Occupational Hearing Loss

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Hearing loss is a significant and unfortunately common occupational malady. Over the past several decades both the Occupational Safety and Health Administration (OSHA) and the National Institute for Occupational Safety and Health (NIOSH) have initiated efforts to better understand and to limit the occurrence of occupational hearing loss, particularly as it relates to excessive noise exposure. This paper briefly addresses the pathophysiology of noise-induced hearing loss and then describes the occupational and non-occupational factors which influence a worker's risk of hearing loss. The primary foci of this discussion are the clinical evaluation, diagnosis, and management of occupational hearing loss. Issues of prevention, OSHA-mandated hearing conservation efforts and compensation are reviewed. Am. J. Ind. Med. 37:112–120, 2000. *© 2000 Wiley-Liss, Inc.*

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INTRODUCTION

Sound

Any discussion of noise, its effects and the Occupational Safety and Health Administration's (OSHA) attempts to limit noise-related injury presumes a grasp of the most basic physics of sound. Important characteristics of this phenomenon are the rate or frequency of the oscillating pressure waves and the amplitude or pressure of these sound waves. The range of each of these characteristics which can be perceived by the human ear is remarkable. Frequency, measured in cycles per second or Hertz, is commonly tested over a range of 250 Hertz (Hz) to 8 kiloHertz (kHz) by the audiologist, but the ear can actually detect tones both above and below these frequencies. The range of pressure of these sound waves (0.0002 to >2000 microbar) is so broad that it is described logarithmically in decibels.

Decibels (dB) actually describe the logarithmic ratio of the intensity of a given sound to that of a sound which is just perceptible to a person with normal hearing. Under this relationship, a doubling of sound intensity will result in an increase of 3 dB. Thus a worker exposed to 60 dB from a machine will experience increased sound intensity if a second similar machine is started next to him. However the net decibels will not be 120 dB, it will be 63 dB.

Because of the shape of the external ear canal and other factors, the human's sensitivity to sound is greatest between 1000 and 5000 Hz. A worker exposed to 90 decibels in this frequency range is at greater risk of injury than one exposed to 90 dB of 250 Hz sound. In an effort to describe noise in a uniform, meaningful fashion for the wide variety of frequencies experienced by workers, occupational noise is measured, studied and regulated with "A filtering". This applies a weighting algorithm to sound pressures as they are being measured so that noise in the range of highest human sensitivity is given a relatively greater decibel value. This sound level is then reported in terms of "dB(A)". Virtually all sound levels of significance to occupational exposures are expressed as dB(A).

Scope of the Problem

Data from both OSHA and the U.S. Environmental Protection Agency (EPA) suggest that exposure to poten-

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tially harmful noise levels is common in the American workplace. The National Institute for Occupational Safety and Health (NIOSH) estimates that the number of workers encountering hazardous levels of noise with or without other potentially ‘‘ototraumatic agents’’ is in the range of 30 million [Franks et al., 1996]. These levels are encountered in construction, mining, agriculture, manufacturing and utilities, transportation, and in the military. Estimates by OSHA suggest that nearly one quarter of the workers in these industries routinely encounter noise levels in the 90–100 dB(A) range [U.S. Department of Labor OSHA, 1981]. Noise surveys cited by NIOSH found that over one quarter of workers in textile mills, petroleum and coal production, lumber and wood production, and food production were exposed to 90 dB(A) or greater. Other industries with significant noise included transportation, fabricated metal production, stone, clay and glass production, primary metal industries, rubber and plastic production, and the manufacture of paper, chemicals, and electrical equipment [NIOSH, 1972]. Such noise exposures have generated a sizable population of workers who meet OSHA's definition of ‘‘material impairment of hearing’’ (an average threshold of $\geq$ 25 dB at 1000, 2000, and 3000 Hz). While noise accounts for the majority of occupational hearing loss (OHL), it is important to recognize that occupational exposure to chemicals, barotrauma, foreign bodies, etc. also may cause hearing loss.

Many affected workers actually experience losses considerably beyond 25 dB and these can have significant effects on their employment, their social interactions, and their family interactions [Hetu et al., 1995; NIH, 1990]. These workers may experience problems ranging from tinnitus to difficulty in detecting and recognizing sounds in the setting of background noise. This problem may impair their ability to detect warning signals, to discriminate between different frequencies, to comprehend speech, and to localize sound sources. The sound attenuation produced in this setting by hearing protective devices (HPD) like earplugs or muffs may affect the hearing impaired worker to a far greater degree than it would the normal worker [Hetu et al., 1995].

Pathophysiology

Perception of sound depends on the conduction of mechanical sound energy through the ossicles of the middle ear to the hydraulic medium of the cochlea. The mechanical energy is translated into neural afferent information by the hair cells of the organ of Corti within the spiral structure of the cochlea. This function depends on the structural integrity of the hair cells and surrounding support cells, local vascular structures, and the immediate microenvironment of the organ of Corti, which is bathed in a potassium-rich fluid. With excessive noise exposure, experimental animals show cellular changes within the hair cells of the cochlea, including loss of cilia and actual disruption of the hair cells [Henderson and Hamernik, 1995]. However, animal studies have repeatedly demonstrated that the relationship of these types of changes in the cochlea to decreases in hearing acuity is, at best, indirect [Hamernik et al., 1989]. Other factors such as leakage of extracellular fluid into the microenvironment and damage to support cells, vascular and neural structures—all may be playing a role in hearing loss secondary to loud noise.

Regardless of the exact cellular mechanism, several phenomena related to loud noise do appear to be well-established. Damage within the cochlea tends to occur initially and to the greatest degree in the portion which detects sound in the 3000–4000 Hz range. This progresses steadily over the initial decade of exposure and then tends to plateau. Typically, the next affected area is in the 6000 Hz region followed by the 8000 and the 2000 Hz regions where losses are more slowly progressive [Taylor et al., 1965]. In most cases this injury will cause the exposed worker to have a relatively symmetrical, bilateral sensorineural deficit. In theory, this damage reflects both the intensity of the noise and the length of exposure in a fashion which is predictable (‘‘*equal energy principle*’’). In reality, the degree of hearing loss is usually not linear with respect to exposure. Rather, the worker may experience a disproportionate loss in the early years of the exposure. The immediate response to damaging noise is a transient blunting of hearing acuity which shifts the subject's threshold of barely audible sound up to a higher level of sound for a period of hours. These episodes of *temporary threshold shift (TTS)* indicate an exposure to a harmful level of noise. Repeated exposure to noise in this range of intensity will eventually lead to *permanent threshold shift (PTS)*.

OCCUPATIONAL RISK FACTORS

Clearly, the major risk factor for occupational hearing loss is excessive noise on the job. After considerable debate, OSHA arrived at the level of 90 dB(A) of exposure over an eight hour period as the highest allowable dose—one which was predicted to produce acceptably low rates of hearing loss in excess of 25 dB in the 1–2–3 kHz range. Based upon the equal energy principle noted above, higher levels of noise can be tolerated for proportionately shorter periods of time. OSHA has specified that a 5 dB ‘‘exchange rate’’ be used to calculate acceptable noise exposures. Thus using the OSHA exchange rate, an exposure of four hours at 95 dB(A) is equivalent to eight hours of 90 dB(A), as is two hours of 100 dB(A). Regular exposure to levels in excess of these limits will substantially increase the risk of most workers for noise-induced hearing loss. This does not rule out similar injury to some workers at levels less than those defined by OSHA [Kryter et al., 1966]. An ‘‘action level’’ of

85 dB(A) for eight hours has been defined by OSHA as the point at which employees are to be entered into hearing conservation programs and provided with hearing protective devices.

Recognition of the importance of tracking losses in the 4000 Hz range and application of nonlinear models to previous NIOSH dose–response data [Prince et al., 1997] have led NIOSH to revise its 1972 recommendations on acceptable levels of exposure [NIOSH, 1998]. Continuing to advise an eight hour dose of 85 dB, the most important change is the proposal for a 3-dB exchange rate. It is argued that this is a more mathematically appropriate reflection of the physical behavior of sound and that it will provide substantially more protection to workers, particularly in the 4000 Hz range which makes an important contribution to speech discrimination. Under this lower exchange rate, the current OSHA standard of 90 dB could only be tolerated for 2.5 h. The currently accepted 8 h of 90 dB would represent 350% of the recommended daily noise exposure. It is estimated that these changes would cut the risk of hearing loss over a 40 year lifetime exposure from 25% to 8% [NIOSH, 1998].

A variety of factors have been considered as potential contributors in increasing the risk of threshold shift. The effects of sudden, explosive peaks of *impulse noise* (drop forge, jackhammer, firearms) may promote damage in excess of that predicted by the use of standard measuring devices. At very high levels (>140 dB), impulse noise can cause substantial mechanical disruption of middle and inner ear structures [Henderson, 1989]. Simultaneous exposure to certain *chemical agents* has been found in animals to exacerbate the effects of noise. These include carbon monoxide [Fechter et al., 1988] and organic solvents [Johnson and Nylen, 1995]. In the case of carbon monoxide, these changes can be noted at low level doses of the gas which produce effects that are at least additive. It is unclear whether any of these interactions with chemical ototoxins produce losses which are greater than additive [Fechter, 1995]. *Vibration*, either hand-arm or whole body, has been related to increased threshold shift in a number of animal studies [Pyyko et al., 1986]. This appears to be particularly notable when the vascular changes typical of vibration white finger are present. *Temperature* also may affect the worker's sensitivity to excessive noise with greater temporary threshold shifts being noted at higher ambient temperatures [Rentzsch and Minks, 1989].

In addition to noise-induced hearing loss (NIHL), people working in some jobs can experience hearing loss related to damage to the external ear or skull arising from blunt trauma or percussive injury. The structures of the middle ear may be damaged from mechanical or barotrauma. Burn injury to the ear canal or tympanic membrane may be seen in welders. Burn injury can also take place with entry of caustic materials into the ear canal. Other foreign bodies or cerumen impaction secondary to the use of ear plugs may occlude the canal and impair hearing.

Finally, exposure to ototoxic substances including heavy metals (mercury, lead, arsenic, cobalt), organic compounds (toluene, styrene, *n*-hexane, trichloroethylene, xylene), potassium bromate, aniline dyes, carbon monoxide, certain pharmaceutical agents and other compounds may play a role in some cases. Many of these agents have been shown to induce physiologic and histologic changes in animal models. Limited reports indicate that exposures in humans may result in hearing loss, though often these cases relate to unintentional high level exposures [Johnson and Nylen, 1995]. While an interaction with noise has been demonstrated for some of these substances, in most cases the combined effect of chemical and noise exposures are unclear.

NON-OCCUPATIONAL RISK FACTORS

Unlike many other occupational exposures, excessive noise is commonly encountered in non-occupational settings. A variety of recreational activities including woodworking, yard care, use of snowmobiles, firearms and rock music—either directly or via headphones—can all cause the same type of injury experienced by workers in excessively noisy work sites. Similarly some exposure to a number of other ototoxins may occur outside of work. Perhaps most notable in this group is a variety of medications ranging from antibiotics to chemotherapeutic agents (Table I). The use of ototoxic drugs and certain patient characteristics (dehydration, fever, renal or hepatic disease) may substantially increase the risk of hearing loss related to these medications. In some animal models there is an interaction between drugs (aminoglycosides and cisplatin) and noise exposures. Other phenomena as diverse as head trauma, infections, inherited disorders and aging—all may contribute to non-occupational losses of hearing acuity.

CLINICAL FEATURES

History

Workers seldom present with primary complaints of OHL. Rather this is often a possibility which must be considered by the practitioner and pursued during the course of the history. Questions should elicit symptoms such as tinnitus or suggestions of temporary threshold shifts. Individuals describing loud noise on the job, the necessity of shouting to communicate with coworkers, or ringing/buzzing following work exposures may well be experiencing excessively high levels of noise. Any worker with a history of potentially significant noise exposure on current or past jobs should be asked about problems with hearing at meetings, social gatherings, movies or while watching TV.

TABLE I. Ototoxicity Related to Medications

Medication	Primary site affected persistence	
Aminoglycosides		
Amikacin	cochlea	permanent
Gentamicin	vestibule	permanent
Kanamycin	cochlea	permanent
Neomycin	cochlea	permanent
Streptomycin	vestibule	permanent
Tobramycin	coch/vestib	permanent
Erythromycin analogues	cochlea	usually transient
Vancomycin	cochlea	permanent
Loop diuretics	cochlea	transient
Non-steroidal antiinflammatories	cochlea	transient
Antimalarials		
Quinine	coch/vestib	usually transient
Chloroquine	coch/vestib	usually transient
Deferoxamine	cochlea	transient
Antineoplastics		
Cis-platin	cochlea	permanent
Carboplatin	cochlea	permanent
Bleomycin	cochlea	permanent
Vincristine	cochlea	usually permanent
Vinblastine	cochlea	usually permanent

Difficulties with word discrimination in the presence of background noise, conflict with family members regarding hearing, or disagreement about TV and radio volume all are indicative of decreased hearing acuity.

In patients with potential occupational hearing loss, additional data must be gathered. The time of first appearance of the symptoms is of interest as is the rate of progression. Detailed historical data regarding noise exposures on all jobs should be explored systematically. The proximity to sources of noise, frequency and duration of exposures, type of work being performed and information on any sound level measurements related to each job all should be noted. Unfortunately these data often are either non-existent or unavailable to the practitioner. In the absence of actual sound measurements, estimates of intensity may be made if the worker experienced symptoms of temporary threshold shift or tinnitus while on a particular job. Likewise, it may be possible to gauge noise levels from other historical information. For example, it is estimated that workers in an 85 dB environment will have to speak loudly, while those in 85–90 dB will have to shout. As noise approaches 95 dB, communication only occurs with shouting even if the workers stand next to each other [Suter, 1986]. Additionally, jobs such as operation of a drop forge, saw mill or chain saw work, metal chipping in foundry work, and certain construction jobs are known to be inherently very noisy. Information on the results of audiometry and on the use of hearing protective devices (HPD) should be obtained for any prior jobs. The physician should review available audiometric records from previous screenings.

Family history of hearing problems, childhood health problems, head trauma, history of ear infection/discharge, and use of antibiotics should be included in the history. Descriptions of hobbies and recreational noise are also key components which must be pursued aggressively and considered in the differential diagnosis of OHL. Information should be sought on other agents that possibly contribute to hearing loss. Questions regarding work or home exposures to vibration, organic solvents, medications, heavy metals or carbon monoxide may be helpful if sufficient detail regarding the intensity and regularity of these exposures can be gathered.

Examination

The examination should include evaluation of the oropharynx, neck, and ears. Neurologic evaluation of the cranial nerves is important, particularly in the case of unilateral hearing deficit such as that caused by an acoustic neuroma. The presence or absence of nystagmus should be noted. The ear canal should be inspected for the presence of any occlusion (cerumen, foreign body, exostoses, etc.). The tympanic membrane may show evidence of previous surgery, perforation, chronic infection, and scarring. Use of a pneumatic bulb can demonstrate the membrane's mobility, which would be decreased with perforation, serous or adhesive otitis, and otosclerosis.

Tests of air conduction and bone conduction can be done with a 512 Hz tuning fork. With the Rinne test, an experienced examiner may pick up losses of air conduction when these are in excess of 25 dB. The demonstration of bone conduction being greater than air conduction suggests a problem in the ear canal or middle ear (*conductive hearing loss*). The Weber test will help to identify unilateral problems and help to confirm the suspicion of conductive loss if the tone is heard best in the affected ear. Conversely, the tone will localize away from the ear in which there is cochlear or eighth nerve dysfunction (*sensorineural loss*).

Laboratory

The gold standard for hearing loss is audiometric testing. Simple pure tone air conduction audiometry has become the foundation for OSHA's approach to surveillance for noise-related occupational hearing loss. This procedure includes measures of hearing ability at multiple frequencies (500, 1000, 2000, 3000, 4000, 6000 and 8000 Hz) in each ear. Care must be taken to limit background noise levels. The audiometers must meet ANSI standards (S3.6–1969) and must be tested for proper function prior to each day's use, with acoustical calibration performed at least yearly. Detailed information on testing is outlined in the OSHA Noise Standard [OSHA, 1983]. Testing at 8000 Hz is not required by OSHA but should be done. Audiometry has become the accepted screening standard because it is non-invasive, relatively inexpensive, and highly reproducible. For these reasons, the examining physician should have a low threshold for ordering audiometry in patients with physical findings suggestive of hearing loss or those who simply have a history which is suggestive of excessive noise or threshold shifts.

Normal function on this screening test is sufficient to rule out any significant hearing deficit. Since temporary threshold shifts related to recent noise exposure can lead to abnormal findings, baseline testing should follow a hiatus of at least 14 h without intense noise exposure. Subsequent testing may or may not continue to observe this 14 h pause. Abnormal function on such testing suggests that more formal audiometric study should be considered. This might be preceded by a repeat simple audiogram following 24 h of strict noise avoidance. If comparison with the worker's baseline audiogram shows an average loss of 10 dB or more over the 2000, 3000 and 4000 Hz range in either ear—a "*standard threshold shift*" (STS)—then notification of the employee is required by the OSHA standard. The finding of an STS is significant and suggests that hearing conservation efforts for the worker be redoubled with further training and re-fitting of HPD's. Additionally referral for full audiologic evaluation or an otologic examination may be appropriate in some of these cases.

A formal audiologic evaluation should include measurement of bone conduction, use of noise masking techniques when indicated, tympanography, and tests of speech perception (speech reception threshold, speech discrimination). This battery of tests can distinguish conductive loss from sensorineural problems. Audiometric patterns may help to distinguish noise-induced problems from other causes of abnormality such as presbycusis and malingering.

Differential Diagnosis

Noise-induced hearing loss (NIHL) is a phenomenon which usually progresses over 10–15 years of intensive noise exposure and then tends to progress more slowly thereafter. It produces a sensorineural defect which evolves over these years. Audiograms show a pattern which is usually bilateral and shows a typical "notch" in the 4000 Hz range on the audiogram (Figure 1). As the loss proceeds, the notch becomes deeper and broader, extending toward both the 2000 and the 8000 Hz range as it begins to seriously affect speech discrimination.

Presbycusis may show a somewhat different pattern on audiogram, but also leads to losses in the higher frequencies. Generally these losses are seen after the age of 60 years. They are greatest at the very highest frequencies leading to a downsloping curve without a notch on audiogram. The distinction of presbycusis from NIHL may be impossible in the setting of combined occupational noise exposure and older age, as the higher frequencies are affected by both.

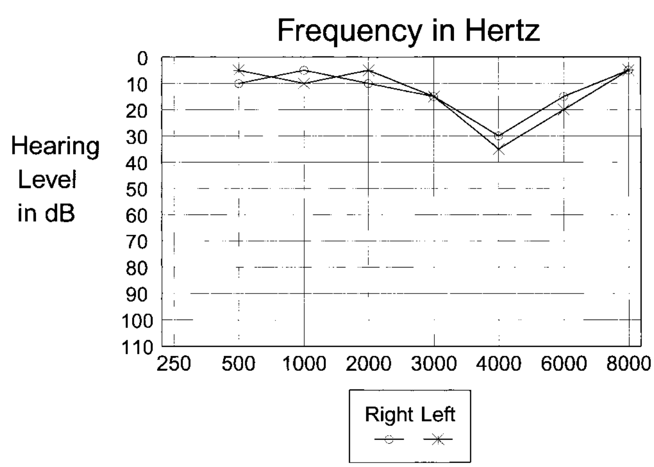

FIGURE 1. Typical audiogram for early NIHL. This audiogram demonstrates the decreased hearing acuity noted early in noise-induced hearing loss. The faintest tone perceived by the subject in each frequency is recorded. The most sensitive hearing level for the right ear is typically depicted with an "o" and for the left ear with an "x". The bilateral symmetry and the decrease in hearing sensitivity at 4000 Hz with recovery of sensitivity at 8000 Hz ("notch") are suggestive of early noise-related losses.

Otosclerosis is a relatively common adult disorder which may lead to conductive, mixed or purely sensorineural patterns of loss. Some types of otosclerosis may be inherited. Some other types of *hereditary hearing loss* may not appear until adult life. When the cause relates to a recessive defect, the family history may not be very helpful.

Metabolic problems such as diabetes, severe lipid abnormalities, hypothyroidism, and other medical problems may lead to slowly progressive, bilateral sensorineural losses.

Sensorineural defects which appear acutely or purely unilateral losses suggest an assortment of etiologies such as viral, vascular, or other causes of inner ear damage:

- a number of *infections* such as meningitis, mumps, lyme disease, syphilis, scarlet fever, typhoid fever and measles can cause bilateral defects.
- *neurologic disorders* such as multiple sclerosis or acoustic neuroma typically have a rapid onset and are unilateral.
- *Meniere's disease* has a more rapid onset than NIHL, tends to be unilateral and may well have audiometric findings of low frequency losses which are atypical for NIHL.
- *head trauma*, ranging from local injury of the external ear to skull fracture commonly produces conductive defects. However, certain types of skull fractures will spare the middle ear but damage either the cochlea or the eighth nerve, producing a sensorineural loss. Certainly some of these injuries (ruptured air hose, other blast injury, welding slag burn, etc.) may be occupational in origin.
- *ototoxic medications* may cause a sensorineural pattern on audiometry which might be mistaken for NIHL. Typically the onset of problems would be more rapid than that seen with NIHL. Prime suspects would include aminoglycoside antibiotics, diuretics, salicylates, and antineoplastic agents (see Table I). Other potentially ototoxic compounds may be encountered in the workplace and should be considered if the exposure can be plausibly related to the hearing loss.

Clearly, a history suggestive of chronicity and findings of bilateral sensorineural involvement can help to rule out a variety of these disorders, leaving presbycusis and otosclerosis as the leading alternative diagnoses to NIHL.

Diagnostic Criteria for Occupational Hearing Loss

The criteria for a diagnosis of hearing loss due to excessive noise are [Sataloff and Sataloff, 1993]:

1. history of intense noise exposure over months to years, with or without associated chemical exposure
2. insidious onset of hearing loss, often in the setting of tinnitus
3. physical exam indicative of sensorineural deficit without any corresponding abnormality of ears, head, and neck
4. audiometric examination with bilateral deficits most prominent in the 3000–4000 Hz range—often with a typical "notch" or upturn at 8000 Hz.

Several other causes of occupational hearing loss have been previously noted. These should be considered at the time that the patient is evaluated for these criteria.

MANAGEMENT

Medical

In many cases, the worker's hearing loss may first become apparent through audiometric screening done for a worksite hearing conservation program. Persistent findings of a standard threshold shift (average shift from a previous baseline audiogram of ≥ 10 dB for the frequencies of 2000, 3000, and 4000 Hz) requires that the employee be notified in writing within 21 days. In light of this standard threshold shift, the employer will have to reassess the employee's training and protection as part of the ongoing hearing conservation program. This finding should stimulate the physician to explore with the employer various other interventions which might benefit the patient and coworkers.

The occupational physician has several additional concerns when an individual with apparent occupational hearing loss is identified. The first is to assure that the diagnosis of occupational loss is correct and that other more remediable or potentially dangerous problems are not present. The American Academy of Otolaryngology–Head and Neck Surgery (AAO–HNS) recommends consideration of referral to an otolaryngologist or audiologist for audiologic criteria which include the following circumstances.

For the baseline audiogram:

1. Average hearing level at 500, 1000, 2000, or 3000 Hz greater than 25 dB in either ear on baseline, or,
2. Asymmetry of hearing levels between the two ears—greater than 15 dB at 500, 1000, or 2000 Hz, or greater than 30 dB at 3000, 4000, or 6000 Hz.

For subsequent periodic audiograms:

1. Decrement from previous baseline with an average of 500, 1000, or 2000 Hz greater than 15 dB, or greater than 20 dB for an average of 3000, 4000, or 6000 Hz.

Additionally, the AAO–HNS recommends referral for medical evaluation in the setting of:

1. Ear pain; drainage; dizziness; severe persistent tinnitus; sudden, fluctuating or rapidly progressive hearing loss; or a feeling of fullness or discomfort in one or both ears within the preceding 12 months, or,
2. Foreign body in the ear canal or cerumen, obstructing the view of the tympanic membrane. [American Academy of Otolaryngology–Head and Neck Surgery, 1997].

In the face of some of these findings, the occupational practitioner may remain confident of the diagnosis of NIHL. However, other findings, particularly asymmetry or rapid progression, may indicate the need for otolaryngological consultation. Similarly, audiometric patterns typical for NIHL might provoke specialty referral.

Another concern is that the worker receives optimal evaluation for rehabilitation to assure that the individual achieves maximal function in the face of permanent losses. Thus once the magnitude of the permanent threshold shift is clear, it is time to consider referral to an audiologist. In addition to the audiologic expertise which may be helpful in assuring the diagnosis of NIHL, assistance should be obtained regarding the potential benefits of a hearing aid. Although the distortion often associated with sensorineural defects presents challenges, hearing aids which selectively amplify the most affected frequencies may assist persons with NIHL to better distinguish speech, even in the presence of background noise. Varying types of aids are available which can help to compensate losses ranging from 25 dB to 110 dB [Sataloff and Sataloff, 1993].

Tinnitus may be present in over half of these patients and for some patients, this can represent a particularly frustrating problem. Options for management of this complaint are limited, though tinnitus maskers may occasionally prove helpful. This is a situation where referral to an audiologist or otolaryngologist should be considered. Measures such as speech-reading classes, amplified telephones, or other devices also may be of importance to the worker. The patient and family should be assisted in dealing with the social impact of the hearing loss. This disorder may generate feelings of isolation and depression among those affected. Spouses and family members may need assistance with feelings of anger and frustration related to the deterioration of communication [Hetu et al., 1995].

Abatement

Unfortunately, persisting threshold shifts documented by audiometry in NIHL represent permanent losses. Once the diagnosis is established, efforts should be directed at the prevention of further loss by evaluating the patient's worksite and providing optimal protection against continuing exposure to high levels of noise. Of highest priority is reduction of exposure to noise, since this does not depend on the vagaries of individual workers' use of protective equipment. As outlined below, consideration must be given to engineering interventions to prevent noise generation or to administrative intervention which might allow the patient to move to a less noisy environment.

Data on area sound pressure levels or preferably, the worker's personal noise dosimetry can indicate the intensity of the exposure and help predict the potential for effective protection with *hearing protective devices* (HPD). Knowledge of the extent of noise exposure, details on the offending frequencies, or the pattern of loss experienced by the worker can aid in the selection of the most appropriate protective devices. Information on the attenuation characteristics of various devices is available from NIOSH [Franks et al., 1994]. It is important to recognize that the level of protection provided by the HPDs may well be overstated. The recommendation from OSHA is that the *noise reduction rating* (NRR) be reduced by 50% after correction for the A filtering of the noise levels (which is NRR−7 dB). Thus one might expect approximately 10 dB of actual protection from a device with an NRR of 27 (27−7 dB for A filtering, times 50% = 10 dB). NIOSH's most recent recommendations also call for derating of the NRR in a fashion dependent on the type of HPD: subtract 25% from the NRR for earmuffs, 50% for slow-recovery formable earplugs and 70% for all other earplugs [NIOSH, 1998]. The patient is unlikely to experience further noise-related losses if the ambient noise level minus the estimated effect of the HPD results in attenuation to less than 85 dB(A), assuming that the HPD is properly used.

There are several other work considerations. The worker must avoid future exposure to any potentially harmful level of noise or of potentially ototoxic compounds which might be in the work environment. Such exposures might be suitably avoided through administrative interventions which actually remove the worker from the exposure or by thoughtful and consistent use of appropriate personal protection. The issue of safety on the job requires some attention. With significant high frequency hearing loss, the worker may not hear high pitched alarms or machinery. Although HPDs have little impact on the ability of normal hearing individuals to function well on the job, the combination of NIHL and sound attenuation produced by HPDs can result in very poor sound perception, which might place the worker at increased risk [Hetu et al., 1995]. All of these issues need review before the worker can safely return to the job.

Prevention

The strategy for prevention of NIHL is well outlined in a variety of sources [Franks et al., 1996; Royster and Royster, 1990; Suter, 1993] as well as the OSHA regulations [OSHA, 1983]. Prevention of occupational loss requires efforts aimed at reducing worker exposure to both injurious impulsive noise as well as chronic excessive noise. Employers should consider a variety of control measures [ILO, 1984], ideally in consultation with an experienced industrial hygienist or noise control expert. Screening for excessive noise can initially be done with a general purpose (type 2) sound level meter (SLM). Noise with unusual frequencies or other characteristics may require further study with other types of SLMs. Employers might be well advised to study the characteristics of the noise more systematically before investing in expensive modifications. Workers who may be consistently exposed to noise in the 85 dB range for periods approaching eight hours should be studied with personal dosimetry, particularly if they are not stationary for much of the day. Once sources of hazardous noise have been identified, the characteristics of the offending noise should be defined so that appropriate noise control procedures may be initiated. Depending upon the situation, these might include purchasing new equipment, modifying existing noisy equipment, and maintaining equipment so that noise is substantially reduced. At times, it is readily evident that loose or worn parts are responsible for noisy machine operation. Isolating noisy processes with barriers or increasing the distance between the source and potentially affected workers can diminish exposure. Measures designed to prevent reverberation and amplification may be quite effective. In any case, it is important to re-measure sound levels after modifications to assure that the goals of noise reduction have been accomplished [Nabelek, 1985].

Those workers still exposed to potentially harmful noise must be identified. The employer needs to provide the employees with annual training regarding noise hazards and effective protection. Employees must be given ready access to at least two types of appropriate HPDs. It is most important that exposed workers be carefully followed with annual audiometric testing performed by trained technicians with properly maintained and calibrated equipment. This screening can be performed by specially trained employees or can be contracted with outside services. Baseline studies should be done following at least 14 h without noise exposure. However some authorities recommend that routine annual tests thereafter be done during the course of the day's work in hopes of detecting employees with temporary threshold shifts [Royster and Royster, 1990]. Subsequent repeat testing of these individuals can help to distinguish temporary from permanent threshold shifts in these workers. In either case such workers will need careful review of their noise exposure and protection practices.

Though not required by OSHA, a basic auditory history of work and recreational exposures may provide important insight into any audiometric finding. Similarly, a few moments spent inspecting the employee's HPD and a brief consultation with the employee at the completion of the audiometry will serve to reinforce the teaching and to better involve the employee in the hearing conservation program.

Evaluation of the individual results and of the overall program are also important components of the process. Abnormal audiograms and any standard threshold shifts must be followed up and promptly referred for further evaluation when indicated. These individuals must be notified of their findings by mail within 21 days. However, notification of all employees of any changes in their results and comparison with age-specific norms is recommended to enhance workers' commitment to the program [Royster and Royster, 1990]. Calculations of the percent of employees with 15 dB worsening in any frequency enables the employer to track the effectiveness of the overall conservation program. Records documenting all of the above must be scrupulously maintained so that intermittent evaluation of the protection program can be carried out [Franks et al., 1996]. A particularly helpful source of information on this is the *Hearing Conservation Manual* from the Council for Accreditation in Occupational Hearing Conservation [Suter, 1993].

COMPENSATION

Since 1953 when New York first proposed specific compensation for noise-induced hearing loss under worker's compensation, recognition of NIHL as a compensable injury has become universal. However, there is considerable diversity among states and federal jurisdictions regarding the criteria used to determine impairment and various considerations which might modify the judgement [ASLHA, 1992]. For example, some states compensate only for total loss in one or both ears while most apply one of several formulas for the calculation of degree of impairment. Adjustment for other factors such as tinnitus or presumed losses due to presbycusis are permitted in certain jurisdictions. Some states deny compensation or penalize workers for failure to use protective devices. A few states reduce the compensation award if the claimant shows improvement with the use of hearing aids. In New York and a minority of other states, a waiting period varying from four days to six months is required prior to filing. Most states have a statute of limitations for filing of claims. The awards usually provide for hearing aids for the worker either as part of the compensation or as a separate award. Since, in the absence of other documentation, the responsibility falls upon the most recent employer, baseline audiometric screening of new employees appears to be a prudent measure. Similarly, employers would be prudent to consider additional audiometry at termination or retirement to avoid being held responsible for losses which might occur subsequently.

REFERENCES

American Academy of Otolaryngology–Head and Neck Surgery Foundation, Inc. 1997. Otologic referral criteria for occupational hearing conservation programs. Alexandria, VA.

American Speech–Language–Hearing Association (ASLHA). 1992. A survey of states' workers' compensation practices for occupational hearing loss. ASLHA 34(March Suppl. 8):2–8.

Fechter LD, Young JS, Carlisle L. 1988. Potentiation of noise-induced threshold shifts and hair cell loss by carbon monoxide. Hearing Res 34:39–48.

Fechter LD. 1995. Combined effects of noise and chemicals. Occup Med State of Art Reviews 10:609–621.

Franks JR, Themann C, Sherris C. 1994. The NIOSH compendium of hearing protection devices. National Institute for Occupational Safety and Health (DHHS-NIOSH Pub. No. 94-130), Cincinnati, OH.

Franks JR, Stephenson MR, Merry CJ. 1996. Preventing occupational hearing loss—A practical guide. DHHS (NIOSH) Publication No. 96-110, p 1.

Hamernik RP, Patterson JH, Turrentine GA, Ahroon WA. 1989. The quantitative relationship between sensory cell loss and hearing thresholds. Hearing Res 38:199–212.

Henderson D. 1989. Effects of noise on hearing. In: Feldman A, Grimes C, editors. Hearing conservation in industry, Robert E. Krieger Publishing, Malabar, FL, p. 15.

Henderson D, Hamernik RP. 1995. Biologic bases of noise-induced hearing loss. Occup Med State of the Art Reviews 10:513–534.

Hetu R, Getty L, Quoc HT. 1995. Impact of occupational hearing loss on the lives of workers. Occup Med State of the Art Reviews 10:495–512.

International Labor Organization (ILO). 1984. Protection of workers against noise and vibration in the working environment. International Labor Office, Geneva.

Johnson AC, Nylen PR. 1995. Effects of industrial solvents on hearing. Occup. Med State of the Art Reviews 10:623–640.

Kryter KD, Ward WD, Miller JD, Eldredge D. 1966. Hazardous exposure to intermittent and steady-state noise. J Acoust Soc Am 39:451–464.

Nabelek I. 1985. Noise measurement and engineering controls. In: Feldman A, Grimes C, editors. Hearing conservation in industry. Malabar, FL: R Krieger Publishing. p 27–76.

National Institutes of Health (NIH). 1990. Noise and hearing loss. NIH Consensus Development Conference Consensus Statement Jan 22–24;8(1).

National Institute for Occupational Safety and Health (NIOSH). 1972. Criteria for a recommended standard—occupational exposure to noise. HSM 73-11001, Washington, DC: US Government Printing Office.

National Institute for Occupational Safety and Health (NIOSH). 1998. Criteria for a recommended standard—occupational exposure to noise. Revised criteria 1998. DHHS (NIOSH) Publ. No. 98-126, Washington, DC: US Government Printing Office.

Occupational Noise Exposure: Hearing Conservation Amendment; Final Rule. 48 Federal Register 9738-9785.

Prince M, Stayner L, Smith R, Gilbert S. 1997. A re-examination of risk estimates from the NIOSH Occupational Noise and Hearing Survey (ONHS). J Acous Soc Am 101:950–963.

Pyyko I, Starck J, Pekarinen J. 1986. Further evidence of a relation between noise-induced permanent threshold shift and vibration-induced digital vasospasms. Am J Otolaryngol 7:391–398.

Rentzsch M, Minks B. 1989. Combined effects of sound, climate and air pollutants on noise-induced hearing loss. Arch Complex Environ Studies 1:41–44.

Royster J, Royster L. 1990. Hearing conservation programs. Chelsea, MI: Lewis Pub. p 61-75.

Sataloff R, Sataloff J. 1993. Occupational hearing loss. New York: Marcel Dekker, Inc. p 403–414, 488–489.

Suter A. 1986. Hearing conservation. In: Berger E, Ward W, Morrill J, Royster L, editors. Noise and hearing conservation manual. Akron, OH: American Industrial hygiene Association, p 7.

Suter A. Hearing conservation manual. 1993. Council for Accreditation in Occupational Hearing Conservation. Milwaukee, WI.

Taylor W, Pearson J, Main A, Burns W. 1965. Study on noise and hearing in jute weaving. J Acoust Soc Am 32:135—137.

U.S. Department of Labor, Occupational Health and Safety Administration (OSHA). 1981. Occupational noise exposure: Hearing conservation amendment. 46 Fed Reg., 4078-4179, 1981.

U.S. Department of Labor, Occupational Safety and Health Administration (OSHA). 1983.