

Now Hear This . . .



Volume 6, No. 1

Spring 2003

Chemically-Induced Hearing Loss (CIHL)

Antony Joseph, AuD, CCC-A

Occupational hearing conservationists have become increasingly conscientious of the importance of obtaining a thorough case history, including information about use of cochleotoxic medications such as aspirin, non-steroidal anti-inflammatory drugs, and cis-platin (Seligmann et al. 1996). The ototoxic side effects of medications are listed in the Physician's Desk Reference (PDR). Use of these medications, alone or in combination with exposure to hazardous noise, can result in high-frequency sensorineural hearing loss. An astute clinician realizes that if an ototoxic pharmaceutical treatment is discontinued promptly, reversal of hearing loss and tinnitus is possible. Due to a higher level of awareness, questions about ototoxic medications are included in most case histories, however harmful agents found in industrial settings typically go uninvestigated. Surprisingly, there is quite an impressive list of ototoxic chemical agents, solvents, gases, paints, heavy metals, and pesticides (Barregard and Axelsson, 1984; Ernest et al. 1995; Fechter, 1995; Morata et al 1995; Uroske et al, 2002). A few common occupations and recreational activities associated with these

substances are listed in Table I. A more complete listing can be found at www.cdc.gov/niosh/noise/noiseandchem/noiseandchem.html.

Toxic substances are widely used in industry, agriculture, and transportation. Some are ototoxins and some neurotoxins. These materials can cause a variety of insults to the auditory mechanism, such as sensorineural hearing loss (Barregard and Axelsson 1984), retrocochlear hearing loss (Hormes, Filley et al. 1986), and lesions in the higher auditory pathways (Moshe et al. 2002). Some substances in Table I have been better studied, including trichloroethylene, styrene, toluene, and xylene (Kowalska 2002). Toluene, styrene, and xylene simultaneously impair the central auditory system as well as the cells of the cochlear (Kowalska 2002). There are still relatively few studies on humans and chemically induced hearing loss (CIHL); therefore, most of our understanding of CIHL is from studies conducted on laboratory animals.

Presently, there is a growing body of medical literature (Fechter 1995; Morata and Lemasters

Table I. Jobs, Activities and Toxic Substances

Industries/Occupations	Ototoxins/Neurotoxins
Artistry, Aviation, Construction, Farming, Fireman, Landscaping, Machinist, Manufacturing, Recreational Activities Boating, Car Racing, Gardening, Home Improvement, Motocycling, Woodstaining	Acetone, Arsenic, Benzene, Carbon Disulfide, Cyanide, Ethyl Benzene, Lead, Manganese, Mercury, Methyl Ketone, N-hexane, Pesticides, Styrene (aromatic hydrocarbons), Thinner, Toluene, Trichloroethylene, Trimethyltin, Xylene

1995) that practitioners may refer to for an explanation of CIHL. Morata and Lemasters highlight the following characteristics of CIHL: (1) bilaterally symmetrical (2) irreversible (3) 3-6 kHz onset (4) usually cochlear or with some cochlear component. CIHL has the same characteristics as noise-induced hearing loss (NIHL). More recently, the National Institutes for Occupational Safety and Health (NIOSH) has used the term “work-related hearing loss” or “occupational hearing loss” to describe CIHL, NIHL, and related occupational hearing impairments (www.cdc.gov/niosh/noise/noisepeg.html). Morata and Lemasters indicate clearly that a challenge exists with differential diagnosis of CIHL, particularly when other ototoxins, noise, and presbycusis co-exist. In addition, there are no workplace regulations regarding interaction between noise and ototoxins (Morata 1998). In order for clinicians to venture a reasonable statement about the etiology of hearing loss a complete work history with both noise and chemical exposure is essential.

Audiologists normally use the clinical test results, the patient’s audiometric history (i.e., hearing conservation tests), and the patient’s health history to form an opinion about the cause of impairment. Given that ototoxicity commonly afflicts the outer hairs cells (OHCs) of the cochlear, an examination that includes otoacoustic emissions might yield substantive data. When assessing for CIHL, a standard test battery should include acoustic reflex testing, otoacoustic emissions, and evoked potentials in order to cover the entire auditory tract. Still, referral to otolaryngologist may be necessary to provide further specificity when peripheral or central neuropathy is suspected.

Because CIHL is not commonly recognized in audiologic practice, it is rarely identified as a cause of significant threshold shift. However, if pertinent health information is obtained from the patient, there might be more of an explanation of a threshold change or hearing loss in an individual without significant noise exposure. Clinicians are therefore encouraged to include the following questions in their existing hearing health intake:

- Have you been exposed to the substances in Table I?

- How long have you been exposed to these materials?
- Was your exposure inhaled, absorbed, or ingested?
- Do you use protective gear when exposed to these materials, and, if so, what do you wear?
- Do you have any hobbies that involve use of the materials in Table I?
- Are you receiving medical treatment for exposure to any of the materials in Table I?

Of course, in many circumstances, individuals may not remember the name of the agent or even know if they’ve actually been exposed to cochleotoxic substances at work. The patient can be asked to provide material safety data sheets (MSDS) on the chemicals in their workplace. By law, companies must have MSDS for all substances in their facilities and these fact sheets must be made available to workers.

Noise elevates blood flow in the inner ear, which, in turn, appears to act as a vehicle for introduction of chemicals into the vast array of cells in that structure. The presence of chemicals in that part of the auditory mechanism may result in decreased perfusion of the cochlear structures; reducing oxygen availability and causing cell damage. Although this is a plausible hypothesis it has yet to be proven (Fechter 1995). The same damage may simultaneously occur in the central nervous system; these chemicals are referred to as neurotoxins. To find recent advances in the area of CIHL and pathogenesis, you can review Best Practices Workshop: Combined Effects of Chemicals and Noise on Hearing (last held April, 2002): www.cdc.gov/niosh/noise/noiseandchem/noiseandchem.html.

A clinical sign of NIHL is a notching effect in the 3000 to 6000 Hz region on the audiogram. This notch worsens with time but rarely exceeds 60-70 dB HL. When it does, this should raise the suspicion of some other cause besides noise. The action level for initiation of hearing conservation in the industry is 85 dB(A). Researchers have suggested that the damage risk criterion level for simultaneous noise and chemical exposures is lower than 85 dB(A) (e.g., 80 dB(A)). Morata and Lemasters stated that CIHL generally occurs

earlier than what is typically seen with exposure to noise only.

According to the International Standards Organization second edition 1999-1990 (ISO 1990), hearing loss is defined as thresholds above 25 dB HL at 500-3000 Hz. The method proposes that after 30 years of unprotected exposure to an 85 dBA TWA(8) noise, less than 10% of the population will demonstrate a hearing loss. At 90 dBA TWA(8), the level of impairment rises to 12%, and at 95 dBA TWA(8) over 25% of the exposed population will incur a NIHL. Cohort studies have shown that up to 23% of solvent exposed individuals develop CIHL versus 5-8% in a non-chemical work environment (Bergstrom and Nystrom 1986). Presumably, exposure to both noise and chemicals increases the incidence of hearing loss, but more work is needed to understand the interaction. Can the prevalence data be added (i.e. 25% from NIHL plus 23% from CIHL = 48% prevalence)? In one study 53% of workers exposed to noise and toluene had hearing loss (Morata, Dunn et al. 1993) consistent with the hypothesis that the risk of noise and chemical exposure are additive. So, in order to protect the maximum number of exposed workers, what needs to be done for persons who are exposed to chemicals and noise simultaneously? Current standards are based on exposures to the individual hazard of noise or a particular chemical and do not protect against a possible increased risk from simultaneous exposure. For individuals who are exposed to

harmful substances and noise above 80 dBA, but not 85 dBA, enrollment in hearing conservation may be indicated, because of the potential synergistic effects of noise and chemicals.

A large number of chemicals exist in the world, and little is known about their propensity to cause auditory damage, particularly when interacting with noise and medications. It has been reported that ototoxins can cause STS when noise exposures are below damage risk levels (Fechter 1995). Worker's exposed to chemicals and high-level noise (e.g., 95 dBA or greater) might be considered for even a more stringent program of semi-annual monitoring.

Noise can interact with industrial agents to exacerbate hearing impairment. But unfortunately for hearing conservationists, readily available sources of toxicology health information such as the Material Safety Data Sheets (MSDS) generally do not list whether the chemical is ototoxic. If you suspect that someone with hearing loss is exposed to industrial chemicals please indicate that when you report the case. The follow-up investigation, a summary report of which will be sent to you, the referring healthcare provider, will include a determination not only of the noise but also the chemical exposure.

You can call Kenneth D. Rosenman, M.D. at 1-800-446-7805 if you have any questions about the ototoxic effects of chemicals. Ways to report a case are on the back page of this newsletter.

References

- Barregard, L., & Axelsson, A. (1984). Is there an ototraumatic interaction between noise and solvents? *Scand Audiol*, 13, 151-5.
- Bergstrom, B., & Nystrom, B. (1986). Development of hearing loss during long-term exposure to occupational noise. A 20-year follow-up study. *Scand Audiol*, 15, 227-34.
- Ernest, K., Thomas, M., Paulose, M., Rupa, V., & Gnanamuthu, C. (1995). Delayed effects of exposure to organophosphorus compounds. *Indian J Med Res*, 101, 81-4.
- Fechter, L. D. (1995). Combined effects of noise and chemicals. *Occup Med*, 10, 609-21.
- Hormes, J.T., C.M. Filley, et al. (1986). "Neurologic sequelae of chronic solvent vapor abuse." *Neurology* 36(5): 698-702.
- ISO (1990). Acoustics- Determination of Occupational Noise Exposure and Estimation of Noise-induced Hearing Impairment, *Geneva: 2nd ed, ISO 1999 1990(E)*.
- Kowalska, S. (2002). Auditory Effects of Occupational Exposure to Solvents, *Best Practices Workshop: Combined Effects of Chemicals and Noise on Hearing*. Dallas, TX.
- Morata, T. C. (1998). Assessing occupational hearing loss: beyond noise exposures. *Scand Audiol Suppl*, 48, 111-6.
- Morata, T. C., Dunn, D. E., Kretschmer, L. W., Lemasters, G. K., & Keith, R. W. (1993). Effects of occupational exposure to organic solvents and noise on hearing. *Scand J Work Environ Health*, 19, 245-54.
- Morata, T.C. and G.K. Lemasters (1995). "Epidemiologic considerations in the evaluation of occupational hearing loss." *Occup Med* 10 (3): 641-56.
- Moshe, S., Bitchatchi, E., Goshen, J., & Attias, J. (2002). Neuropathy in an artist exposed to organic solvents in paints: a case study. *Arch Environ Health*, 57, 127-9.
- Seligmann, H., Podoshin, L., Ben-David, J., Fradis, M., & Goldsher, M. (1996). Drug-induced tinnitus and other hearing disorders. *Drug Saf*, 14, 198-212.

Now Hear This...

**Michigan State University
College of Human Medicine
117 West Fee Hall
East Lansing, MI 48824-1316
Phone (517) 353-1955**

Address service requested.

In this issue:
Chemically-Induced Hearing Loss

Printed on recycled paper.

**Michigan Law Requires the
Reporting of Known or Suspected
Occupational NIHL**
Reporting can be done by:
FAX 517-432-3606
Telephone 1-800-446-7805
E-Mail ODRREPORT@ht.msu.edu
Web www.chm.msu.edu/ocm
Mail MDCIS Occ Health Div
P.O. Box 30649
Lansing, MI 48909-8149
**Suggested Criteria for Reporting
Occupational NIHL**
1. A history of significant exposure to noise
at work; AND
2. A STS of 10 dB or more in either ear at an
average of 2000, 3000 & 4000 Hz. OR
3. A fixed loss.*
*Suggested definitions: a 25 dB or greater loss in
either ear at an average of: 500, 1000 & 2000
Hz; or 1000, 2000 & 3000 Hz; or 3000, 4000 &
6000 Hz; or a 15 dB or greater loss in either ear
at an average of 3000 & 4000 Hz.

Project SENSOR Staff
*At the Michigan Department
of Consumer and Industry Services*
Douglas J. Kalinowski, C.I.H., M.S.,
Director
Bureau of Safety and Regulations
Project SENSOR, Co-Director
John Peck, C.I.H., M.S., Chief
Occupational Health Division
Bill Dehede, M.P.H.
Regional Supervisor
Project SENSOR-MDCIS Liaison
Byron Panasuk, I.H.
Project SENSOR Specialist
*At Michigan State University—
College of Human Medicine*
Kenneth D. Rosenman, M.D.,
Professor of Medicine
Project SENSOR, Co-Director
Mary Jo Reilly, M.S.
Project SENSOR Coordinator
Amy Sims, B.S.
Project SENSOR NIHL Coordinator
Now Hear This...., Editor
Project SENSOR Office Staff:
Tracy Carey
Ruth Vanderwaals
Patient Interviewers:
Danielle Arnold Amy Krizek
Noreen Hughes Diana Okuniewski

Advisory Board
Phyllis Berryman, RN
Michigan Occupational
Nurses' Association
Patricia Brogan, Ph.D.,
Wayne State University
Wayne Holland, Ph.D.,
Michigan Speech-Language-
Hearing Association
Jerry Punch, Ph.D.,
Michigan State University
Constance Spak, M.A., CCC-A
University of Michigan
Michael Stewart, Ph.D.,
Better Hearing
Central Michigan University
Jeffrey Weinarten, M.D.,
Michigan Otolaryngology Society
Now Hear This is published quarterly by Michi-
gan State University-College of Human Medicine
with funding from the Michigan Department of
Consumer and Industry Services and is available
at no cost. Suggestions and comments are wel-
come.
(517) 353-1846
MSU-CHM
117 West Fee Hall
East Lansing, MI 48824-1316