Considerations on assessing the risk of work-related hearing loss

Donald Henderson[1], Thais C. Morata[2] and Roger P. Hamernik[3]

[1]*Center for Hearing & Deafness, State University of New York at Buffalo, Buffalo, New York, USA*
[2]*Dept. of Communication Disorders, Universidade Tuiuti do Parana, Curitiba, Parana, Brazil*
[3]*Auditory Research Laboratory, State University of New York at Plattsburgh, Plattsburgh, New York USA*

The large intersubject variability observed in demographic studies of noise-induced hearing loss illustrates how difficult it can be to estimate with precision the risk posed by exposure to noise. One possible source of the variability is the result of evaluating a diverse set of acoustic conditions with a simple metric A-weighted energy. In this paper the limitations of the energy-based criteria are reviewed. The benefits of evaluating a noise exposure in terms of energy and the metrics of frequency and time-domain kurtosis are discussed. A second source of variability in industrial noise studies may be related to non-acoustic factors such as chemical exposures that contribute to hearing loss acquired on the job
Keywords: equal energy, kurtosis, noise, ototoxicity, solvents

Introduction

ISO 1999 and current U.S. noise standards were established using the results of a number of demographic studies done at least 30 years ago (Burns and Robinson, 1970; Passchier-Vermeer, 1983). The original noise standards reflected a reasonable synthesis of prevailing scientific understanding and had merit when they were first advanced. However, the ISO and Department of Labor standards were based on demographic studies that are fundamentally limited by large intersubject variability. In addition, during the 30 years since the original formation of the current noise standards, science has produced important new insights into the relation between a subject's noise exposure and the resultant hearing loss. This paper reviews our current approach to assessing risk from noise and discusses noise metrics as well as the possibility of noise interaction in the workplace.

Assessing Risk from Noise Exposures

The study of weavers by Taylor et al. (1965) dramatically illustrates the problem of variability in demographic studies. Taylor studied metal workers in a setting where the noise exposure was well documented. Individual worker's medical records were available and the noise level in the factory had been relatively stable for years. Taylor et al. (1965) quantified the noise exposure using the noise emission level, an energy measure that is based on daily A-weighted levels and number of years of exposure. At that time the prevailing wisdom was that hearing loss was proportional to the total acoustic energy received by the individual over the course of their work history. In spite of their careful attention to experimental control, the data are plagued with great individual variability (i.e., individuals with nominally the same exposure could have levels of hearing that ranged over 60 dB). Several explanations for this variability can be put forward. Among these are:

(1) It is difficult to accurately estimate an individual's noise exposure history, including both industrial and recreational. (2) Alternatively, a simple energy based prediction is not adequate (i.e., exposures with the same total A-weighted energy may be different in the potential for creating hearing loss. This possibility is suggested by a comparison of data collected by Burns and Robinson (1970) and by Passchier-Vermeer (1974). Both studies used acoustic energy as a measure of noise exposure. Surprisingly, Passchier-Vermeer's (1974) subjects have a higher level of hearing loss for the same energy of the exposure than do those of Burns and Robinson. The differences in degree of hearing loss between these two studies may be related to the nature of the noise exposures. The Burns and Robinson study involved workers exposed to a relatively constant level of noise, but the noise exposure in the Passchier-Vermeer consisted of irregular combinations of continuous and impact noise. The hazards associated with combinations of continuous and impulse noise were identified in an early study by Hamernik and Henderson (1976). They reported that combinations of impulse and continuous noise could dramatically increase the effect of either the continuous or impulse noise. For example, chinchillas exposed to either 150 dB impulses (1 msec A-duration) or continuous noise (4 kHz OB @ 90 dBA) develop small hearing losses and cochlear lesions. However, if the two noises are combined the increase in total energy of the exposure is trivial but the traumatic effects are greatly increased — i.e., more than a simple addition of each effect. While the impulses used in this study are not typically found in industry, the results suggested that non-Gaussian noises (combinations of impulse and continuous noise) may pose an increased risk of hearing loss when compared to Gaussian noises with equivalent energy and spectra. The comparison of the demographic results of Passchier-Vermeer and those of Burns and Robinson and the experimental results of Hamernik and Henderson cast some doubt on the validity of noise standards that are based simply on an energy model and point to the importance of the noise waveform as a factor influencing the extent of hearing loss.

As early as 1980, Bruel reported on the influence of crest factor in NIHL. Additional experimental animal model studies (Roberto et al., 1985; Nilsson et al., 1980; Hamernik et al., 1993; Ahroon et al., 1993) have demonstrated quite clearly that an A-weighted Leq is not a sufficient measure of hazard associated with non-Gaussian noise environments. Demographic data (Passchier-Vermeer, 1983; Sulkowski et al., 1983; Taylor et al., 1984; Thiery, 1987; Thiery and Meyer-Bisch, 1988) show that non-Gaussian noise exposures are more hazardous to hearing than are Gaussian noises of similar Leq. Our own recent animal studies (Hamernik et al., 1993; Lei et al., 1994) have shown that noises that have the same Leq and the same spectra but that differ considerably in their temporal structure produce very different pattern and severity of hearing loss. Histological data confirm this result. It should be noted that unlike the stimuli used by Hamernik and Henderson (1976) the noises synthesized by Lei et al. (1994) were typical of many industrial noise environments. Examples are shown in Figure 1. The highest peak levels of the impacts did not exceed 126 dB. Other non-Gaussian stimuli were composed of band limited Gaussian noise bursts in combination with broad band Gaussian noise to construct non-Gaussian stimuli. Collectively, the experiments were clear in showing that an understanding of the temporal structure of a noise exposure is critical to predicting its hazard to hearing and, as a corollary, that time-averaged metrics such as energy are not adequate for estimating the hazard to hearing from industrial/military noise environments. These environments are usually very complex (i.e., in the most general case, non-stationary stochastic signals) and require, in addition to energy,

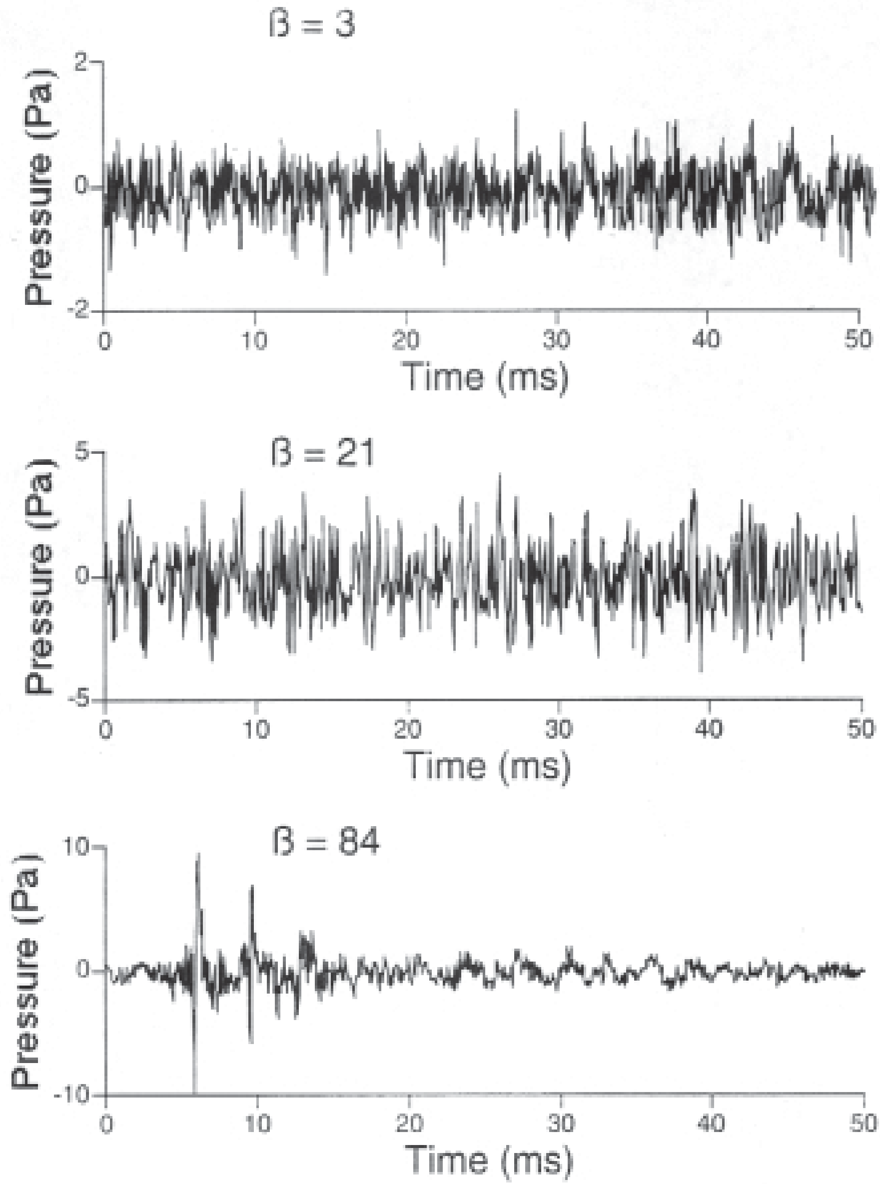


Figure 1. Waveforms of three noises, each with same spectrum and average power but differing in time-domain kurtosis value

metrics that quantify the specific temporal and peak properties of a noise exposure in order to estimate the risk to hearing.

Unfortunately, while (A-weighted) energy is not sufficient to characterize a noise exposure for the purpose of hearing loss prevention there is, at present, no other generally accepted metric or combination of metrics to be used as an alternative. Twenty years ago when the demographic data incorporated into ISO-1999 were assembled, energy metrics were the only measures that could readily be extracted from a noise environment with conventional noise survey equipment. Thus, by default, energy became the metric for estimating the auditory consequences of a noise exposure. The accumulating experimental data provide evidence that the enormous variability seen in the hearing loss data that were incorporated into the ISO-1999 standard (Mills et al., 1996) may be, in part, the result of organizing the diverse exposure conditions to which the subject population was exposed around the single descriptive metric; energy.

Alternative Approach to Noise Analysis

A review of experimental and demographic studies shows that an energy based model of hearing loss prevention may be suitable for Gaussian noise but an alternate approach to noise analysis is needed for statistically more complicated exposures. On the basis of preliminary experimental data, we suggest that an energy metric in combination with the statistical metrics of frequency- and time-domain kurtosis and the joint peak-interval histogram may provide necessary (and possibly sufficient) information to evaluate any industrial noise environment's potential for causing hearing loss.

Kurtosis – A Critical Parameter for NIHL?

The first moment of a distribution is the arithmetic mean; the second central moment is the variance of the distribution; the third standard moment is the skew of the distribution; the fourth standard moment, defined below, is referred to as the kurtosis of the distribution. A distribution may be symmetric but still deviate from normality. Kurtosis is a metric used to quantify the departure from normality. The probability density distribution of a random variable may be flatter (have thinner tails) or more peaked (have fatter tails) than normal. The former is referred to as platykurtic the latter as leptokurtic. The normal distribution is also referred to as mesokurtic. Note that the standard moments incorporate the square root of the variance. The kurtosis of the normal distribution is 3, while distributions that are more or less outlier prone have kurtosis values that are greater or less than 3 respectively. Frequency domain kurtosis is essentially the time domain kurtosis computed on consecutive frequency bands of the non-Gaussian signal. The width of the bands on which β is calculated can be variable. The use of this metric was first introduced by Dwyer (1984).

Kurtosis is defined as the ratio of the fourth-order central moment to the squared second-order central moment of the sample distribution. The kurtosis of a non-Gaussian stochastic signal can be used to measure or estimate the "peakedness" of the amplitude distribution of noise exposure. Erdreich (1986), without the backing of direct experimental data, suggested that the kurtosis of the amplitude distribution could be used as a metric to evaluate the effects of the temporal peak distribution properties of a noise exposure on hearing.

Lei et al. (1994) developed the following working hypothesis: for the same total energy and spectrum a high-kurtosis noise exposure is more hazardous to hearing than a Gaussian noise exposure, and that this effect is frequency dependent.

Using a very limited set of exposure parameters, they found that the kurtosis (statistic) metrics in both the time, $\beta(t)$, and frequency domains, $\beta(f)$, were shown to: (1) rank order the level of hearing trauma and (2) reflect the frequency specificity of trauma. Figure 1 shows the waveform for three of the noises used in their experiments. It should be noted that each noise had the same total acoustical energy and long-term spectrum. Figures 2 and 3, taken from the Lei et al. (1994) paper, illustrates the relations that were found between the effects of non-Gaussian noises and the metrics. In this set of experiments five different noise exposures were used. All exposures had the same total energy and energy spectrum. Each exposure lasted for the same length of time. They differed only in their temporal and peak properties, producing

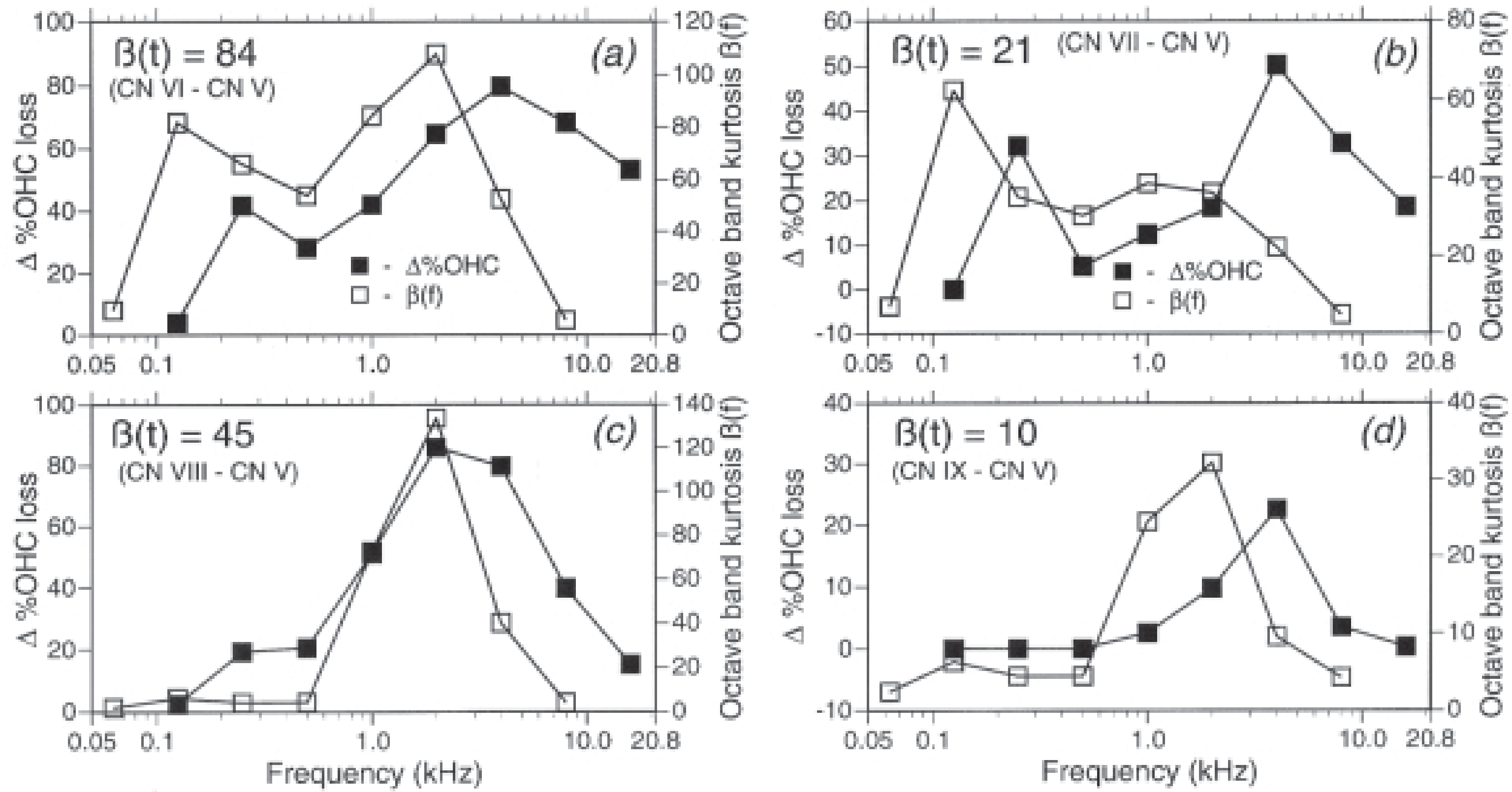


Figure 2. A comparison of the β(f) spectrum and the difference between the OHC loss produced by the indicated noise exposure and the β(t) = 3 noise exposure. (From Lei et al., 1994.)

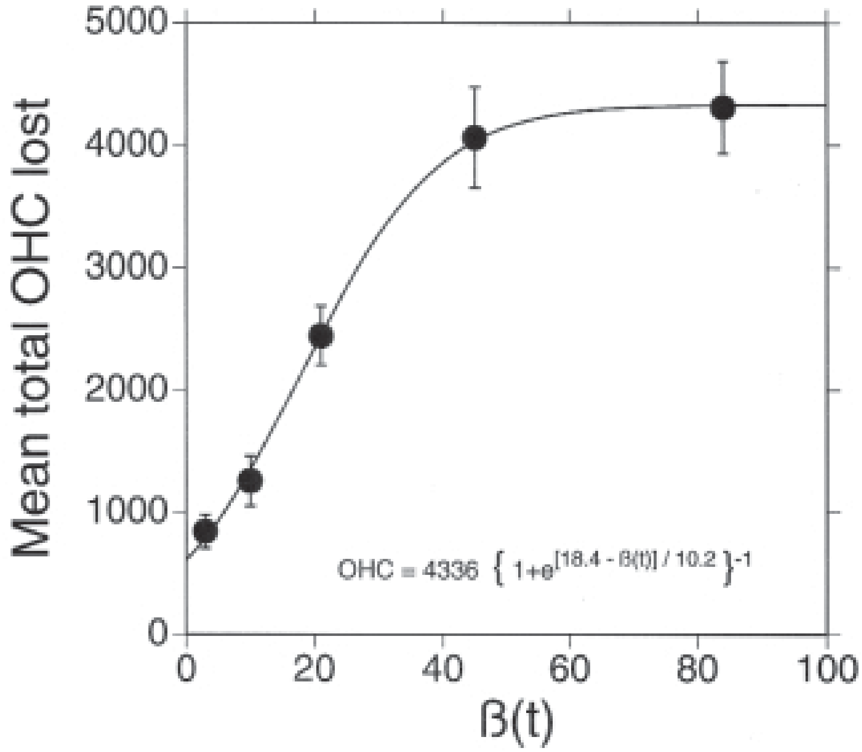


Figure 3. A comparison of the β(t) spectrum and the total OHC loss. (From Lei et al., 1994.)

differences in the kurtosis metrics computed on both the time and frequency domain signal**.** Both band limited impacts or noise bursts superimposed on a background Gaussian signal produced variable values of kurtosis in both the time and frequency domains. Figure 2 shows the pattern of hair cell loss along the basilar membrane and the β(f) distribution. Figure 3 shows the β(t) statistic and how it relates to the group average total OHC loss.

These figures clearly show that there are good correlations between both frequency domain kurtosis and the permanent effects of a nonGaussian noise exposure. Especially impressive is the close relation between β(f) and the profile of outer hair cell loss shown in Figure 2 where the difference between the OHC loss measured in the Gaussian and each of the four non-Gaussian conditions is presented as a function of octave band center frequency at which cell loss and β(f) were measured. Figure 3 shows the relation between β(t) and the total OHC loss in each of the five exposure conditions. β(t) clearly rank orders the OHC loss. Note also, that the Gaussian condition, β(t)=3, produced less than a mean total of 1,000 lost OHC while the same energy and spectrum β(t)=84 condition produced a mean of over 4,000 missing OHCs.

The results presented in the Lei et al. (1994) paper are a clear indication that in addition to energy, temporal and peak variables are important determinants of hearing loss. These results showed that β(t) and β(f), the time- and frequency-domain kurtosis statistic respectively, are highly correlated with trauma.

The application of kurtosis based metrics to predict hearing loss from exposure to noise needs to be studied more thoroughly, and under a wider range of acoustic parameters. However, the strength of the initial data suggests that metrics for evaluating the risk associated with an exposure should be modified to include not only the total energy of the exposure but also how the energy is distributed over time. This proposal can be reasonably approached at this time because current noise measurement instruments could be modified to also measure the statistical properties of a noise.

Non-Acoustic Interactions in Workplace

Prevention of hearing loss typically has meant controlling subject's noise exposure and in industrial settings, occupational hearing loss was considered to be synonymous with NIHL. However, new information has shown that certain non-acoustic factors in the work place may directly affect hearing, or interact with noise, and are considered possible contributors to the individual variability in susceptibility to noise-induced hearing loss (Franks, Davis & Krieg, 1989; Phaneuf & Hétu, 1990; Morata, Franks & Dunn, 1994). Besides noise, the contribution of vibration, extreme temperatures and chemicals has been examined in the occupational arena. Ototoxic effects have been reported after exposure to metals, solvents and asphyxiants (for reviews see Franks and Morata, 1996; Johnson and Nylon, 1995). If exposure to some of these chemicals occur in sufficiently high concentrations, hearing may be affected despite lack of exposure to excessive noise (Morris, 1969; Nikolov, 1974; Bencko & Symon, 1977; Barregård & Axelsson, 1984; Schwartz & Otto, 1987; Mizukoshi et al., 1989; Jacobsen et al., 1993; Discalzi et al., 1993; Farahat et al., 1997; Morata et al., 1993, 1997a, 1997b). These findings have serious implications to hearing loss prevention in the workplace. When the current noise standards were originally promulgated, there was scant evidence for chemically induced hearing losses or interaction between noise and industrial chemicals. What is clear today is that most noisy industrial settings can have other potentially dangerous factors for hearing.

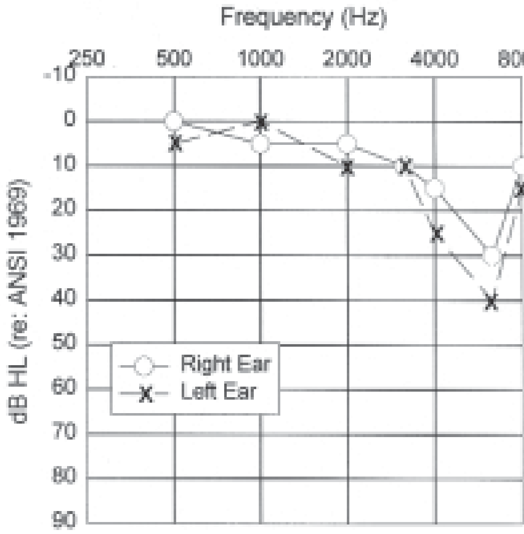
The National Occupational Research Agenda (NORA), result of an inclusive consultation initiative coordinated by the National Institute for Occupational Safety and Health, US, identified multiple exposures as a research priority for the occupational safety and health community (NIOSH, 1996). The risk to hearing posed by combined exposures to noise and chemicals was given as a specific example of issues in need of investigation.

Additionally, in the NIOSH publication "Preventing Occupational Hearing Loss: A Practical Guide" (Franks, Stephenson & Merry, 1996), an argument is presented for broadening the scope of risk assessment and preventive initiatives. It also recommended that the term "occupational hearing loss" should not be a synonym for "noise-induced hearing loss". Moreover, it is advised that hearing loss prevention programs take chemical exposures into account when monitoring for hazards, assessing hearing and controlling exposures.

The American Conference of Governmental Industrial Hygienists in its 1998 Threshold Limited Values and Biological Exposure Indices (TLVs® and BEIs®) publication, went a step beyond, including a note in its Noise Section which states: "In settings where exposure to toluene, lead, manganese or n-butyl alcohol occurs, periodic audiograms are advised and should be carefully reviewed." It also lists other chemicals under investigation. If such recommendation is followed, data should be available in a few years that will allow recommendations to be formulated regarding: ototoxicity exposure limits for chemicals, exposure limits for noise-chemical combined exposures, and hearing loss prevention strategies that are not limited to exposures to excessive noise levels.

A

Date of Birth: Nov, 1965
Date of Test: Nov, 1995

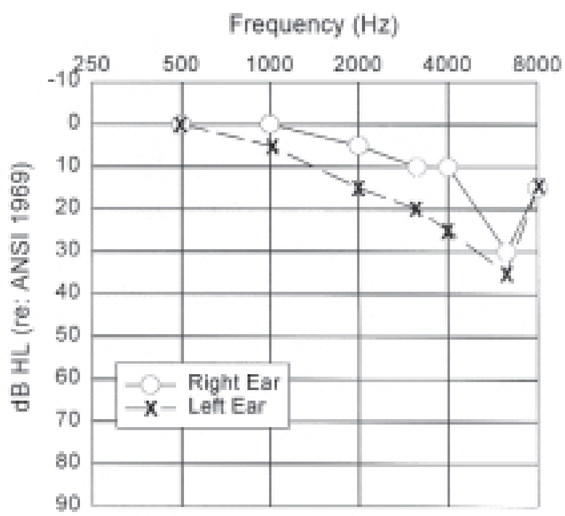


Interrupted Speech: (Korsan-Bengtsen, 1973)	97%

Acoustic Reflexes Probe/Tone	500 Hz	1000Hz
Right/Right	Normal	Normal
Right/Left	Normal	Normal
Right/Right Decay	None	None
Right/Left Decay	None	None
Left/Left	Normal	Normal
Left/Right	Normal	Normal
Left/Left Decay	None	None
Left/Right Decay	None	None

B

Date of Birth: May, 1965
Date of Test: Nov, 1995



Interrupted Speech: (Korsan-Bengtsen, 1973)	78%

Acoustic Reflexes Probe/Tone	500 Hz	1000Hz
Right/Right	Normal	Normal
Right/Left	Normal	Normal
Right/Right Decay	None	None
Right/Left Decay	Present	Present
Left/Left	Normal	Normal
Left/Right	Normal	Normal
Left/Left Decay	Present	Present
Left/Right Decay	None	Present

Figure 4. Examples of audiometric profiles from workers who were either noise exposed (A) or noise plus solvent exposed (B) (from Johnson et al., 1998).

Investigating The Effects Of Industrial Chemicals On Workers' Hearing

The American Conference of Governmental Industrial Hygienists (ACGIH,1998) alluded to the complexity of investigating industrial chemicals effects. The hearing loss from industrial chemicals can be very similar to the hearing loss from ototoxic drugs such as aminoglycosides and cisplatin, as well as to the hearing loss from noise. General descriptors of these disorders are very similar: bilaterally symmetrical, irreversible, high frequency (3 to 6 kHz) sensorineural hearing loss with damage mainly to cochlear hair cells. Comparison of these descriptors reveals how difficult it may be to make a differential diagnosis and to determine causation of hearing loss among workers. It also may explain why the effects of environmental/occupational chemical exposure have been long overlooked. For instance, the effects of occupational exposure to solvents became evident only after the prevalence of mild to moderate high frequency hearing loss among groups with different exposure conditions were compared (Bergström and Nyström, 1986; Morata et al., 1993). A higher prevalence of hearing loss has been associated with combined exposure to noise and solvents compared with exposure to noise alone. Such effect could have been overlooked if only audiometric threshold means were compared (Morata et al., 1993).

Literature on the on the effects of solvents on the human auditory system suggests that hearing loss may have both cochlear and retrocochlear sites (Morata et al., 1993, Ödkvist et al., 1987; Möller et al., 1989). Audiometric findings associated with exposure to solvents revealed mild to moderate hearing losses. However, despite a mild audiometric effect, the hearing loss from solvents may significantly impact the individual's ability to communicate. Consequently, use of audiological tests that evaluate the central auditory system becomes crucial, and may elucidate effects of chemicals from effects from noise. Figure 4 displays two cases from a pilot study by Johnson et al. (1998). It illustrates that noise and solvent exposed workers may have audiometric thresholds that are equivalent to workers exposed exclusively to noise, but performance in other audiological tests can differ. Figure 4a, displays results of a noise- exposed worker, with a mild high frequency audiometric loss, and normal acoustic reflexes and normal results in one sensitized speech test (Interrupted Speech). Results on figure 4b are from a worker exposed to noise and the solvent styrene. Audiometric thresholds are comparable to the noise-exposed worker, but results on other tests are not all normal, and suggest retrocochlear involvement.

Swedish researchers have used comprehensive audiological test batteries to investigate workers exposed to mixtures of solvents in different work settings (Ödkvist et al., 1982, 1987; Möller et al., 1989). The test battery included pure-tone audiometry, speech reception threshold, maximum speech discrimination score, discrimination of interrupted speech, acoustic reflex thresholds and decay, auditory brainstem and cortical response audiometry. The findings of pure-tone audiometry and speech discrimination testing were essentially normal for age and noise exposure history, and did not indicate measurable cochlear damage due to solvent exposure. However, significant abnormalities were found in tests that assessed central portions of the auditory pathways, especially discrimination of interrupted speech and cortical potentials evoked by frequency glides (Ödkvist et al., 1987; Möller et al., 1989).

Analysis of Audiometric Data

The majority of investigations on occupational hearing loss has relied on averaging pure tone thresholds to assess noise effects on auditory function. To investigate the effects of chemical exposure, this traditional approach may not be sufficient, or even appropriate because chemically induced hearing loss may be trivial in terms of threshold shift, but serious in terms of listening, particularly in noise.

Because of the potential for noise to act as a confounder, approaches that are more robust indicators of the risk posed by occupational chemical exposures on hearing include: classification of audiometric results using specific criteria and subsequent estimation of prevalence or incidence rates and relative risk from analyzing hearing as a binary variable (normal vs. high frequency hearing loss). Some examples of these alternatives were discussed previously by Morata and Lemasters (1995). The prevalence of hearing loss between groups with different exposure conditions should be examined even if audiometric thresholds, by themselves, do not allow for easy identification of the effect of chemicals on hearing, and especially when pure-tone audiometry is the only available test.

Other Considerations

Heretofore, few human studies have examined the time necessary for chemical exposures to affect the auditory system, and there is still uncertainty whether it is a chronic or acute process. Investigations that examined solvents effects over time indicated that hearing loss may be observable two to three years earlier than is usually seen with noise exposure (Morata, 1989; Morata et al., 1993). Another study, however, observed a significant effect of solvents only after 5 or more years of exposure (Jacobsen et al., 1993). This issue of latency is certainly dependent on the specific ototoxicant and patterns of exposure, and needs further investigation.

Another issue in need to investigation concerns the concentration of chemicals necessary to affect the auditory system. Recent reports have indicated that levels below current recommended limits for certain solvents might be harmful to the auditory system (Liu and Fechter, 1997; Morata et al., 1997a, b). More research on solvent-induced hearing losses is needed to address the issue of adequacy of recommended limits.

In summary, occupational hearing loss risks need to be redefined to include hazards other than noise. Specifically, there is evidence that chemical exposure alone can lead to auditory dysfunction, and combined exposure with noise may lead to interactive effects. To understand the interactions of noise with chemicals, both field and laboratory research are necessary. Field studies will elucidate risk from exposures to chemicals and noise-chemical combinations. Laboratory studies will establish dose-response relationships, leading to hearing-damage-risk criteria for chemicals and combined exposures.

Conclusion

Industrial hearing loss continues to be a major health problem. There is no question that well managed hearing conservation programs prevent or reduce significant hearing loss in many work situations. Risk assessment is a fundamental step of hearing conservation programs. Currently it relies exclusively on the noise dose measurements (i.e., dBA levels x duration). Evidence available today indicates that risk assessment can be markedly improved with attention to two major factors: (1) statistical variability of the noise distribution, and (2) the possibility for noise/chemical interactions. Given the improvements made in instrumentation, a more precise assessment of risk is a reasonable goal.

Correspondence Address

Donald Henderson,
Center for Hearing & Deafness
State University of New York at Buffalo
215 Parker Hall
Buffalo, New York 14214 USA

References

Agency for Toxic Substances and Disease Registry, ATSDR. (1993). ATSDR/ Division of Health Studies: National Exposure Registry-Trichloroethylene (TCE) Subregistry- Baseline Technical Report- 1993. Washington, DC.

American Conference of Governmental Industrial Hygienists, ACGIH. (1998). Threshold Limit Values and Biological Exposure Indices for 1998-1999, 1998. ACGIH, Cincinnati.

Ahroon, W.A., Hamernik, R.P. and Davis, R.I. (1993). Complex noise exposures: An energy analysis. *J. Acoust. Soc. Am*. 90:997-1006.

Barregård, L., Axelsson, A. (1984). Is there an ototraumatic interaction between noise and solvents? *Scandinavian Audiology*, 13:151-155.

Bencko, V., Symon, K. (1977). Test of environmental exposure to arsenic and hearing changes in exposed children. *Environmental Health Perspectives*, 19:95-101.

Bergström, B., Nyström, B. (1986). Development of hearing loss during long term exposure to occupational noise. *Scandinavian Audiology*, 15:227-234.

Bruel. P.V. (1980). The influence of high crest factor noise on hearing damage. *Scan Audiol. Suppl*. 12, pp. 25-32.

Burns, W. and Robinson, D.W. (1970) An investigation of the effects of occupation noise on hearing. In Sensorineural Hearing Loss, G.E.W. Wolstenholme and J. Knight (eds). Williams and Wilkens, Baltimore.

Discalzi, G.L., Fabbro, D., Meliga, F., Mocellini, A., Capellaro, F. (1993). Effects of occupational exposure to mercury and lead on brainstem auditory evoked potentials. *International Journal of Psychophysiology*, 14:21-25.

Dwyer, R.F. (1984). Use of the kurtosis statistic in the frequency domain as an aid in detecting random signals, IEEE *Journal of Oceanic Engineering*, Vol. OE-9:85-92.

Erdreich, J. (1986). A distribution based definition of impulse noise, *J. Acoust. Soc. Am*.. 79: 990-998.

Farahat, T.M., Abdel-Rasoul, G.M., El-Assy, A.R., Kandil, S.H., Kabil, M.K. (1997). Hearing thresholds of workers in a printing facility. *Environmental Research*, 73:189-192.

Franks, J.R., Davis, R.R., Krieg, E.F., (1989). Analysis of a hearing conservation program database: factors other than workplace noise. *Ear and Hearing*, 10(5):273-280.

Franks JR, Morata TC (1996). Ototoxic effects of chemicals alone or in concert with noise: a review of human studies. In: Axelsson A, Borchgrevink HM, Hamernik RP, Hellström PA, Henderson D, Salvi R, (Eds.), Scientific basis of noise-induced hearing loss. Thieme, New York, 472 pp.

Franks JR, Stephenson MR, Merry CJ.(1996). Preventing Occupational Hearing Loss- A Practical Guide. USDHHS, PHS, CDC, NIOSH publication no.96-110.

Hamernik, R.P., Ahroon, W.A., Hsueh, K.D., Lei, S-F. and Davis, R.I. (1993). Audiometric and histological differences between the effects of continuous and impulsive noise exposures. *J. Acoust. Soc. Am*. 93:2088-2095.

Hamernik, R.P., and Henderson, D. (1976) The potentiation of noise by other ototraumatic agents. In The Effects of Noise on Hearing, D. Henderson, R.P. Hamernik, J.Mills and D.S. Dosanjh (eds). Raven Press, NY.

International Standard ISO 1999 (1990). Acoustics- Determination of occupational noise exposure and estimation of noise-induced hearing impairment. Technical Comm. ISO/TC 43-Acoustics, International Organization for Standardization, Geneva, Switzerland.

Jacobsen, P., Hein, H.O., Suadicani, P., Parving, A., Gyntelberg, F. (1993). Mixed solvent exposure and hearing impairment: an epidemiological study of 3284 men. The Copenhagen male study. *The Journal of Occupational Medicine* 43(4): 180-184.

Johnson A-C, Nylon P.R. (1995) Effects of industrial solvents on hearing. In Morata TC, Dunn DE (Eds), Occupational Hearing Loss, Occupational Medicine: State of the Art Reviews, 10(3):623-640. Hanley and Belfus, Inc., Philadelphia.

Lei, S.-F., Ahroon, W.A., and Hamernik, R.P. (1994). The application of frequency and time domain kurtosis to the assessment of hazardous noise exposures, *J. Acoust. Soc. Am*. 96, 1435-1444.

Lei, S.-F, Hamernik, R.P. (1995) Wavelet Transform and Frequency Domain Kurtosis: Application to Assessment of Hearing Hazard from Noise Exposure. Proceedings of 1995 IEEE ASSP workshop on Applications of Signal Processing to Audio and Acoustics. pp.1.4.1-3, New Paltz, NY, Oct. 1995. IEEE publishers, NJ.

Lei, S.-F. and Hamernik, R.P. (1996). Extraction of frequency domain kurtosis from non-Gaussian and nonstationary signals using the wavelet transform. International Symposium on Optical Science, Engineering and Instrumentation: Wavelet Applications in signal and image processing. Proc. SPIE 2825:785-795.

Lei, S.-F. and Hamernik, R.P. (1998). Construction of a joint peak-interval histogram using higher-order cumulant based inverse filtering. Paper submitted to the 1998 International Converence on Acoustics, Speech and Signal Processing, Seattle, WA, May 12-15.

Liu, Y., Fechter, L.D. (1997) Toluene disrupts outer hair cell morphometry and intracellular calcium homeostasis in cochlear cells of guinea pigs. *Toxicology and Applied Pharmacology*, 142(2):270-277.

Mills, J.H., Lee, F-S., Dubno, J., and Boettcher, F.A. (1996). Interactions between age-related and noise-induced hearing loss. In Scientific Basis of Noise-Induced Hearing Loss, A. Axelsson, H.M. Borchgrevnik, R.P. Hamernik, P-A. Hellström, D. Henderson and R.J. Salvi (eds). Thieme Medical Publishers, Inc., NY.

Mizukoshi, K., Watanabe, Y., Kobayashi, H., Nikano, Y., Koide, C., Inomata, S., Saitoh, N. (1989). Neurotological follow-up studies upon Minimata disease. *Acta Otolaryngologica, Suppl* 468:353-357.

Möller, C., Ödkvist, L.M., Thell, J., Larsby B., Hydén, D., Bergholtz, L. (1989). Otoneurological findings in psycho-organic syndrome caused by industrial solvent exposure. *Acta Otolaryngologica*, 107:5-12.

Morata, T.C. (1989). Study of the effects of simultaneous exposure to noise and carbon disulfide on worker's hearing. *Scandinavian Audiology*, 18:53-58.

Morata, T.C., Dunn, D.E., Kretschmer, L.K., Lemasters, G.K., Keith, R.W. (1993). Occupational exposure to organic solvents and noise: effects on hearing. *Scandinavian Journal of Work, Environment and Health*, 19:245-254.

Morata, T.C., Engel, T., Durao, A., Costa, T.R.S., Krieg, E.F., Dunn, D.E., Lozano, M.A., (1997a). Hearing loss from combined exposures among petroleum refinery workers, *Scandinavian Audiology*, 26:141-149.

Morata, T.C., Fiorini, A.C., Fischer, F.M., Colacioppo, S., Wallingford, K.W., Krieg, E.F., Dunn, D.E., Gozzoli, L., Padrao, M.A., Cesar, C.L.G. (1997b). Toluene-induced hearing loss among rotogravure printing workers, *Scandinavian Journal of Work, Environment and Health* 23:289-298.

Morata, T.C., Franks, J.R., Dunn, D.E. (1994). Unmet needs in occupational hearing conservation. *The Lancet*, 344(8920):479.

Morris, T.M.O. (1969). Deafness following carbon monoxide poisoning. *Journal of Laryngology and Otology*, 83:1219-1225.

National Institute for Occupational Safety and Health, NIOSH (1996). National Occupational Research Agenda. Cincinnati: USDHHS, PHS, CDC, NIOSH, publication no.96-115.

National Institute for Occupational Safety and Health, NIOSH (1998). Criteria for a recommended standard. Occupational exposure to noise. Revised Criteria. Cincinnati: USDHHS, PHS, CDC, NIOSH, publication no.98-126.

Nikolov, Z. (1974). Hearing reduction caused by manganese and noise. J*ournal Français d' Oto-Rhino-Laryngologie, Audiophonologie at Chirurgie Maxillofaciale*, 23:231-234.

Nilson, P., Erlandsson, B., Hakanson, H., Ivarsson, A., and Wersall, J. (1980). Morphological damage in the guinea pig cochlea after impulse noise and pure tone exposures. *Scand. Audiol. Suppl*. 12:155-162.

Ödkvist, L.M., Arlinger, S.D., Edling, C., Larsby, B., Bergholtz, L. (1987). Audiological and vestibulo-ocul-o-motor to findings in workers exposed to solvents and jet-fuel. *Scandinavian Audiology*, 16(2):75-84.

Ödkvist, L.M., Bergholtz, L.M., Åhlfeldt, H., Andersson, B., Edling, C., Strand, E. (1982). Otoneurological and audiological findings in workers exposed to industrial solvents. *Acta Otolaryngologica, suppl*. 386:249-251.

Occupational Safety and Health Administration, OSHA (1981). Final regulatory analysis of the hearing conservation amendment. Report number 723 860/752 1-3. Washington, DC: US Government Printing Office.

Passchier-Vermeer, W. (1983). Measurement and rating of impulse noise in relation to noise-induced hearing loss. Proceedings 4th International Congress - Noise as a Public Health Hazard. ed: G. Rossi, Torino, Italy, pp. 143-157.

Phaneuf, R., Hétu, R. (1990). An epidemiological perspective of the causes of hearing loss among industrial workers. *Journal of Otolaryngology*, 19(1):31-40.

Roberto, M., Hamernik, R.P., Salvi, R.J., Henderson, D. and Milone, R. (1985). Impact noise and the equal energy hypothesis. *J. Acoust. Soc. Am*. 77: 1514-1520.

Schwartz, J., Otto, D. (1987). Blood lead, hearing thresholds, and neurobehavioral development in children and youth. *Archives of Environmental Health*, 42:153-160.

Sulkowski, W.J., Kowalska, S., and Lipowczan, A. (1983). Hearing loss in weavers and drop-forge hammermen: Comparative study on the effects of steady-state and impulse noise. In: Proceedings of the 4th International Congress - Noise as a Public Health Problem, ed. G. Rossi, Turin, Italy, pp. 171-184.

Taylor, W., Lempert, B., Pelmear, P., Hemstock, I., and Kershaw, J. (1984). Noise levels and hearing thresholds in the drop forging industry. *J. Acoust. Soc. Am.*,76:807-819.

Theiry, L. (1987). Characterization of industrial impulse noise by discriminant analysis. *J. Sound and Vibration* 113:404-406.

Theiry, L. and Meyer-Bisch, C. (1988). Hearing loss due to partly impulsive industrial noise exposure at levels between 87 and 90 dB. *J. Acoust. Soc. Am.* 84:651-659.