to determine the hearing loss produced at one job site 35 yrs in the past as well as the noise-induced hearing loss produced at another job site 10 yrs later, and in addition, specify the hearing loss associated with increased chronological age, socioacusis, and nosoacusis. A traditional, retrospective assessment of the effects of noise on hearing for an individual has been achieved by the judicious assessment of serial audiograms in combination with accurate, quantitative noise exposure data. In the absence of serial audiograms and quantitative noise-exposure data, it was considered impossible to assign, with suitable accuracy, hearing losses to different work sites or employers, or to differentiate socioacusis from nosoacusis and from occupational exposures to noise. In the past few years and particularly because of the availability of an international standard (ISO 1999) quantitative methods have emerged which are designed to separate the effects of exposure to occupational noise from the effects of other factors, particularly aging, but implicitly including socioacusis and nosoacusis. Currently, we are examining some of the allocation methods recently proposed by applying them to some of the data available in the scientific literature.

ABSTRACT

ESTIMATION OF OCCUPATIONAL CONTRIBUTION TO HEARING HANDICAP

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Purpose. A simple method, based on ISO-1999, of allocation of hearing handicap between noise-induced hearing loss (NIHL) and age-related hearing loss (ARHL) is described and demonstrated.

Methods. Distributions of noise-induced permanent threshold shift (NIPTS), age-related permanent threshold shift (ARPTS), and resultant hearing levels (HLs) are shown graphically. Assumptions for combining and separating NIPTS and ARPTS are discussed, including the correlation of NIPTS and ARPTS susceptibilities. Methods of allocation are then demonstrated using audiograms.

Results. Neither the "age-correction" approach, allocating median ARPTS, nor the opposite approach using median NIPTS, fairly represents the relative contributions of noise and aging. Methods assuming perfect ($r = 1$) or absent ($r = 0$) correlation yield allocations which are reasonable, but differ slightly from one another. A simpler method based on the ratio of medians of ARPTS and NIPTS yields results which are usually intermediate between the $r = 1$ and $r = 0$ methods.

Conclusion. For many medical-legal and compensation settings, the median-ratio method provides a straightforward and fair allocation between NIHL and ARHL.

PSYCHOACOUSTIC PERFORMANCE IN WORKERS WITH NIHL.

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Psychoacoustic performance impairment is examined within an ecological perspective which focusses on the interaction between individual capacities and environmental demands in terms compatibility or mismatch. Accordingly, the effects of hearing loss are described within work environments in which hearing impaired workers evolve. First, Hearing impairments associated with NIHL are reviewed briefly, including reduced hearing sensitivity and reduced frequency, temporal and spatial resolution. Secondly, typical auditory demands in the work place are examined, considering the type of sound sources used, the prevailing propagation conditions in industry and the resulting characteristics of typical auditory signals. Thirdly, performance impairment in the work setting is analysed in terms of detection, discrimination and identification of auditory warning signals and localization of their sources. Finally, a procedure is proposed to match auditory demands in the work place with the altered auditory capacities of hearing impaired workers. Specifically, a clinical procedure for measuring auditory filters is used in conjunction with a computer model (DetectsoundTM) adapted for predicting individual masked thresholds among hearing impaired individuals. A test of the validity of predicted masked thresholds between 0.5 and 4 kHz as been conducted with 52 individuals with different degrees of sensorineural hearing loss using four spectral configurations of a high level wideband noise. Individual errors of prediction were inferior to 5 dB for a large majority of the cases with an intended bias towards a slight overestimation of the predicted thresholds. It is concluded that it is feasible to adjust auditory warning signals characteristics in order to meet the constraints imposed by residual hearing capabilities among hearing impaired workers.

(Acknowledgement: The development of the clinical procedure and its validation were part of a project financially supported by the Institut de recherche en santé et sécurité du travail du Québec).

DISTRIBUTIONS OF HEARING THRESHOLD LEVELS IN POPULATIONS EXPOSED TO NOISE

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Investigation of noise-induced hearing loss in humans is restricted to observational studies. Deliberate experimental manipulation of noise exposure parameters in order to assess their effect on hearing thresholds is not ethically justifiable. Hence, study of noise-induced hearing loss entails comparison of groups of subjects who have been exposed to noise, usually as a part of their occupation and lasting many years, with controls subjects who have not been exposed to noise. The long-term nature of noise exposures implies that exposed subjects will also have accrued hearing loss with age, and the control subjects must be chosen to reflect a similar effect of age. The same argument applies to any other factors that may influence hearing, apart from the noise exposure in question.

Many studies have been published that describe the distributions of hearing threshold levels in subjects who have been exposed to noise. This historical database is of particular importance because it is increasingly difficult to find current groups of individuals with prolonged unprotected exposure to high levels of noise, due to improved regulation in industry. It is in such groups with high levels of exposure that the effects of noise are most easily distinguished from extraneous factors.

Examination of the historical data indicates a wide variation in median hearing threshold levels of subjects with minimal noise exposure. Few studies included rigorously matched control groups, and many studies relied on the literature for normative controls. Hence, a major factor influencing the interpretation of the historical data is the choice of appropriate controls.

Epidemiological work in the UK has demonstrated hearing threshold levels in a representative sample of the general population that are higher than many previously reported studies, even after careful screening for ear disorder and risk factors including noise exposure. There is an important socioeconomic factor. The present analysis examines historical data on noise-exposed groups in the light of this new knowledge from formally sampled controls. It also examines hearing threshold levels in the population sample as a function of self-reported exposure to occupational, recreational and gunfire noise. The main conclusion is that many previous studies have under-estimated the extent of hearing loss in non-exposed controls and have hence over-estimated the influence of noise. One reason for this under-estimation is lack of consideration of socioeconomic status, a factor that has a profound influence on most aspects of health.

COMPENSATION FOR TINNITUS IN NOISE INDUCED HEARING LOSS

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Noise exposed individuals as well as representatives for unions demand that occupational noise induced tinnitus should be compensated similiary to occupational noise induced hearing loss. However, there are several problems, particularly the completely subjective character of the tinnitus symptom. On the other hand, many patiens suffer more from tinnitus than from hearing loss and like hearing loss the treatments available are as yet of somewhat limited effectiveness. If noise induced tinnitus shall be compensated for, a requirement is detailed history and work-up. In doubtful cases, this should preferably be repeated after an interval of some weeks-months. We maintain that it is rather the general annoyance by tinnitus than its possible influence on speech recognition which is handicapping for the individual.

HEARING LEVELS OF U.S. INDUSTRIAL WORKERS EMPLOYED IN LOW-NOISE ENVIRONMENTS

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The purpose of this project was to develop a control population which describes hearing threshold levels of American industrial workers free from exposure to excessive occupational noise. This control population would then be utilized studies of the risk of occupational hearing loss in workers exposed to significant occupational noise. The recently adopted international standard ISO 1999, "Acoustics-determination of occupational noise exposure and estimation of noise-induced hearing impairment" (ISO, Geneva, 1990) contains two control or comparison databases as appendices. One database (Annex A) is obtained from a highly screened population. The database is useful for determining hearing loss due to age alone (pure presbycusis); it is not generally useful for evaluating occupational noise-induced hearing loss in an industrial workforce because it underestimates hearing levels of individuals who are unscreened except for occupational exposure to noise. The other database (Annex B) describes hearing threshold levels for the better ear from an unscreened population. However, surveys derived from random samples of the entire population may not be appropriate for comparison to industrial workers because of differing nonoccupational noise exposure histories in the two groups.

To develop a control database for industrial workers, hearing threshold levels were evaluated from the database of hearing conservation programs of twenty-two American and Canadian companies produced to ANSI working group S12.2 and provided by the National Institutes for Occupational Safety and Health (NIOSH, 1988). Ten of the databases which included workers exposed to daily noise levels with time-weighted average (TWA) exposures at, or below 85 dBA were selected for further evaluation. The sample was also restricted to audiometric tests obtained within the first two years of employment. Hearing threshold levels were evaluated by age, gender and race. The major findings of the study were:

A. Hearing levels of U.S. industrial workers were worse than those reported in Annex B. The difference was larger for males than females, and was as much as 20 dB at 3,000, 4,000 and 6,000 Hz for males over age 40.

B. Hearing levels of young individuals did not differ by gender or race. However, with increasing age, hearing in white males deteriorated more severely at 3,000, 4,000 and 6,000 Hz with age than that observed in other groups. By age 60 the median hearing threshold level at 4,000 Hz for white males was 16 dB worse than for black males.

These findings show that U.S. industrial workers have worse hearing than that observed in a random sample from the U.S. population, and that white males lose more hearing at 3,000, 4,000 and 6,000 Hz as they age. The principal factor in causing the additional loss is exposure to noise from hunting and target shooting, an avocation enjoyed by 60-70 percent of the white male workers, but by few blacks or women.

CURRENT STANDARDS FOR OCCUPATIONAL EXPOSURE TO NOISE

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The vast majority of national standards for occupational exposure to noise specify an upper limit of 85 dB(A) as a daily average exposure level. A number of them (the U.S.A., for example) permit an average exposure level of 90 dB(A) for purposes of engineering controls. With respect to the exchange rate (the relationship between exposure duration and level), most nations follow the lead of ISO 1999 (1990) and use L_{Aeq} , the 3-dB rule, with only a few exceptions (such as Brazil, Israel, and civilian U.S.A.). Several national standards specify an upper limit for non-impulsive noise at 115 dB(A), and many prescribe an upper limit of a peak sound pressure level of 140 dB for impulsive noise.

Certain issues are inherent in the development of any standards for occupational exposure to noise. Important regulatory or legislative issues include not only the permissible exposure limit, the exchange rate, and limits for impulse noise exposure. These issues also include methods for classifying and assessing the attenuation of hearing protection devices, and particulars of audiometric testing, such as the frequency of tests and the training of audiometric technicians. There appear to be certain weaknesses in standards: for example, the absence of methods for evaluating hearing conservation program effectiveness and the absence of procedures for dealing with the problem of progressive hearing loss.

There is considerable variation in the structure and enforcement of noise standards. Enforcement of some national programs appears to be more vigorous than others, where both resources and resolve can be lacking. The noise standards of some nations are brief and concise, relying on codes of practice for their interpretation and thus providing employers some flexibility in their methods of compliance. Other nations use non-mandatory guidelines.

The International Institute of Noise Control Engineering (I-INCE) has formed a Working Party composed of nine I-INCE member societies, to review current knowledge and practice concerning "Upper Noise Limits in the Workplace." The Working Party's proposed recommendations include an 85-dB(A) exposure limit, a 3-dB(A) exchange rate, and various strategies for controlling noise exposure.

INTERNATIONAL REVIEW OF FIELD STUDIES OF HEARING PROTECTOR ATTENUATION

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A quantity of fundamental importance in the understanding and description of hearing protector device (HPD) performance, is the attenuation provided by devices under field conditions. Laboratory studies, no matter how ambitious or comprehensive are of little value without a means to reference them to protection under conditions of actual utilization. An ever increasing number of studies examining delivered attenuation in work-site settings have continued to document the discrepancy between labeled attenuation values and real-world performance.

The first author has been developing a data base of field performance studies for over a decade, and in this report updates the results to summarize 22 field studies of HPD attenuation conducted from 1975 to 1993. The combined data comprise over 100 industries in 8 countries, with a total of approximately 2900 subjects. The studies were all conducted at work sites with actual employees or in military settings with conscripts as subjects. Various techniques, such as real ear attenuation at threshold using large circumaural cups to generate a sound field, or miniature microphone tests with microphones mounted outside and inside of the earmuff cups, were utilized to acquire the attenuation values.

The data were examined in terms of mean octave-band attenuation values and related standard deviations, as well as computed Noise Reduction Ratings (NRRs), a single-number attenuation descriptor very similar to the Single Number Rating (SNR) specified in ISO 4869-Part 2 (1993). Regardless of which metric was utilized, NIOSH's statistical analyses found the differences between work-site and laboratory data were significant and had meaningful implications for selection of appropriate hearing protectors. The differences also varied enough from protector to protector that it would be questionable practice to apply a single "correction factor" to laboratory data as it is presently established.

In terms of NRRs, the field values achieved by 84% of the target population were approximately 1/4 of the labeled values for earplugs, and somewhat greater than 1/2 of the labeled values for earmuffs. Work-site NRRs for many earplugs were less than 10 dB and exceeded 15 dB for no single hearing protector. The need to develop laboratory methods that provide data predictive of work-site performance is clear. The efforts of ANSI Working Group S12/WG11 (*Field Effectiveness and Physical Characteristics of Hearing Protectors*), as well as NIOSH-funded researchers, are directed towards just such developments. Meanwhile, hearing conservationists should de-emphasize the importance of published attenuation values and focus on issues that can make a difference in hearing conservation practice, such as training, motivation, supervision, and rigorous enforcement of HPD utilization. /fifthont.wp

ASSESSMENT OF HEARING PROTECTOR PERFORMANCE IN IMPULSIVE NOISE: UPDATE OF RESEARCH ACTIVITIES WITHIN THE EC-FUNDED IMPRO PROJECT.

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The present standard for assessment of hearing protector performance, ISO 4869-1, is based on subjective measurements near the human threshold of hearing. Due to its very nature, this standard is not suited to determine the attenuation of peak levels of impulsive sounds, nor is it suited to assess performance of hearing protectors that show level-dependent behavior. Given that both, maximum allowable peak levels and hearing protector attenuation are subject to EC directives, it is mandatory that new methods for assessment of hearing protector performance are developed. The aim of the EC-funded IMPRO project, which started in 1993, is to investigate whether methods based on acoustic measurements can be used for this purpose.

In the present paper, a survey will be given of research activities within the IMPRO project. These fall into four categories: (1) development of a MIRE (Microphone-In-Real-Ear) technique for measuring sounds under ear plugs, suitable for all types of plugs including custom moulded ones (a MIRE technique for sound measurements under ear muffs was already developed in a feasibility study preceding the IMPRO project); (2) MIRE measurements and simultaneous measurements outside the hearing protector of impulsive sounds, performed both in the laboratory and in actual workplaces; (3) measurements of hearing protector attenuation for impulsive sounds performed with ATF's (Acoustic Test Fixtures); and (4) analysis of the data in order to determine the simplest adequate means of quantifying the attenuation of linear and level-dependent hearing protectors in impulsive noise with regard to risk of hearing loss.

Though the survey will touch upon all four categories, special attention will be given to the MIRE technique, applied to ear plugs, and to the analysis of the simultaneous sound recordings under and outside hearing protectors. MIRE measurements with plugs are of interest because it turns out to be very difficult, perhaps even impossible, to satisfy demands of accuracy, feasibility and subject safety while leaving the plug intact. The recordings are discussed because they provide detailed insight into the time and level dependent response of hearing protectors to impulsive sounds.

**ESTIMATED REDUCTIONS IN NOISE-INDUCED HEARING LOSS BY
THE APPLICATION OF ANR HEADSET.**

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ACTUAL PERFORMANCE OF HEARING PROTECTORS IN IMPULSE NOISE/ NONLINEAR BEHAVIOR

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Recent observations conducted on soldiers exposed to large impulse noises indicate that the protection afforded by the earplugs and the earmuffs is currently underestimated (Dancer et al., 1992; Patterson et al., 1993). These results could be wholly or partly due to some hearing damage mechanisms peculiar to the high level impulse noises (Price and Kalb, 1991). They could also be related to our lack of knowledge regarding the actual attenuation afforded by the Hearing Protectors (HPs) when exposed to very large impulses. Sound attenuation measurements performed by subjective method (ISO 4869-1) at low sound pressure level (steady-state noise) cannot account for the actual performance of the protectors for impulses of very high peak level (140 to 190 dB SPL). The same is true for the "Microphone-In-Real-Ear" (MIRE) technique. The only possibility to assess the actual behaviour of the HPs when exposed to impulse noise (up to 190 dB SPL), to characterize their nonlinearity (if any), and to measure spectrum and peak pressure attenuations, is to use an Acoustical Test Fixture (ATF): an artificial head with an ear simulator.

We developed an artificial head equipped, (i) with the Head Acoustics® external ear which provides circumaural, pinna and earcanal flesh simulation and, (ii) with a Brüel & Kjaer ear simulator. To allow the measurement of sound pressure levels up to 190 dB, the original 1/2" microphone was replaced by an underpolarized 1/4" microphone. The dynamics of this ATF is better than 80 dB from 0.4 to 5 kHz. The attenuation afforded by 7 earplugs (E.A.R.® foam, E.A.R. Ultrafit®, E.A.R. Ultratech®, E.A.R. Link®, RACAL Airsoft®, RACAL Gunfender®, and a perforated earplug prototype) and 2 earmuffs (Willson SB 258® and E.A.R. Ultra 9000®) was measured during exposure to Friedlander waves of 150, 170 and 190 dB peak pressures (A-durations: 0.2 and 2 ms) under normal and grazing incidences.

Some HPs are quasi linear, i.e., their spectrum and peak pressure attenuations are almost independent of the peak pressure and of the duration of the impulses (E.A.R. Ultrafit®, RACAL Airsoft®), some others are nonlinear, i.e., their spectrum and peak pressure attenuations depend on the peak pressure of the impulses. However, the nonlinearity can be "favourable": i.e., the attenuation increases with the level of the impulse (by 10 dB for each increase of 20 dB of the impulse level beyond 140 dB for the RACAL Gunfender® earplug) or "unfavourable": i.e., the attenuation decreases with the level of the impulse (beyond 170 dB for the Willson SB 258® earmuff). These two nonlinear mechanisms can even combine (E.A.R. Ultra 9000® earmuff).

Each HP behaves differently and requires specific measurements in order to assess its actual attenuation characteristics when exposed to large impulse noises.

**TEMPORARY THRESHOLD SHIFTS PRODUCED BY HIGH INTENSITY FREEFIELD IMPULSE NOISE
IN HUMANS WEARING HEARING PROTECTION**

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Exposure to high intensity impulse noise produced by modern military weapons is known to be hazardous to hearing. Hearing protection is required; however, there is no generally accepted theoretical way to predict whether protection will be adequate for the highest noise levels. This has led us to empirically determine the safe limits of exposure to impulse noise when hearing protection is used by exposing human volunteers under controlled conditions. Over the past 5 years, a series of studies has been conducted to determine the maximum safe exposure to high intensity freefield impulse noise. An exposure was considered to be safe if it produced only a small temporary threshold shift (TTS < 25 dB) in a small percentage of the volunteers exposed. Three different impulses were used with A-durations of 0.8, 1.4, and 2.9 ms. Both the level and number of impulses were varied to find the maximum tolerable exposure for combinations of these parameters. The peak sound pressure levels ranged up to 196 dB. The number of impulses was varied from 6 to 100. Approximately 60 volunteers were exposed to each type of impulse, allowing high confidence estimates of the exposures which would produce no significant TTS in 95% of the exposed population. The hearing protection used was an ear muff which had been modified to simulate a poor fitting protector. The results of these studies indicated that even with a relatively poor hearing protector, combinations of level and number of impulses which far exceed our currently accepted exposure limits could be tolerated by 95% of the volunteers.

UNDERWATER HEARING AND OCCUPATIONAL NOISE EXPOSURE

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There is a risk of damage to hearing due to underwater occupational noise exposure, and there are no widely accepted noise exposure limits for underwater use. New experiments to establish the underwater hearing thresholds and hearing mechanisms were carried out using 54 normally hearing sport divers. The results show that hearing threshold is less sensitive underwater than in air by 22 dB at 0.5 kHz and 60 dB at 4 kHz. The bone conduction pathway is the dominant route for underwater hearing mechanisms. The results mean that the "A-weighting scale", the well established decibel unit to describe noise exposure limits in air, is not suitable for underwater use. A "W-weighting scale" equivalent to the A-weighting scale is developed, and noise exposure limits are proposed for prevention of occupational noise induced hearing loss underwater.

EFFECTS OF INTENSE NOISE ON FETAL SHEEP AUDITORY BRAINSTEM RESPONSE

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The extent to which the fetus detects sounds originating outside its mother continues to be of interest to scientists in many fields of inquiry. Because sounds penetrate easily to the fetal head, intense sounds generated outside the mother may produce changes in fetal hearing. This study was designed to evaluate the effects of noise exposure on the auditory brainstem response (ABR) of fetal sheep. Cochleae were removed from randomly selected animals and prepared for light microscopy.

Fetuses were prepared for chronic recordings during sterile surgery. A midline abdominal incision was made and the uterus was opened. The head was exteriorized and instrumented with a bone oscillator and electrodes fixed to the skull. The fetus was replaced and the uterus and abdomen were closed. Electrodes and the oscillator wires were brought outside the ewe and stored in a pouch secured to the maternal flank.

ABRs were recorded from in utero fetal sheep from 111 to 136 days gestational age (dGA) (Total dGA is 145). For two groups, ewes were exposed to 120 dB SPL noise for 16 hours while carrying fetuses of either 113 or 130 dGA. A third group of animals was nonexposed. ABRs were recorded to clicks and tone bursts before and at different times following the exposure.

Between 111 and 136 dGA, ABR thresholds improved 35-45 dB in the nonexposed fetuses. Increases in post-exposure thresholds and latencies were identified for the animals exposed at 130 dGA. Latencies for three of the four vertex-positive ABR peaks were prolonged ($p < 0.05$) after the exposure. ABR thresholds were temporarily elevated in this group ($p < 0.01$). The animals exposed at 111 dGA did not show a threshold shift immediately after the exposure. However, this group had higher thresholds at later dGA than did the nonexposed group ($p < 0.05$). Using serial celloidin sections, no differences were found in the appearance of hair cells between groups of nonexposed and exposed fetal cochleae.

Intense noises penetrated the uterus and resulted in alterations of the fetal ABR. For animals exposed later in fetal life, ABR thresholds recovered by 48 hours. For fetuses exposed at 113 dGA, ABR thresholds did not develop as well as thresholds for nonexposed animals when measured 17-23 days after the exposure. Light microscopy did not show sensory cell differences between noise-exposed and nonexposed animals regardless of when the exposure occurred.

Supported by NIH HD 20084 and March of Dimes Birth Defects Foundation.

REVIEW OF NON-AUDITORY EFFECTS OF BLAST OVERPRESSURE

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The hearing of a soldier can be protected from blast noise to the extent that non-auditory effects set the upper limits of safe exposure. Therefore, it is important for hearing professionals working with blast noise to understand the current knowledge base with respect to non-auditory blast injury.

The etiology and treatment of blast injury were developed during and just after World War II. Continued research over the years has resulted in a good understanding of the effects of classical Friedlander blast waves. The fact that gas-containing organs are more vulnerable to direct blast than solid organs has led to the development of injury prediction curves for single exposures to Friedlander waves in man. These criteria assume the lung to be the primary target organ in terms of immediate pathophysiological effects. Recent studies have shown that the upper respiratory tract can be an important indicator of the presence of other non-auditory blast injuries during medical triage. It was observed that demonstrable hemorrhagic changes tended to occur in the upper respiratory tract either before or with injuries to the lungs. Contusions and/or ruptures of the gastroenteric tract are also important injuries which can occur concurrently with upper respiratory tract injuries. Care must be taken that such injuries are not overlooked as the magnitude of these effects can increase with time.

The degree to which these findings are extendable to complex wave environments is not as well known. Current experimental results with sheep exposed to complex waves in enclosures are presented and compared to the previously established Friedlander injury prediction curves. While there appears to be agreement between Friedlander and some complex waveforms, the wide variety of complex wave environments dictates caution at this time.

REVIEW OF GENERAL EFFECTS OF ENVIRONMENTAL NOISE WITH SPECIFIC
REFERENCE TO ANNOYANCE, SLEEP AND WELLBEING.

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The effects on sleep and wellbeing are probably the most serious general effects of environmental noise. Other effects of noise are general annoyance reactions, effects on communication, work performance as well as on rest and relaxation.

These different effects of noise are related not only to different factors in the noise exposure, such as level, duration and type of source, but also to different individual factors. One such important individual factor is noise sensitivity as defined by the individual her/himself.

In the paper different studies in laboratory and field on effects of road traffic noise on sleep, general annoyance and wellbeing are described more in detail. The importance of subjective noise sensitivity for different effects of noise will be discussed.

ABSTRACTS FOR POSTERS

SOLVENT EXPOSURE CONTRIBUTES TO LOSS OF AUDITORY SENSITIVITY.

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Toluene and several other widely used solvents have been shown to cause a loss of auditory sensitivity in animal studies. The hearing loss in humans occupationally exposed to noise has been reported to be aggravated by simultaneous exposure to solvents (e.g. Barregård & Axelsson 1984) and an interaction between toluene and noise has been shown in printers exposed to this combination (Morata et.al. 1993).

The aim of the present series of investigations was to study auditory sensitivity in rats after exposure to toluene together with other agents, such as noise, n-hexane, ethanol, and acetyl salicylic acid (ASA). Exposure to the solvents xylene and/or n-hexane was also investigated.

Rats were exposed to 1000 ppm toluene, xylene, or n-hexane by inhalation for 16-21 h/d during 2-8 weeks. Ethanol was administered in the drinking water (8%) simultaneous with the toluene exposure. The rats were exposed to noise (100 dB L_{eq} 10 h/d for 4 weeks) before or after the toluene exposure. ASA was administered by gavage (100 mg/kg twice daily during 10 days) simultaneously with exposure to toluene. The effect on auditory sensitivity was measured by frequency specific auditory brainstem response (ABR) between 1.6 and 20 kHz or by click ABR.

The results show that combined exposure to noise and toluene resulted in a synergistic permanent loss of auditory sensitivity, when the toluene exposure preceded the noise exposure whereas the reversed exposure order, i.e. noise before toluene exposure resulted in an addition of the damage. Combination of toluene with a reversible ototoxic drug, ASA, also resulted in a synergistic permanent loss of auditory sensitivity as did the combined exposure to two solvents, toluene and n-hexane, and xylene and n-hexane. In contrast, the exposure to ethanol slightly attenuated the ototoxicity induced by toluene.

In conclusion solvent exposure may enhance the loss of auditory sensitivity caused by both noise exposure and exposure to other ototoxic agents.

References:

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ATTENUATION OF ACTIVE NOISE REDUCING HEADSET AT HIGH SOUND PRESSURE LEVELS

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Active headsets, which combine electronic noise reduction with conventional passive attenuation in circumaural hearing protectors, are rapidly being developed and manufactured commercially. These devices combine the potential of an earmuff for reducing sound pressures at frequencies above approximately 1 kHz, with that of an electroacoustic control system to attenuate noise within the ear cup at lower frequencies.

The purpose of this study was to measure the attenuation of a typical commercial device at high sound pressure levels and low frequencies, to explore the performance of the active noise reduction (ANR) system. In this headset, the ANR system consists basically of a miniature microphone, to detect the sound pressure in the ear cup, and an analogue amplifier and filter, to drive an earphone. The attenuation of the headset was measured in two experiments. In the first, an ear cup was mounted on an ear "simulator" in a small enclosure: in the second, a complete headset was worn by human subjects in a controlled sound field.

The enclosure consisted of a 20 cm diameter aluminium tube, 20 cm in length, to which a massive, flat base plate had been attached at one end, and a loudspeaker at the other. Sound pressure levels of up to 140 dB (re 2×10^{-5} Pa) could be generated at frequencies up to 100 Hz, 130 dB up to 300 Hz, and 120 dB up to 1000 Hz. The cushion of the ear cup was sealed against the base plate using a force characteristic of the headband force. The sound pressure within the cavity formed by the cup and the base plate was recorded by a microphone flush-mounted in the latter. The attenuation of the ear cup was determined from measurements of the sound pressure within this cavity, and the sound pressure in the enclosure (outside the cup). The latter was measured using a probe microphone.

The experiments involving human subjects were conducted with four multiple-element sound sources in an anechoic chamber. The sources were configured to produce a diffuse field in the horizontal plane at the centre-head position in the absence of the subject, within the specification of ASCC 61/103N(3). The maximum overall sound pressure level generated with pink noise at the centre-head position was 105 dB (noise bandwidth 31.5 - 16 000 Hz). The insertion loss of the headset was determined by a miniature microphone mounted in the cavum of the concha, from measurements of sound pressure when the headset was worn and removed. The attenuation of the headset was determined using three subjects, and from two to four replications of the measurement procedure over a period of several days.

The mean attenuation of the headset, derived from the measurements on human subjects, differed from the results obtained on an ear cup by no more than 2 dB at frequencies from 31.5 to 500 Hz, and 3 dB from 500 to 1000 Hz. At a sound pressure level of 105 dB, active attenuation of up to 17 dB was obtained at frequencies below 315 Hz. At frequencies from 315 to 800 Hz, amplification of up to 6 dB was observed in the ear cup when mounted within the enclosure, and up to 3 dB when the headset was worn by human subjects. It should be noted that amplification introduced by the ANR system degrades the performance of the headset, so that the total attenuation is less than that obtained with passive attenuation.

In view of the similarity between the results obtained in the enclosure and with human subjects, the performance of the headset at high sound pressures was derived from measurements conducted solely using the former. The attenuation introduced by the ANR system was measured using frequency swept pure tones with sound pressures levels from 90 to 140 dB. The active attenuation recorded at levels less than 120 dB was independent of sound pressure. At higher sound pressures, the active attenuation disappeared. This self muting of the active system, which is presumably used to protect components within the device, occurred abruptly and was usually complete within 2-3 dB of onset. Muting continued for several seconds after termination of the pure tone responsible for its onset. The sound pressure level outside the headset at which self muting occurred was dependent on frequency, and was approximately 120 dB at 100 Hz, and 130 dB at 20 and 200 Hz.

In conclusion, while ANR systems can clearly increase the attenuation of circumaural hearing protectors at low frequencies, their performance characteristics must be carefully selected for the noise environment in which they are to function. The device employed in these experiments was designed for use in helicopters. In this environment the sound pressure is unlikely to provoke self muting of the active elements in the system, except perhaps at infrasound frequencies.

[Work done in collaboration with the Defence and Civil Institute of Environmental Medicine, Toronto, Ont.]

Modelling the effect of occupational noise exposure on hearing thresholds in populations: A new statistical approach.

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The implications for health policy and industrial insurance practice might be much greater if there were robust effects of noise immission at 'low levels' (eg 80 dB(A) Leq for a 50 year equivalent exposure) rather than at higher levels (eg 90 dB). Previous attempts to demonstrate this (eg Robinson, 1987; Lutman and Spencer, 1990) have not been successful.

Many factors make this task very difficult from a statistical point of view including the non-orthogonality of noise immission and age, the non-normal distribution of hearing thresholds and the substantial variation of the variability of hearing thresholds as a function of age and noise immission. The first of these factors is a nuisance that limits interpretation, particularly in identifying the effect of noise in those 60 years of age and older, where normal ageing factors may cloud the issue. However, using new statistical techniques it becomes possible to overcome the problems posed by the remaining two factors.

We have transformed the hearing thresholds to obtain a lognormal distribution with three parameters; an offset factor that is the minimum of the distribution, as well as a mean and SD of the lognormal. Using an iterative method we have simultaneously estimated linear models for the three parameters. These models show that the effect of 80 dBA noise immission is significant on **both** the mean and variance parameters of the distribution. At the average threshold of 1,2 and 3 kHz this implies a robust 3-5 dB increase in thresholds, with a proportionate increase in variance. This method of analysis appears to be more sensitive to the influence of factors that may affect hearing impairments than any other. Its benefits and drawbacks will be discussed in detail.

EFFECTS OF NOISE AND EXERCISE ON DISTORTION PRODUCT OTOACOUSTIC EMISSIONS

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The present study was designed to evaluate whether physical exercise modifies the temporary effects of noise on human cochlear function. Test subjects were 8 normal hearing volunteers in good physical shape aged 25-34 years. Békésy audiometry, tympanometry and distortion product otoacoustic emissions (DPOAE) were measured before and after noise exposure, before and after a combination of noise exposure and exercise at 60% of maximal oxygen uptake (VO_{2max}) and before and after exercise without any noise exposure. The noise exposure, which lasted for 10 minutes, was a narrow band noise of 102 dB SPL centred at 2000 Hz. The temporary threshold shift (TTS) defined as the change in the Békésy threshold at 3 kHz was greater compared to the reduction of the DPOAE-level averaged in the 2-4 kHz octave. There was no effect of physical exercise without noise exposure on either the TTS or the DPOAE-level. However, physical exercise significantly increased the noise-induced TTS and the noise effect on the DPOAE-level.

**TRANSIENT EVOKED OTOACOUSTIC EMISSIONS AND EARLY
DIAGNOSIS OF NOISE-INDUCED HEARING LOSS IN MILITARY
SERVICE**

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The present study was designed to compare the sensitivity and specificity of transient evoked otoacoustic emissions (TEOAE) and pure-tone audiometry in the detection of noise-induced hearing loss. Further, the sensitivity to detect any possible permanent changes of cochlear function after a period of noise exposure, was compared for the two methods. TEOAEs, pure-tone audiometry and tympanometry were recorded in 95 male recruits for obligatory military service before 2-3 months of gunfire exposure at the school for military training. 61 of these recruits were retested with the same test paradigm after their training period. By using pure-tone audiometry as a "gold standard", the sensitivity of different parameters of the TEOAEs was analysed for detecting hearing losses greater than 30 dB. Generally, the method was best for detecting losses in the low and middle frequency ranges. When detecting hearing losses in the 3-6 kHz frequency range, the amplitude of the TEOAEs in different frequency bands combined with the reproducibility and amplitude of the non-filtered TEOAE-response, gave the highest sensitivity. Results from the retest show no significant change in the pure-tone audiogram and there was no systematic change in middle ear pressure. There was seen a significant reduction of TEOAE amplitude in the unfiltered response and in filtered bands of 1, 2 and 3 kHz. Recording variables such as noise and probe position are known to affect the TEOAEs. There was no significant change in stimulus parameters, but the noise-level was significantly reduced reflecting a more quiet second test situation. There was a good test-retest correlation in TEOAE-amplitude between the two test-sessions with correlation coefficient of 0.94 and a standard deviation of 1.1 dB.

In conclusion, measuring the TEOAEs is less time consuming and offers more objectivity than pure-tone audiometry, although the sensitivity of the test has to be improved in order to be used as a screening procedure for detecting noise-induced hearing loss. The sensitivity of the TEOAE method seems to be better for detecting small changes in cochlear function compared to pure-tone audiometry performed under the conditions normally found when testing recruits.



Attenuation of impulsive sound by ear muffs as a function of sound incidence

Björn Hagerman

We have measured the attenuation of ear muffs as a function of the incidence of the sound in our anechoic chamber.

Since the highest levels are expected to occur at the same side as the sound source, only this part was covered. Our investigations are restricted to linear assumptions at relatively low levels without risk of hearing damages. Any nonlinearities will probably be compressive, meaning that we will get the worst cases when we then extrapolate the results to 140 dB. The measurements were carried out in our anechoic chamber.

METHOD

We used the probe microphone method developed at TNO, Soesterberg.

Signal type:	Periodic, with low crest factor
Hearing protectors:	3 muffs: Peltor H7A, Bilsom Viking 2421, Wilson 358A.
Subjects:	16 normal-hearing
Incidences:	9: 0°, +15°, +30°, +45°, +60°, +75°, +90°, +180°, 0° (retest)

From the measurement results the impulse response of the transfer function was calculated with and without protector. These were then convoluted with different impulsive sounds in order to obtain the attenuation of the peak values for these specific sounds. We used four different impulses which we got recorded on DAT-tape from the Salford-group.

Some preliminary results are shown in the two figures. For all muffs the lowest attenuation in the 1-2 kHz region was obtained from behind (180°). Frequency analyses of the impulses to be made.

We have also performed measurements according to the subjective method (ISO 4869-1) and with the MIRE-method in the same sound field. From all these measurements it might be possible to predict the (lowest) attenuation of impulsive sounds from the traditional subjective attenuation values. These analyses are to be made.

**LATENCIES OF THE TRANSIENT EVOKED OTOACOUSTIC EMISSION
IN ANALYSIS OF THE NOISE INJURIES**

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We have compared transient evoked otoacoustic emissions and audiometric thresholds among different types of injured ears. The latency of otoacoustic emissions from frequency bands is estimated using multiresolution time-frequency analysis. In comparison made between the audiometric thresholds and the otoacoustic emissions the latencies give essential information. The analysis is based on the fact that the location in the basilar membrane changes with the sound intensity and thus also the delay of the otoacoustic emission changes whereas the corresponding frequency remains constant. In order to obtain an increased clarity, we developed a new type of graphical and numerical analysis presentation for clinical use.

TESTING DISTORTION-PRODUCT OTOACOUSTIC EMISSIONS IN THE SWISS
HEARING CONSERVATION PROGRAM (SUVA)

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Distortion-product otoacoustic emissions (DPOAEs) are reduced in amplitude when humans or animals are exposed to noise either briefly or over longer time periods. Clinically, DPOAEs are measured for the identification of hearing loss. Therefore, their measurement in employees exposed to noise in the workplace could be an efficient way to monitor the effects of noise and to identify hearing loss. To investigate this possibility, a cooperative project between the University of Basel and the Swiss Hearing Conservation Program (SUVA) has been instituted. The purpose is to determine the feasibility of incorporating tests of DPOAEs into the existing hearing screening program. In the initial phase, a commercially available test instrument was installed in one test station of a SUVA mobile van. Distortion-product otoacoustic emissions were tested bilaterally at frequencies (F_2) of approximately 0.5, 1.0, 2.0, 3.0, 4.0 and 6.0 kHz with L_1 at 65 dB SPL and L_2 varying from 65 to 41 dB SPL in 6-dB steps. Trial testing was performed by an experienced examiner for 7 days during a 2-month period in three separate test locations. One-hundred twelve employees were tested and additional repeated measures were performed on selected technicians working in the mobile test van. Employees were cooperative and accepted the procedure without difficulty. Of the 112 employees entering the test room, complete results were obtained on 95. Three employees had occluding cerumen in one ear and the remaining test failures were due to problems with instrumentation. Results were grouped on the basis of audiometric configuration into three broad categories: Normal hearing, hearing loss above 2 kHz, hearing loss above .5 kHz. There were differences in mean DPOAE amplitudes between the three groups. Experience in this trial phase has lead us to several conclusions: (1) Instrumentation is a critical factor in assuring success of DPOAE measurements in this environment, (2) The procedure is fast, well accepted by employees and can be integrated easily into pre-existing audiometric procedures, (3) With improvements in hardware and software, DPOAE measurements will be useful as an addition to current methods used in hearing conservation programs.

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THE EFFECTS OF MILITARY NOISE ON HEARING

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Noise-induced hearing loss is the single largest category of disability seen after military service. In the British Army there is a 28% prevalence of acoustic trauma among serving infantry personnel, despite the Hearing Conservation Programme initiated in 1965. Recruit training suffers a high wastage from this cause.

There is a requirement for good hearing for communication by radio with high background noise, often in multinational settings. Misinterpretation of orders can be disastrous.

Weapons continue to be noisy, and the greater use of armoured vehicles adds to the problem. Tactical requirements often make it difficult for adequate hearing protection to be worn.

The problem is tackled in three ways: prevention by means of protective devices and better design of equipment, early diagnosis which may be linked to career changes and treatment of any deafness which is sustained.

DIAGNOSTIC VALUE OF EVOKED- AND DISTORTION PRODUCT OTOACOUSTIC EMISSIONS IN OCCUPATIONAL NOISE-INDUCED HEARING LOSS

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Otoacoustic emissions (OEs) provides a noninvasive assessment of the functional condition of preneural, micromechanical mechanisms of the cochlea, which is of great importance in noise-induced cochlear damage. The clinical applications of OEs in occupational noise-induced hearing loss are still to be defined.

The purpose of this study was to compare click-evoked otoacoustic emissions (e-OAE) and distortion-product otoacoustic emissions (DPOE) in respect to pure-tone audiograms in the population of a weaving-mill factory workers. The study was conducted in 82 ears of 41 persons (20 women and 21 men), aged from 21 to 58 years (mean 44.9 ± 8.5), who has been working in noisy environment for 0.5 to 40 years (mean 22.9 ± 9.0).

We found, that e-OAE were never present when the hearing loss for 1 kHz was above 30 dB HL, and the mean hearing threshold for 1, 2 and 4 kHz was above 45 dB HL. The greater high-frequency hearing loss was present, the narrower e-OAE spectrum in high frequency-bands was found. DPOE were never present when the hearing loss was above 40 dB, 45 dB and 55 dB HL respectively for 1, 2 and 4 kHz. DP-grams show usually similar pattern to pure-tone audiometric curves; frequencies around 3-4 kHz were firstly affected. However, no simple relation between severity of cochlear hearing loss and intensity of otoacoustic emissions was revealed. The best linear correlation was found between pure-tone audiometric thresholds and DPOE amplitudes for the frequency 4 kHz ($r=0.6$, $p<0.001$). There was poor correlation between audiometric thresholds and DPOE detection thresholds.

We concluded that, because of a narrow dynamic range and a great inter-subject variability, otoacoustic emissions measurements have limited value in reconstruction of a pure-tone audiogram, comparing i.e. with frequency specific auditory brainstem audiometry. Instead, otoacoustic emissions can be used as a screening test in malingers applying for occupational noise-induced hearing loss.

**EFFECTIVENESS OF A HEARING CONSERVATION PROGRAM:
A NEW SIMPLE METHOD**

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Noise induced hearing loss (NIHL) is the most prevalent professional disease. This has led to the establishment of hearing conservation programs (HCP) in industry in order to reduce the incidence and severity of this disease. However, there are no easy ways to evaluate the effectiveness of an existing HCP. Based on our experience as director of the HCP for the Quebec Metal Mining Association in the Province of Quebec, we have developed an easy way to use an approach which with the use of a single chart can allow to determine if an HCP is efficient.

TEMPORARY THRESHOLD SHIFT DOES NOT PREDICT PERMANENT THRESHOLD SHIFT IN A "LOW-FREQUENCY" EXPOSURE OF MICE.

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Occupational safety and health laws generally do not differentiate between low- and high-frequency noises. The A-weighted filter function on sound level meters and dosimeters emphasizes the mid-frequencies of the human auditory range. This frequency region has the highest audiometric sensitivity and also is the area most vulnerable to noise. The purpose of this report is to present an observation of a low-frequency noise exposure in two strains of mice.

Hearing tests consisted of ABR thresholds to clicks and tone pips (8, 16 and 32 kHz) before and a number of times after noise exposure.

Two strains of mice, one strain genetically susceptible to noise-induced hearing loss (C57BL/6J), the other not (CBA/J*C57BL/J), were exposed to a 110 dB broadband noise (500 Hz to 16 kHz with maximum level at 4 kHz) for one or two hours. This exposure produced profound temporary threshold shift (TTS) immediately after exposure which resolved in both strains to no measurable permanent threshold shift (PTS). The exposure was repeated on the same animals with the same result.

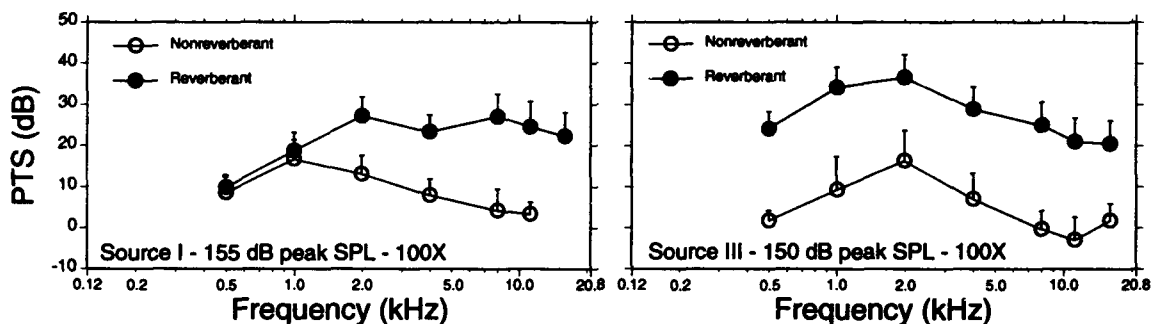
A second set of mice from the same two strains were exposed to a second 110 dB broadband noise with a higher frequency spectrum (4 kHz to 31.5 kHz with maximum level at 16 kHz) for 1 or 2 hours. This produced TTS equivalent to the previous exposure, but this time the noise-susceptible mice showed massive PTS of 40 dB. The non-susceptible mice returned to normal hearing levels.

Conclusions: The magnitude of the temporary threshold shift did not predict the magnitude of a permanent threshold shift. In both strains, the temporary threshold shift to this low-frequency noise band was significant. Yet neither strain showed any hearing loss. Only in the case of a noise band placed within the sensitive part of the mouse audiogram was permanent threshold shift noted. In summary, the low-frequency and high-frequency noise stimuli produced comparable TTS in both strains of mice, but only the high-frequency noise produced PTS in the susceptible C57BL/6J mice.

THE EFFECT OF REVERBERANT AND NONREVERBERANT BLAST WAVES ON HEARING.

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The effects of exposure to high levels of reverberant impulse noise on hearing were studied in the chinchilla. Blast waves were generated by discharging shock tubes in a reverberant or nonreverberant environment. Two different sources produced wave signatures having different spectral distributions of energy. Exposures took place at 150, 155 or 160 dB peak SPL with either 1, 10, or 100 impulse presentations having repetition rates of one impulse per minute. Auditory evoked potentials were used to estimate permanent threshold shifts. A total of 255 animals were used in these experiments. The audiometric data show that there is a systematic increase in hearing loss as the energy of the exposure is increased through manipulation of number of presentations, peak SPL, or reverberation. The effect of a reverberant environment is to exacerbate permanent hearing loss as much as 25 dB relative to the nonreverberant environment. [Research sponsored by USAARL Contract DAMD17-91-C-1113.]



Mean permanent threshold shift for the groups exposed to 100 impulses in a reverberant or nonreverberant environment from (a) Source I at 155 dB peak SPL and (b) Source III at 150 dB peak SPL. Error bars represent the standard errors of the mean.

Modeling categorical loudness perception in normal and hearing impaired listeners.

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Abstract

The summation of perceived categorical loudness across critical bands in normal and hearing impaired listeners was measured as a basis for a loudness model. Loudness scaling experiments were performed using different stimulus configurations (narrow-band stimuli, broadband stimuli and combinations of narrowband stimuli separated in frequency). The perceived loudness was evaluated using a categorical scale consisting of 11 different categories. The results of these experiments were compared to the predictions of a model which basically adapts Zwicker's loudness model to categorical loudness perception. To account for hearing impairment this model was modified in two aspects: First, elevated absolute threshold was accounted for by adding an internal noise. The second modification was to broaden the auditory filters. The experimental data for the normal hearing subjects clearly show effects of stimulus bandwidth and frequency separation of combined stimuli on perceived loudness. The modified Zwicker model describes this data very well. For hearing impaired subjects the data show much less influence of bandwidth and frequency separation on perceived loudness. Hence, the parameters of an individually fitted loudness model for each hearing impaired listener can only be estimated with a limited accuracy.

**APPLICATION OF EVOKED RESPONSE AUDIOMETRY (ERA) IN
DIAGNOSIS OF OCCUPATIONAL DEAFNESS**

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A commonly known fact is that diagnosis of occupational noise-induced hearing loss in compensation cases is a difficult task because it is circumstantial and ultimately made by exclusion of other non-occupational causes of deafness.

In our own series of more than 1600 industrial compensation claims in about 30% of cases, the other reasons of hearing loss e.g. otosclerosis, otitis media. Meniere's disease, as well as malingering were recognized.

The aim of this study is share with our recent experience with diagnostic strategies, using the objective methods of audiological assessment to establish the site of lesion and to quantify the hearing thresholds.

The application of different evoked reponse audiometry techniques, namely cortical middle and late responses, and brainstem responses, combined with impedance measurements is discussed, considering benefits and limitations of the several tests in compensation evaluations.

Finally, the best in our hands procedure of examination, based on the frequency-specific brainstem evoked responses audiometry is summarized, and illustrated by typical cases from our medicolegal work.

SCHOOL SURVEYS OF EVOKED OTOACOUSTIC EMISSIONS ON SUSCEPTIBILITY TO NIHL

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It is a matter of common knowledge that there are large individual differences in susceptibility to NIHL. Our previous studies (Tanaka et al., 1988 and 1990) reported that evoked otoacoustic emissions with prolonged duration (continuous EOAEs) were frequently seen in ears with dip type hearing loss and even in the healthy side of ears with unilateral dip type hearing loss. In order to know a relationship between dip type hearing loss and continuous EOAEs, surveys were performed in 2 school brass bands and in a school kendo (Japanese traditional fencing) team.

The surveys of brass band members revealed that incidence of micro-dip hearing loss in Békésy's audiogram was 82% with ϕ coefficient of 0.48 in 50 continuous EOAE ears of H-school and it was 81% with $\phi = 0.39$ in 36 continuous EOAE ears of F-school. The follow-up study of 32 ears in H-school revealed that 4 ears changed in micro-dip hearing loss but not in continuous EOAEs. The kendo team of D-school showed the incidence of micro-dip was 96% in 24 continuous EOAE ears, with ϕ being 0.49. These results demonstrated the presence of the relationship between dip type hearing loss and continuous EOAEs. It seems probable that the continuous EOAEs express an inner ear vulnerability to noise exposure. The EOAE measurement may be a valuable tool in predicting individual susceptibility to NIHL.