Occupational Noise-Induced Hearing Loss

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Occupational hearing loss is preventable through a hierarchy of controls, which prioritize the use of engineering controls over administrative controls and personal protective equipment. The occupational and environmental medicine (OEM) physician plays a critical role in the prevention of occupational noise-induced hearing loss (NIHL). This position statement clarifies current best practices in the diagnosis of occupational NIHL.

Noise-induced hearing loss (NIHL) continues to be one of the most prevalent occupational conditions and occurs across a wide spectrum of industries. Occupational hearing loss is preventable through a hierarchy of controls, which prioritize the use of engineering controls over administrative controls and personal protective equipment. The occupational and environmental medicine (OEM) physician works with management, safety, industrial hygiene, engineering, and human resources to ensure that all components of hearing loss prevention programs are in place.[1] The OEM physician should emphasize to employers the critical importance of preventing hearing loss through controls and periodic performance audits rather than just conducting audiometric testing. Nevertheless, audiometric testing, besides documenting the permanent loss of hearing, can be of value in the identification of hearing loss at a time when early preventive intervention is possible. The American College of Occupational and Environmental Medicine (ACOEM) believes that OEM physicians should understand a worker's noise exposure history and become proficient in the early detection and prevention of NIHL.

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THE OEM PHYSICIAN AS PROFESSIONAL SUPERVISOR OF THE AUDIOMETRIC TESTING COMPONENT OF A HEARING CONSERVATION PROGRAM

The OEM physician also plays a critical role in the prevention of occupational NIHL by serving as a professional supervisor of the audiometric testing element of hearing conservation programs. The Occupational Safety and Health Administration (OSHA) defines a requirement for professional supervisors in the 1983 Hearing Conservation Amendment.[2] The responsibilities of the professional supervisor can be found in the ACOEM position statement *The Role of the Professional Supervisor in the Audiometric Testing Component of Hearing Conservation Programs*.[3] Responsibilities include interpretation of audiograms, work-relatedness determinations, referral of problem cases, quality oversight of audiometric testing, and determination of the effectiveness of the hearing conservation program.

This statement clarifies current best practices in the diagnosis of NIHL. On the basis of current knowledge, it updates the previous ACOEM statement[4] regarding the distinguishing features of occupational NIHL.

DEFINITION

Occupational NIHL develops gradually over time and is a function of continuous or intermittent noise exposure. This is in contrast to occupational acoustic trauma which is characterized by a sudden change in hearing as a result of a single exposure to a sudden burst of sound, such as an explosive blast. The diagnosis of NIHL is made by the OEM physician, by first taking into account the worker's noise exposure history and then by considering the following characteristics.

CHARACTERISTICS

The principal characteristics of occupational NIHL are as follows:

- It is always sensorineural, primarily affecting the cochlear hair cells in the inner ear.
- It is typically bilateral, since most noise exposures affect both ears symmetrically.
- Its first sign is a "notching" of the audiogram at the high frequencies of 3000, 4000, or 6000 Hz with recovery at 8000 Hz.[5]
  - This notch typically develops at one of these frequencies and affects adjacent frequencies with continued noise exposure. This, together with the effects of aging, may reduce the prominence of the "notch." Therefore, in older individuals, the effects of noise may be difficult to distinguish from age-related hearing loss (presbycusis) without access to previous audiograms.[6]
  - The exact location of the notch depends on multiple factors including the frequency of the damaging noise and size of the ear canal.
  - In early NIHL, average hearing thresholds at the lower frequencies of 500, 1000, and 2000 Hz are better than average thresholds at 3000, 4000, and 6000 Hz, and the hearing level at 8000 Hz is usually better than the deepest part of the notch. This notching is in contrast to presbycusis, which also produces high-frequency hearing loss but in a down-sloping pattern without recovery at 8000 Hz.[7]
  - Although OSHA does not require audiometric testing at 8000 Hz, inclusion of this frequency is highly recommended to assist in the identification of the noise notch as well as age-related hearing loss.[8]
- Noise exposure alone usually does not produce a loss greater than 75 dB in high frequencies and greater than 40 dB in lower frequencies. Nevertheless, individuals with non-NIHL, such as presbycusis, may have hearing threshold levels in excess of these values.[8]

- Hearing loss due to continuous or intermittent noise exposure increases most rapidly during the first 10 to 15 years of exposure, and the rate of hearing loss then decelerates as the hearing threshold increases.[9] This is in contrast to age-related loss, which accelerates over time.
- Available evidence indicates that previously noise-exposed ears are not more sensitive to future noise exposure.
- There is insufficient evidence to conclude that hearing loss due to noise will progress once the noise exposure is discontinued.[8] This is primarily based on a National Institute of Medicine report which concluded that, on the basis of available human and animal data, it was felt unlikely that such delayed effects occur.[9,10] However, recent animal experiments indicate although there appears to be threshold recovery and no loss of cochlear cells following noise exposures to rodents, there is evidence of cochlear afferent nerve terminal damage and delayed degeneration of the cochlear nerve, thus suggesting that delayed effects could be seen in the future.[11]
- Although the OSHA action level for noise exposure is 85 dB (8-hour time-weighted average), the evidence suggests that noise exposure from 80 to 85 dB may contribute to hearing loss in individuals who are unusually susceptible. The risk of NIHL increases with long-term noise exposures above 80 dB and increases significantly as exposures rise above 85 dB.[12,13]
- Continuous noise exposure throughout the workday and over years is more damaging than interrupted exposure to noise, which permits the ear to have a rest period. At the present time, measures to estimate the health effects of such intermittent noise are controversial.
- Real world attenuation provided by hearing protective devices may vary widely between individuals. The noise-reduction rating of hearing protective devices used by a working population is expected to be less than the laboratory-derived rating.[14,15] Hearing protective devices should provide adequate attenuation to reduce noise exposure at the eardrum to less than 85 dB time-weighted average. In addition, technology is now available, which can provide an individualized attenuation rating for hearing protective devices and continuous monitoring of noise at the eardrum.[16–18]
- The presence of a temporary threshold shift (ie, the temporary loss of hearing, which largely disappears 16 to 48 hours after exposure to loud noise) with or without tinnitus is a risk indicator that permanent NIHL will likely occur if hazardous noise exposure continue.[19] Barring an ototraumatic incident, workers will always develop temporary threshold shift before sustaining permanent threshold shift.[1]

ADDITIONAL CONSIDERATIONS IN EVALUATING THE WORKER WITH SUSPECTED NIHL

The OEM physician evaluating possible cases of NIHL should consider the following issues:

- Unilateral sources of noise such as sirens and gunshots can produce asymmetric loss, as can situations in which the work involves fixed placement of the affected ear relative to the noise source. When evaluating cases of asymmetric loss, referral to rule out a retrocochlear lesion, such as an acoustic neuroma,[20] is warranted before attributing the loss to noise. The physician should consult criteria, such as from the American Academy of Otolaryngology—Head and Neck Surgery, which can assist in making referrals for further evaluation.[21,22]
- Animal exposure data suggest that the addition of very intense and frequent impulse/impact noise to steady-state noise can be more harmful than steady-state noise of the same A-weighted energy exposure. (A-weighting is the most common noise measurement scale. A-weighting best approximates the way the human ear perceives loudness at moderate sound levels and it de-emphasizes high and low frequencies that the average person cannot hear.) Nevertheless, human data are currently too sparse to derive an exposure metric, which can practically estimate such a hazardous noise risk.[23,24]
- Animal models suggest that exposure to ototoxic agents, such as solvents (notably ethylbenzene, methylstyrene, n-hexane, n-propylbenzene, p-xylene, styrene, trichloroethylene, and toluene), may act in synergy with noise to cause hearing loss. Asphyxiants (carbon monoxide and hydrogen cyanide), some nitriles (such as acrylonitrile), and metals (lead, mercury, and tin) have also been implicated as causing ototoxicity. The involvement can be seen as damage to cochlear hair cells, central nervous system, or both. Although the scientific understanding of the role of all these chemicals in human ototoxicity is still evolving, a thorough exposure history to these chemicals should be obtained and taken into consideration when evaluating sensorineural hearing loss.[25–27] Further, the hierarchy of primary prevention controls should be implemented in order to mitigate the risk of an acquired dose to workers, or others, potentially exposed to ototoxic chemicals.
- Individual susceptibility to the auditory effects of noise varies widely.[28] The biological basis for this remains unclear. In addition, the contribution of comorbid conditions such as cardiovascular disease, diabetes, and neurodegenerative disease to hearing loss is unclear.[29]
- There are a number of other causes of sensorineural hearing loss besides occupational noise. Of primary concern is non-occupational noise exposure from a variety of sources, especially recreational noise, such as loud music, weapons firing, motor sports, etc. Other causes include a wide variety of genetic disorders, infectious diseases (eg, labyrinthitis, measles, mumps, syphilis), pharmacologic agents (eg, aminoglycosides, diuretics, salicylates, antineoplastic agents), head injury, therapeutic radiation exposure, neurologic disorders (eg, multiple sclerosis), cerebral vascular disorders, immune disorders, bone (eg, Paget disease), central nervous system neoplasms, and Menière's disease. A medical history can help in determining whether any of these conditions could contribute to an individual's hearing loss.[30] Nevertheless, the Genetic Information Nondiscrimination Act in some instances precludes the OEM physician from obtaining a family history,[31] which could give insight into genetic disorders such as Alport syndrome. There is an exception for when the family medical history is collected for diagnostic or treatment purposes. In such cases, when genetic or any other non-occupational condition noted earlier is suspected, a referral to an otolaryngologist or other appropriate specialist is recommended.
- Individuals with NIHL may experience significant morbidity due to hearing loss, concomitant tinnitus, and/or impaired speech discrimination. On the job, such hearing loss can impact worker communications and safety. Other conditions associated with noise exposure and/or hearing loss are hypertension, depression, dementia,[32] social isolation,[33] increased risk of accidents,[34–36] and retrocochlear lesions.[37–41] Workers with evidence of hearing loss require an individualized evaluation that takes into account both the need to communicate safely and effectively and the need for protection from additional damage due to noise.
- Because hearing loss due to noise is irreversible, early detection and

intervention is critical to prevention of this condition. Ensure baseline audiograms are obtained for new hires and/or employees newly identified as working within a noise-laden environment. A 10-dB confirmed threshold shift from baseline in pure-tone average at 2000, 3000, and 4000 Hz (OSHA standard threshold shift or STS), while not necessarily resulting in significant impairment, is an important early indicator of permanent hearing loss.[42] A temporary threshold shift is an important early and reversible indicator that potential cochlea hair cell damage can progress to an STS, unless preventive interventions occur. Tinnitus is another early warning symptom for NIHL.[8] Other early warning flags, such a 10-dB non-age-corrected STS or an 8-dB age-corrected STS, may have a higher positive predictive value in identifying those individuals who will progress to impaired hearing.[43] Therefore, individuals in hearing conservation programs who exhibit such shifts on serial audiometric testing should be carefully evaluated and counseled regarding avoidance of noise and correct use of personal hearing protection.

- Age correction of audiograms is a method of age standardization, which allows comparisons of hearing loss rates among working populations. OSHA allows, but does not require, the use of an age-correction procedure.[2] Age-correction factors are averages for a population—some individuals will exhibit more age-related loss and some less. Therefore, the application of age correction to the surveillance audiograms of a noise-exposed population can result in fewer confirmed 10-dB shifts being reported. Thus, when applying age correction to the audiometric results of an individual who has experienced a threshold shift, the OEM physician should consider whether, in that individual, a preventable noise component of hearing loss could play a role.
- Any assessment of hearing loss requires the review of all previous audiograms, as well as noise exposure records, hearing protection data, and clinical history, to assist in the diagnosis of NIHL. A referral for a comprehensive audiology evaluation, including bone conduction testing, can assist in verifying the nature of hearing loss.[44]

THE OEM PHYSICIAN'S ROLE IN DIAGNOSING NIHL

The OEM physician plays a major role in the prevention of NIHL, and to make an evidence-based clinical diagnosis, must understand factors contributing to noise exposure in the workplace, non-occupational sources of noise, chemicals known to be ototoxic, comorbidities impacting hearing, and the pathophysiology of NIHL and its clinical and audiometric characteristics. Making a diagnosis of NIHL is an important step in preventing further hearing loss in the affected worker and for identifying the potential for NIHL in coworkers. The OEM physician must work with management and other safety and health professionals to evaluate the workplace for noise exposure, educate the workers regarding the risk of noise exposure (occupational and non-occupational), and reduce the potential for noise exposure.

REFERENCES

1. NIOSH. Criteria for a Recommended Standard: Occupational Noise Exposure. Cincinnati, OH: National Institute for Occupational Safety and Health; 1998:98–126. Available at: https://www.cdc.gov/niosh/docs/98-126/pdfs/98-126.pdf. Accessed March 26, 2018.
2. Occupational Health and Safety Administration. 1910.95 CFR occupational noise exposure: hearing conservation amendment (final rule). *Fed Reg*. 1983;48:9738–9785.
3. Rabinowitz P. ACOEM Position Statement. The role of the professional supervisor in the audiometric testing component of the hearing conservation program. [website.]; 2007.
4. Kirchner DB, Evenson E, Dobie RA, et al. ACOEM guidance statement. Occupational noise-induced hearing loss. *J Occup Environ Med*. 2012;54:106–108.
5. McBride D, Williams S. Audiometric notch as a sign of noise induced hearing loss. *Occup Environ Med*. 2001;58:46–51.
6. Consensus Conference. Noise and hearing loss. *JAMA*. 1990;263:3185–3190.
7. Coles R, Lutman M, Buffin J. Guidelines on the diagnosis of noise-induced hearing loss for medicolegal purposes. *Clin Otolaryngol Allied Sci*. 2000;25:264–273.
8. Dobie RA. *Medical-Legal Evaluation of Hearing Loss*. 3rd ed. San Diego, CA: Plural Publishing; 2015.
9. Durch JS, Joellenbeck LM, Humes LE, eds. Noise and Military Service: Implications for Hearing Loss and Tinnitus. Washington, DC: National Academies Press; 2005.
10. Lee FS, Matthews LJ, Dubno JR, Mills JH. Longitudinal study of pure-tone thresholds in older persons. *Ear Hear*. 2005;26:1–11.
11. Kujawa SG, Liberman MC. Adding insult to injury: cochlear nerve degeneration after ''temporary'' noise-induced hearing loss. *J Neurosci*. 2009;29:14077–14085.
12. American National Standards Institute. *Determination of Occupational Noise Exposure and Estimation of Noise-Induced Hearing Impairment, ANSI S3.44*. Acoustical Society America; 1996
13. National Institute for Occupational Safety and Health (NIOSH). Comments from the National Institute for Occupational Safety and Health on the Occupational Safety and Health Administration's Request for Comments on Determining the Work-Relatedness of Occupational Hearing Loss.
14. Lusk SL, Kerr MJ, Kauffman SA. Use of hearing protection and perceptions of noise exposure and hearing loss among construction workers. *Am Ind Hyg Assoc J*. 1998;59:466–470.
15. Berger EH, Franks JR, Behar A, et al. Development of a new standard laboratory protocol for estimating the field attenuation of hearing protection devices. Part III. The validity of using subject-fit data. *J Acoust Soc Am*. 1998;103:665–672.
16. Hager LD. Fit-testing hearing protectors: an idea whose time has come. *Noise Health*. 2011;13:147–151.
17. Michael K, Tougaw E, Wilkinson R. Role of continuous monitoring in a hearing conservation program. *Noise Health*. 2011;13:195–199.
18. Schulz TY. Individual fit-testing of earplugs: a review of uses. *Noise Health*. 2011;13:152–162.
19. Moshammer H, Kundi M, Wallner P, Herbst A, Feuerstein A, Hutter HP. Early prognosis of noise-induced hearing loss. *Occup Environ Med*. 2015;72:83–84.
20. Zapala DA, Shaughnessy K, Buckingham J, Hawkins DB. The importance of audiologic red flags in patient management decisions. *J Am Acad Audiol*. 2008;19:564–570.
21. Suter AH, Berger EH. *Hearing Conservation Manual*. Milwaukee, WI: Council for Accreditation in Occupational Hearing Conservation; 2002.
22. American Academy of Otolaryngology Head and Neck Surgery. Policy Statement: Red flags-warning of ear disease [website.]. Available at: http://www.entnet.org/?q=node/912. Accessed May 14, 2018.
23. Qiu W, Hamernik RP, Davis B. The kurtosis metric as an adjunct to energy in the prediction of trauma from continuous, nonGaussian noise exposures. *J Acoust Soc Am*. 2006;120:3901–3906.
24. Goley GS, Song WJ, Kim JH. Kurtosis corrected sound pressure level as a noise metric for risk assessment of occupational noises. *J Acoust Soc Am*. 2011;129:1475–1481.
25. Morata TC. Chemical exposure as a risk factor for hearing loss. *J Occup Environ Med*. 2003;45:676–682.
26. European Agency for Safety and Health at Work. Combined Exposure to Noise and Ototoxic Substances; 2011. Available at: https://osha.europa.eu/en/tools-and-publications/publications/literature_reviews/combined-exposure-to-noise-and-ototoxic-substances. Accessed May 14, 2018.
27. Campo P, Morata TC, Hong O. Chemical exposure and hearing loss. *Dis Mon*. 2013;59:119.
28. Stucken EZ, Hong RS. Noise-induced hearing loss: an occupational medicine perspective. *Curr Opin Otolaryngol Head Neck Surg*. 2014;22:388–393.
29. Ward WD. Endogenous factors related to susceptibility to damage from noise. *Occup Med*. 1995;10:561–575.
30. Arts H. Sensorineural hearing loss in adults. In: Otolaryngology: Head and Neck Surgery, 5th ed. Philadelphia, PA: Mosby Elsevier; 2010.
31. US Equal Employment Opportunity Commission/US Congress. The Genetic Information Non-Discrimination Act; 2008. Available at: https://www.eeoc.gov/laws/statutes/gina.cfm. Accessed May 14, 2018.
32. Lin FR, Metter EJ, O'Brien RJ, Resnick SM, Zonderman AB, Ferrucci L. Hearing loss and incident dementia. *Arch Neurol*. 2011;68:214–220.
33. Hétu R, Getty L, Quoc HT. Impact of occupational hearing loss on the lives of workers. *Occup Med*. 1995;10:495–512.
34. Zwerling C, Sprince NL, Davis CS, Whitten PS, Wallace RR, Heeringa SG. Occupational injuries

among older workers with disabilities: a prospective cohort study of the Health and Retirement Survey, 1992 to 1994. *Am J Public Health*. 1998;88:1691–1695.

35. Girard SA, Leroux T, Courteau M, Picard M, Turcotte F, Richer O. Occupational noise exposure and noise-induced hearing loss are associated with work-related injuries leading to admission to hospital. *Inj Prev*. 2015;21:e88–e92.
36. Lin FR, Ferrucci L. Hearing loss and falls among older adults in the United States. *Arch Intern Med*. 2012;172:369–371.
37. Edwards CG, Schwartzbaum JA, Lönn S, Ahlbom A, Feychting M. Exposure to loud noise and risk of acoustic neuroma. *Am J Epidemiol*. 2006;163:327–333.
38. Hours M, Bernard M, Arslan M, et al. Can loud noise cause acoustic neuroma? Analysis of the INTERPHONE study in France. *Occup Environ Med*. 2009;66:480–486.
39. Preston-Martin S, Thomas D, Wright W, Henderson B. Noise trauma in the aetiology of acoustic neuromas in men in Los Angeles County, 1978–1985. *Br J Cancer*. 1989;59:783–786.
40. Prochazka M, Feychting M, Ahlbom A, et al. Occupational exposures and risk of acoustic neuroma. *Occup Environ Med*. 2010;67:766–771.
41. Schlehofer B, Schlaefer K, Blettner M, et al. Environmental risk factors for sporadic acoustic neuroma (Interphone Study Group, Germany). *Eur J Cancer*. 2007;43:1741–1747.
42. Dobie RA. Audiometric threshold shift definitions: simulations and suggestions. *Ear Hear*. 2005;26:62–77.
43. Rabinowitz PM, Galusha D, Ernst CD, Slade MD. Audiometric ''early flags'' for occupational hearing loss. *J Occup Environ Med*. 2007;49:1310–1316.
44. Lasak JM, Allen P, McVay T, Lewis D. Hearing loss: diagnosis and management. *Prim Care*. 2014;41:19–31.

