85 Occupational Exposure to Noise

John D. Meyer, MD, MPH *Robert J. McCunney, MS, MD, MPH*

Damage to human hearing from exposure to noise has long been recognized as an occupational hazard. Rammazini (1) described deafness in Venetian coppersmiths, which arose from constant hammering. The advent of the Industrial Revolution and the widespread increase in the use of heavy machinery in manufacturing led to an abrupt rise in prevalence of deafness in a variety of trades, including textile workers and boilermakers (2). Indeed, in this latter group, hearing loss was so common that the condition was referred to as "boilermaker's disease." Hazardous noise levels remain present in a variety of work environments, including military service, manufacturing, construction, transportation, and communications, as well as in leisure-time pursuits, such as music and hunting. Reduction of exposures will reduce or obviate the damage that arises from noise; occupational noise-induced hearing loss (NIHL) thus is typical of many work-related conditions in its susceptibility to preventive measures.

EPIDEMIOLOGY

Data from the 1977 National Health Interview Survey and the National Occupational Hazard Survey indicate that approximately 3.2% of those surveyed had some degree of hearing loss. The proportion of those with hearing loss increased with age; within age groups, rates were consistently greater for those who worked in industries defined as noisy (3). The Occupational Safety and Health Administration (OSHA) has estimated that mild degrees of hearing loss are present in 17% of production workers, while a further 16% have more substantial impairments of hearing. Overall, it is estimated that more than three million workers are affected in the manufacturing sector alone (4). The National Institute for Occupational Safety and Health (NIOSH) has suggested that nearly one in four workers older than 55 years who have been exposed to high noise levels beyond 90 decibels (dB) has some degree of material impairment (5).

Although work duties across a broad range of industries present a risk to hearing, some sectors have a greater proportion of workers at risk for NIHL. In the petroleum, lumber, and food-processing industries, as much as 25% of the workforce may be exposed to levels beyond the OSHA permissible exposure level of 90 dB on an 8-hour time-weighted average. Manufacturing industries, including furniture, metals, rubber, and plastics, also present risks to human hearing if workers are not properly protected from hazardous levels of noise.

Similar occupations at risk are identified in surveillance reports from European sources. The Finnish Register of Occupational Diseases notes an incidence rate for occupational hearing loss of 50.3 per 100,000 workers; this figure most closely approaches the probable true incidence of NIHL, at least in Finland, as it results from mandatory reporting of cases from all physicians in the country (6). Other data sources yield lower figures, as they may be less comprehensive in their covered population or in case definitions of the disorder. Surveillance data based on cases reported separately by audiological and occupational physicians in Great Britain resulted in an estimated annual incidence of NIHL of 1.9 and 1.2 cases per 100,000 workers respectively; it is likely that this is a substantial underestimation, as many workers do not have access to occupational health services (7).

The largest numbers of occupational hearing loss cases in this scheme were reported in armed forces personnel, followed by miners, construction workers, and employees in manufacturing industries.

Risk Factors for Hearing Loss

The major risk factor for NIHL is prolonged unprotected exposure to levels of noise above 85 dB. NIOSH has estimated that the excess risk of hearing impairment after a working lifetime of exposure to an average daily noise level of 85 dB is roughly 8%; this figure jumps to 25% when average exposure increases to 90 dB (8). The decibel scale is logarithmic, and therefore a 3 dB increase represents a doubling of noise intensity. Predictive models of NIHL at higher exposures indicate that hearing damage follows this scale proportionately, although other factors, such as the intermittency of noise, appear to modify the extent of hearing loss in the more extreme ranges.

The cumulative nature of NIHL mandates an awareness of subclinical impairment and the need to consider exposures across a working lifetime. Individual workers may have had exposures to noise in a variety of occupational settings, through past service in the military or reserves, or in community work such as volunteer firefighting. Continued exposure in these settings may accelerate hearing loss, and a history of full- or part-time work in such settings should be obtained when evaluating individuals and worksites. Further complicating the assessment of hearing loss are the avocations and recreation in which workers engage outside of employment; hunting, recreational shooting, metalwork, and music are common activities that may produce significant hearing impairment. Lastly, the decline in acuity produced by presbycusis or age-induced hearing loss can accentuate impairment already present from noise exposure and other factors; from 25% to 40% of people older than 65 years have some degree of hearing loss (3).

Industrial solvent exposures in the workplace may potentiate hearing loss from noise exposure (9). Hearing deficits have been demonstrated in experimental animals exposed to toluene, styrene, xylenes, and trichloroethylene. Solvent abusers, with exposure primarily to toluene, have also demonstrated balance disorders and hearing impairment. Epidemiologic studies of hearing loss in solvent-exposed workers have shown more variable results, possibly because of the role of other factors such as concomitant workplace noise, aging, and smoking. The most consistent effects have been shown for styrene, with indications that in humans, NIHL is potentiated by exposure in the occupational setting (10,11). High-frequency hearing loss has been described in workers exposed to mixed solvents and noise. Several cohorts of workers exposed to solvents in the absence of noise have also been noted to have abnormalities on pure-tone audiometry or on brain stem auditory evoked response testing, indicating an effect on more central pathways of the auditory response (9).

A number of other risk factors have also been proposed, including lipid and cholesterol abnormalities, diabetes, cigarette smoking, and thyroid abnormalities. Smoking may represent both an independent and predisposing factor for NIHL. Office workers in Japan who smoked one pack or more per day had a relative risk for hearing loss that was twice that of nonsmokers, even when controlled for other risk factors (12). Major risk factors in a cohort of noise-exposed white males in an aerospace company were cigarette smoking, a noisy hobby such as shooting, and the number of years worked at a noisy plant (13). The implication of smoking as a risk factor supports the hypothesis that susceptibility to NIHL may be due to relative ischemia of the vasculature of the inner ear.

Type II or adult-onset diabetes may increase the risk of severe hearing loss in those with occupational exposure to noise. Imprecise data, especially regarding the duration and severity of disease, and small sample sizes of workers with insulin-dependent diabetes have hampered attempts to draw a link between it and NIHL (14). Patients with diabetic retinopathy, however, had no greater prevalence of sensorineural hearing impairment than controls (15). The pathogenesis of hearing loss associated with diabetes is not entirely clear but appears to be due to metabolic disturbances that affect nerve function. Despite the possibility of increased risk of NIHL among diabetic patients, scientific evidence does not appear to warrant restriction of diabetic individuals from noisy work if appropriate measures for reducing noise exposure are followed.

Extra-auditory Effects of Noise

The nonauditory effects of environmental noise on human health, most notably hypertension, have also aroused concern. Health effects arising from ambient noise present substantial scientific challenges in study design, implementation, and analysis, particularly with respect to confounding factors, and as such have not yet attracted well-controlled epidemiologic studies. A theoretical basis exists for a proposed relationship between noise and hypertension, grounded in the stress response; as a result of noise exposure, posited release of adrenocortical hormones and sympathomimetic mediators leads to increased heart rate and eventually higher blood pressure. Investigation is made more difficult because the prevalence of both hypertension and presbycusis as well as NIHL increases with age. Cross-sectional studies indicate a correlation of NIHL with high diastolic blood pressure, particularly for those with the most severe hearing loss (16). Longitudinal observation of a mining cohort has, however, failed to show an association between noise exposure and hypertension (17). At this point, the

relationship between the two must be considered as possible but lacking sufficient evidence to draw causal associations. Various hormonal responses have also been described secondary to noise; effects range from increased levels of urinary catecholamines to increased concentration of 17-hydroxycorticoids. Increased postshift urinary cortisol excretion has been noted in workers exposed to high ambient noise levels compared with those wearing hearing protection equipment (18). These findings bolster the hypothesis that noise acts as a general stressor in the setting of normal work demands.

Pregnancy and Noise

Exposure to noise has caused teratogenic effects in laboratory rats, including reduced fertility and enlargement of the ovaries. Results in human studies have been mixed and may be confounded by exposures to stressors other than noise. A case–control study in Finland showed no relationship between occupational noise exposure (greater than 80 dB) and risk of either premature birth or low birth weight, although only 3% of the study group reported any exposure to noise at work during pregnancy (19). An association of noise exposure with low birth weight in a prospective cohort study was noted by the same investigators; these findings were more pronounced in women in standing work positions or performing shiftwork, indicating the possible contribution of other stressful factors on outcome (20). Exposure to noise in utero may affect hearing later in life. In a study of 131 offspring of Quebec women, there was a threefold increase in the risk of high-frequency hearing loss in the children whose mothers were exposed in utero to noise in the range of 85 to 95 dB, and a significant increase in the risk of hearing loss at 4,000 Hz when there was a strong component of low-frequency noise exposure (21).

NOISE AND HEALTH

In occupational medical practice, noise presents three fundamental risks to health:

1. Acutely, through blasts, explosions, or other high-impulse noises that lead to hearing deficits.
2. Chronically, through continued exposure to unsafe levels of noise that lead to sensorineural hearing impairment.
3. Through extra-auditory effects, including alterations in blood pressure and adverse influences on existing illnesses such as hyperlipoproteinemia and diabetes.

Acute Acoustic Trauma

Exposure to sudden intense levels of noise can cause abrupt acute and subsequent permanent damage to the middle and inner ear. Acute acoustic trauma (AAT) may occur in any setting where loud impulsive noise is present, though military operations present the greatest risks for suffering an acute injury to the ear. A survey of World War II casualties indicated that aural injuries accounted for 5.8% of the patients treated at a U.S. military hospital in Paris (22). In the Falklands war, military personnel who operated heavier weapons suffered greater hearing loss than those not so exposed (23). Soldiers operating the heavier artillery, on average, had at least 5 dB loss in each ear at certain frequencies. Blast injuries are particularly difficult to prevent in military operations due to the reluctance of personnel to wear hearing protection devices for fear that they will interfere with communications and place their lives at risk. Unusual explosions have also occurred in certain settings, especially in concert with terrorist activities. One such event in Belfast, Northern Ireland, was described (24). Nearly a year after an explosive blast in a restaurant, 30% of those present suffered from high-frequency sensorineural hearing loss.

In one review of 52 cases of AAT, the most common symptoms were persistent objective hearing loss (95%) and tinnitus (70%) (25). Noise levels of 140 to 160 dB were estimated in most of these cases. Military service accounted for the majority of cases (45%); about one in four had bilateral damage.

Results of audiometric evaluation in AAT may reflect conductive hearing loss secondary to traumatic rupture of the tympanic membrane, disruption of the ossicular chain, and mechanical damage to the oval window as well as sensorineural loss from cochlear hair cell disruption. Higher-frequency pure tone hearing loss is more common in AAT, with frequencies between 4,000 and 8,000 Hz most affected (26,27). A period of weeks to months may be required for hearing to stabilize; the pathologic process resulting in progression of hearing loss from AAT appears not to extend beyond a year unless other factors are present (28). Even if the audiometric results return to normal, however, permanent damage may have occurred to the sensory cells of the inner ear and continued exposure to noise may result in further deterioration of hearing (28). An interesting finding of evaluations of AAT is that most people do not seek medical attention immediately following a blast explosion or traumatic event. It appears that tinnitus, rather than pain or decreased hearing acuity, was the symptom most likely to prompt people to seek a medical evaluation (25,29).

Clinical Evaluation

On physical examination, the ear is usually normal unless the tympanic membrane is ruptured, which occurs in one third of the cases of AAT. Damage to the cochlea, vestibular system, and ossicles of the inner ear can also occur. The diagnostic use of the auditory brain

stem response has been found to be effective in the clinical evaluation of a blast injury to the ear (30). Note that it is not necessarily the ear most proximal or directed toward the blast that sustains the injury, because blast waves may bounce off walls and surrounding objects to cause an injury in the ear not directly exposed to the source.

Complications following such injuries include persistent perforation of the tympanic membrane, permanent hearing loss, and cholesteatoma. About 10% to 20% of tympanic membrane ruptures require surgical correction, with the remainder generally healing without intervention (31). The patient with a persistent perforation should be advised to keep water, foreign bodies, and other potential contaminants out of the external auditory meatus. Large perforations and those that appear not to be healing mandate referral to an otolaryngologist. Relatively little information is available on the extent of occupationally related acute hearing damage that progresses to the sensorineural pattern typical of NIHL, although it has been suggested that progression requires additional noise exposure beyond that which was responsible for the initial injury (32). Tinnitus can persist long term, even in the absence of hearing deficit, in as many as one quarter of individuals incurring AAT and may be the most distressing and disabling effect of these injuries (33).

Treatment

Although prevention of AAT should be emphasized, these injuries can rarely be predicted. Where prevention fails, proper treatment depends on access to medical care. A number of treatment measures have been attempted that are based on the premise that the blast has caused metabolic disturbances in the sensory cells of the inner ear. Evaluation of the effectiveness of medications, however, is impeded by the lack of pre-exposure audiometric values (34).

Evidence for the efficacy of a variety of proposed treatments for AAT remains sparse. No convincing evidence has been noted to support the use of vitamin A, B, or E; nicotinic acid; papaverine hydrochloride; or a number of other substances (35). Dextran has been widely used by the German military with variable results, which may have been in part due to better pretreatment thresholds in the treated subjects (36). Hyperbaric oxygen has similarly been proposed as an effective therapy for tinnitus subsequent to trauma, although review of trials indicates that numbers of subjects are small and that treatment was given following the failure of other standard therapies (37). The strength of claims of efficacy for any of these treatments is difficult to evaluate in light of the absence of controlled, double-blinded, clinical evaluations.

A thorough understanding of the mechanisms of AAT would enhance both prevention and treatment. Animal studies have suggested that certain pathologic features are consistent within species, especially the acute mechanical failure associated with AAT. Consistent findings include separation of the organ of Corti from the basilar membrane and disturbances in function of the tympanic membrane and ossicles (38). In an attempt to understand how various military operations affect the hearing of troops, the U.S. Army sponsored an evaluation of 67 sheep and pigs that were exposed to military operations while they were positioned in an armored vehicle. Tympanic membrane rupture was a consistent finding in the animals, and the authors concluded, "The prevalence and severity of ear drum injury is greater for large anti-armor artillery and that the injury correlated with increasing peak pressure" and therefore blast intensity (39).

Chronic Hearing Loss

A combination of mechanical, metabolic, and vascular factors are involved in the destructive changes that lead to NIHL. The effects of noise occur in the organ of Corti, within the cochlea of the inner ear (Fig. 85.1). This structure has three outer rows and one inner row of hair cells, the sensory receptors of the ear, with the tectorial membrane suspended above them. The hair cells contain cilia that project toward the tectorial membrane. The energy transmitted from the tympanic membrane via the ossicles to the cochlea vibrates the cilia, which convert this mechanical energy into nerve impulses transmitted by the acoustic nerve. These hair cells are highly susceptible to the mechanical trauma of loud noise. The cell bodies swell with repeated exposure to loud noise, and ultimately, the hair cells are destroyed. In addition, high noise levels disrupt the vascular supply of the basilar membrane. Capillary vasoconstriction in response to loud noise may result in reduced oxygen tension and local hypoxia within the cochlea (40). Eventually, the organ of Corti breaks down, with separation of segments of sensory cells from the basilar membrane, leading to elimination of sensory structures and replacement by a single flat cell layer (41). Electron photomicrographs of the cochlea in experimental animals subjected to noise show dropout and progressive destruction of hair cells. Hair cells of the basal turn of the cochlea, which conduct sound at higher frequencies (4,000 to 6,000 Hz), appear to be preferentially affected, most probably due to their location in areas of high shear stress along the organ of Corti. This explains the preferential loss of hearing in this range in early NIHL. Eventually, disruption of the adjacent medial and apical areas occurs as well, leading to hearing loss at a wider range of frequencies. Cochlear blood vessels, the stria vascularis, and nerve endings associated with the hair cells can also be damaged.

Animal investigations have confirmed the mechanisms described above. Edema and swelling of the

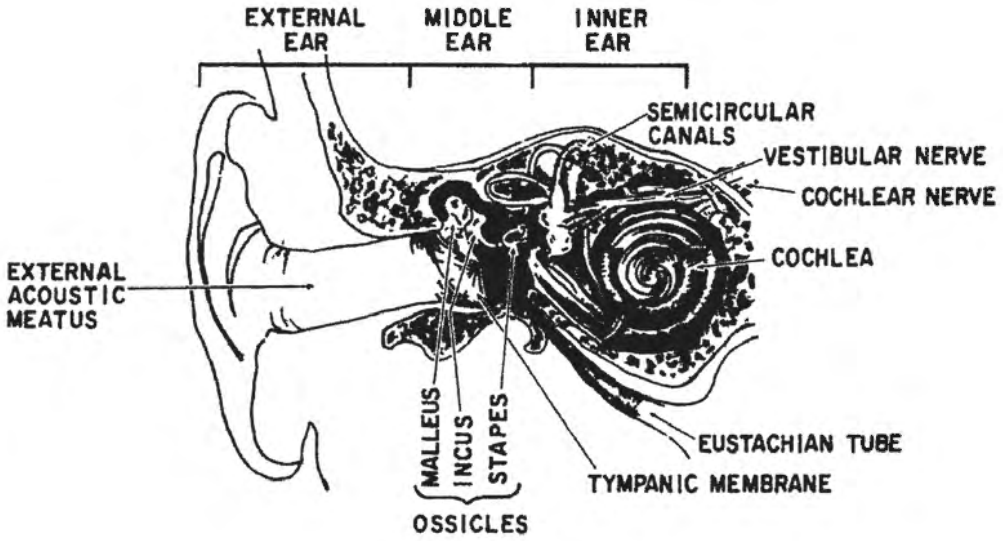


Figure 85.1 Illustration of a cross-section of the human auditory system. High noise levels can damage the tympanic membrane, middle ear conducting system, and sensor cells in the inner ear (cochlea).

afferent nerve endings below the inner hair cells were noted on transmission electron microscopy following noise exposure (42). Following this acute reaction in which the hair cell was distended, a cytoplasmic protrusion occurred, indicating cell damage. Mean cochlear blood flow was much reduced in noise-exposed rats compared to those unexposed (43). An interesting finding of potential clinical application was noted; rats that were spontaneously hypertensive tended to have a greater decrease in blood supply than those that were not hypertensive. This finding may have some relevance in evaluating the extra-auditory health risks associated with noise, such as hypertension. This observed reduction in cochlear blood flow could lead to hypoxia and ultimately disruption in inner ear metabolism. The finding that hypertensive rats were at greater risk for NIHL was confirmed by another study (44). It remains unclear, however, whether the decrease in blood supply associated with impaired hearing is either a primary or secondary pathologic response. Another animal investigation noted vasoconstriction of the cochlear blood vessels in response to exposure to high noise levels (45). These authors also proposed impaired blood flow in the inner ear capillary as the major mechanism leading to NIHL.

Pathologic abnormalities associated with NIHL are distinct from those due to presbycusis. Prolonged noise exposure is associated with disruption of the outer and inner hair cells of the organ of Corti; ultimately, degeneration of nerve fibers and ganglion cells occurs. Presbycusis, by contrast, arises from changes across the entire auditory system, including loss of elasticity of the tympanic membrane and reduction of mobility of the ossicular chain. Loss or malfunction of hair cells in presbycusis initially occurs at higher frequencies (8,000 Hz) than in NIHL.

Although the risk of NIHL tends to increase with advancing age and length of employment, most noise-related effects occur within the early phases of exposure to noise, principally in the first 10 years. Persons with sensorineural hearing loss, however, do not usually recognize early changes in their ability to hear. A study of army helicopter pilots indicated that only one of four who exhibit decrements on audiometric monitoring was aware of any hearing deficit (46). Nonetheless, early changes can usually be documented by audiometric monitoring.

CLINICAL EVALUATION OF HEARING IMPAIRMENT

History and Physical Examination

The physician's role in the clinical evaluation of NIHL is to obtain an objective assessment of hearing impairment, prevent further deterioration of hearing, and recommend patients for further evaluation and treatment. A particular problem in diagnosis is the insidious nature of the injury. The early symptoms of NIHL tend to be subtle and may not be readily recognized by the patient. Initial complaints tend to focus on clarity of sound, particularly speech, rather than its intensity. As NIHL progresses, the person's ability to distinguish softer sounds usually diminishes first. For example, the sounds of birds and other high-frequency sounds such as voices may be difficult to discern. People with high-pitched voices, such as children, may speak in a way that presents difficulties for a person with NIHL. There is difficulty with higher-pitched sibilant consonant sounds, for example distinguishing *fish* from *fist*, or *hat* may become *has*. Speech is rendered less intelligible, as opposed to lower in volume. There will often be a complaint of being unable to understand speech in a noisy room. NIHL rarely, if ever, produces profound deafness,

but the condition tends to be progressive. Hearing handicaps are usually noticed when the threshold hearing level of frequencies in the normal speech range (usually from 500 to 3,000 Hz) averages more than 25 dB.

An overall health history and review of systems should be taken when evaluating suspected NIHL, as other disease entities may cause both conductive and sensorineural hearing loss. The presence of contributory chronic disease including diabetes, hypertension, metabolic disorders, and autoimmune conditions should be ascertained. Past infections including mumps, congenital rubella, and central nervous system infections (meningitis) may affect hearing, as may a history of head injury. The physician should also inquire into past and current medications, particularly those given during hospitalizations. The drugs most commonly associated with deafness include furosemide and aminoglycoside antibiotics such as gentamicin. Analgesics such as salicylates and antihistamines as well as tricyclic antidepressants have also been associated with ototoxicity. Salicylates, in particular, are well known to cause reversible tinnitus. Sensorineural hearing loss may be hereditary as well, and a family history of deafness should be taken. A history of accompanying symptoms, particularly those referable to the inner ear, is useful in the differential diagnosis of hearing loss. Vertigo is often the first symptom of inner ear disorders and, along with decreased acuity and high-pitched tinnitus, may indicate the possibility of an acoustic neuroma. Its presence may also suggest Meniere disease. Vertigo, however, is seldom associated with NIHL or presbycusis.

The diagnosis of NIHL is straightforward when the physician incorporates a clear occupational history of noise exposure with the results of audiometric testing. Evaluation of occupational exposures should include an estimate of years of exposure in conjunction with any information on noise levels in the workplace (Table 85.1). Area survey or individual monitoring data is particularly useful in establishing the exposure history; however, these are infrequently performed, rarely accessible, and may not reflect actual or long-standing exposures. In the absence of such data, a careful description of the processes and equipment used in the workplace may give the evaluating physician a reasonable estimation of exposure. A history describing personal protective equipment and other measures to reduce noise in the workplace should also be taken. The

Table 85.1
Decibel Levels of Noises in Different Environments

	Community	
Industrial and Military	**Outdoor**	**Indoor**
Uncomfortably loud (over 100 dB)		
Diesel engine room (125 dB)	50 hp siren at 100 ft (125 dB)	Live rock-and-roll band (114 dB)
Armored personnel carrier (123 dB)	Thunderclap overhead (120 dB)	
Oxygen torch (121 dB)	Jet plane at ramp (117 dB)	
Scraper-loader (117 dB)	Chain saw (110 dB)	
Compactor (116 dB)	Jet flyover at 1,000 ft (103 dB)	
Riveting machine (110 dB)		
Textile loom (106 dB)		
Electric furnace area (100 dB)		
Loud (80 to 99 dB)		
Farm tractor (98 dB)	Power mower (96 dB)	Inside subway car, 35 mph (95 dB)
Newspaper press (97 dB)	Compressor at 20 ft (94 dB)	Shouted conversation (90 dB)
Cockpit of propeller aircraft (88 dB)	Rock drill at 100 ft (92 dB)	Food blender (88 dB)
Milling machine (85 dB)	Motorcycles at 25 ft (90 dB)	Garbage disposer (80 dB)
Cotton spinning (83 dB)	Propeller aircraft flyover at 1,000 ft (88 dB)	Lathe (81 dB)
Tabulating (80 dB)	Diesel truck, 40 mph at 50 ft (84 dB)	Diesel train, 40 to 50 mph at 100 ft (83 dB)
Moderately loud (60 to 79 dB)		
	Passenger car, 65 mph at 25 ft (77 dB)	Clothes washer (78 dB)
	Auto traffic near freeway (64 dB)	Living room music (76 dB)
	Air-conditioning unit at 20 ft (60 dB)	Dishwasher (75 dB)
		Television (70 dB)
Quiet (40 to 59 dB)		Vacuum cleaner (70 dB)
	Large transformer at 200 ft (58 dB)	Normal conversation (50 dB)
	Light traffic at 100 ft (50 dB)	
Very quiet (20 to 39 dB)		
	Rustling leaves (20 dB)	

contributory effects of solvent exposures should be considered, and an evaluation of concomitant exposures should be made. Physicians evaluating the contribution of workplace noise to hearing loss should also consider nonoccupational causes such as target shooting, motorcycle riding, hunting, loud music, and portable radios. Personal stereos with headphones, for example, are clearly capable of generating sound levels in excess of 85 to 90 dBA standards, although evidence of a contributory effect of these devices on NIHL has not yet been demonstrated (47,48).

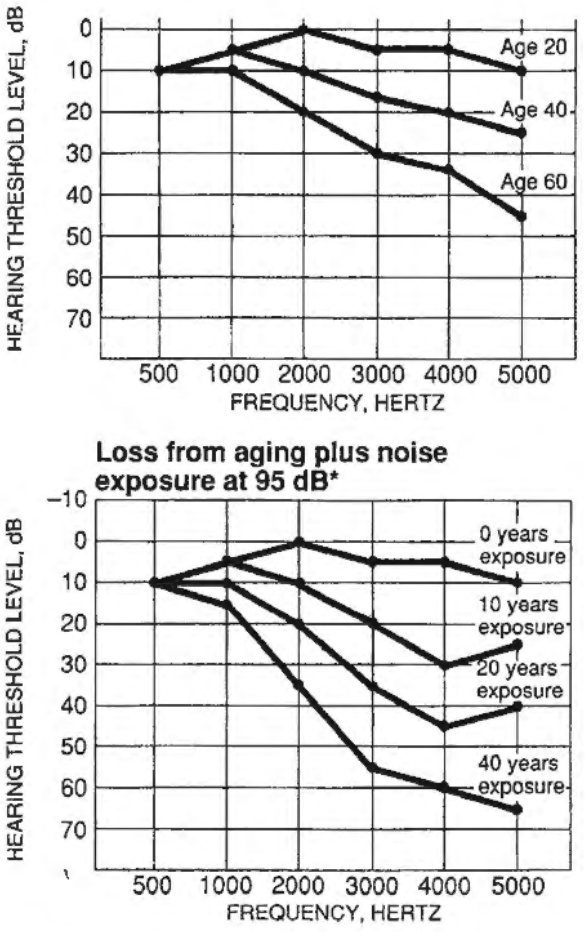
Physical examination should be targeted toward the assessment of the extent and possible contributing causes of hearing loss. Examination of the external meatus should show a canal free from cerumen impaction; if this is noted, the impaction should be removed (generally by irrigation) and audiometry deferred until another day to allow time for the minor trauma of removal to resolve. The tympanic membrane should be examined for signs of scarring or trauma; bulb insufflation may be useful in determining the presence of a persistent middle ear effusion, such as that arising from chronic otitis media. A rapid assessment of hearing may be made using a whispered voice, although results of audiometry will be more informative. Performing Weber and Rinne tests with a tuning fork will assist the examiner in differentiating conductive from sensorineural hearing loss, particularly if loss is unilateral or asymmetric. The remainder of the cranial nerves should be examined, as should coordination, gait, and balance, to evaluate the possibility of neurological disease.

Audiometry and Other Testing

Pure tone audiometric testing, which assesses the ability to hear various standardized frequencies, is the mainstay of evaluation. During the test, tones in the frequency range between 25 and 8,000 Hz are increased in volume until the person recognizes the sound. The decibel reading at which the sound is first recognized is the *hearing threshold* for that frequency. Normal threshold values range from −0.5 dB to 20 dB; those at or above 25 dB are considered abnormal and are especially important when the speech frequency ranges (500 to 4,000 Hz) are affected.

Early impairment due to NIHL tends to occur at 4,000 Hz, with relative sparing of hearing at higher frequencies (Fig. 85.1). These findings are typical of NIHL, though they are not pathognomic, as solvent exposure in the absence of noise may cause a similar pattern. In presbycusis, the audiometric pattern has a similar decrement in the 4,000 Hz range; however, the loss tends to be greater still in the 8,000 Hz range. Audiometric findings of hearing loss due to ototoxicity are similar to those of presbycusis, while those from infections such as mumps will demonstrate equal hearing loss across the spectrum of pure tones. With continued exposure, the 4,000 Hz notch of NIHL will persist and deepen, eventually involving the speech frequencies in the 2,000 to 3,000 Hz range. Despite the contrast in audiometric patterns, differentiating NIHL from presbycusis can be a difficult exercise. Moreover, presbycusis and NIHL can act concurrently to affect hearing. The combination of persistent noise exposure with aging will cause accelerated hearing loss in the higher frequency ranges, and the resultant pattern indicates the additive effects of NIHL and presbycusis (Fig. 85.2).

The finding that a pure tone presented at two unequal frequencies will be subjectively "heard" only on the side of the louder tone forms the basis of the Stenger test to detect malingering that involves claim of unilateral hearing loss. The individual with true unilateral loss will not hear the louder tone in the damaged ear but will indicate instead that he hears the softer tone in the good ear. The malingering patient, by contrast, will localize the sound to the feigned affected ear, as would an individual with normal hearing, and will therefore deny hearing any tone at all.



*The trends are less severe for females.

Figure 85.2 Audiogram results depicting hearing loss from aging and noise.

Other diagnostic and screening tools used to identify hearing impairment and distinguish between differing etiologies have been described. Speech discrimination testing, which assesses the ability to identify words in addition to hearing them, may provide a finer discrimination of impairment than pure tone audiometry. The patient is presented with 50 selected monosyllabic words at the intensity level that audiometry suggests they would be recognized. The proportion of words correctly identified is the speech discrimination score (SDS). Speech discrimination may be affected not only by sensorineural hearing loss at the cochlea but also by abnormalities of the neural pathways along the eighth nerve or in the auditory cortex, which might render easily heard sound unintelligible. Individuals with conductive hearing loss, by contrast, will recognize words as long as they are presented at a sufficient volume. Shorter versions of the SDS, in which words are presented with a competing sound, have been used in workplace screening tests to identify practical difficulties in everyday communication and as a research tool (16).

Additional diagnostic tools used to evaluate hearing loss include brain stem auditory evoked potentials (BAEP), which tracks the brain stem response to auditory stimuli. This test may be especially valuable in assessing persons who report hearing loss but whose audiometric test results are equivocal or nonrevealing and may be particularly useful in the diagnosis of acoustic neuroma and other conditions affecting neural pathways to the auditory cortex.

The evaluation of otoacoustic emissions (OAE), or low-intensity sounds produced by cochlear hair cells either spontaneously or in response to sound, has been useful in some areas of hearing assessment, particularly in newborn screening programs. Their association with the clinical evaluation of NIHL remains at the research stage. Epidemiologic findings of a greater sensitivity of OAEs as compared with pure tone audiometry to hearing loss occurring early in the course of exposure warrant further investigation into their utility as a screening tool in working populations (49).

Threshold Shifts and Changes in Hearing Acuity

The importance of periodic audiometric monitoring in preventing NIHL cannot be overemphasized. Audiometry serves as an effective tool for surveillance if used regularly and properly. Decrements in hearing can occur without being noticed by the worker, especially in the early years of noise exposure. The physician interpreting audiometric results must look for deviations from the baseline values. A threshold shift refers to a significant hearing decrement as documented by audiometry and may be classified as temporary and permanent. While definitions of a threshold shift vary, the most frequently used is the OSHA criterion of a standard threshold shift (STS), which refers to a 10 dB or greater change from baseline for the average of hearing thresholds at 2,000, 3,000, and 4,000 Hz in either ear. Noise-induced temporary threshold shifts (NITTS) are changes in hearing associated with transient overexposures to noise that can be observed and documented by serial audiometry testing. These may persist for hours or even several days, depending on the magnitude and length of exposure to the noise that produced the shift. The person experiencing a NITTS notices diminished hearing acuity that is most pronounced after noise exposure. Retesting after an adequate period of auditory rest will usually demonstrate a return of hearing to baseline values, unless some degree of permanent damage has occurred. Most audiometric examinations should be performed at least 14 hours after the last unprotected exposure to noise to avoid the effect that a NITTS may have on determination of NIHL.

Prolonged and ongoing exposure to hazardous levels of noise may result in permanent damage to hearing. These noise-induced permanent threshold shifts (NIPTS) are irreversible and serve as an important signal that noise levels are not well-controlled. Permanent decrements in hearing may arise even in the absence of documented termporary threshold shifts, and the relationship between the two is not well-defined. Animal experiments suggest that the two may proceed through different mechanisms, with an uncoupling of the outer hair cell stereocilia from the tectorial membrane in NITTS but without overt hair cell damage (50). An employee meeting the OSHA criteria for STS must be retested within 30 days to see if the shift persists. If the increased hearing thresholds persist, the new audiogram, reflecting the STS, is used as a new baseline from which to measure any further hearing decrements. The employee with confirmed STS needs to be informed and evaluated to ensure that hearing protection devices fit properly and are being used as directed. Confirmed threshold shifts with an average decrement of 10 Hz or greater in either ear must be recorded as an occupational illness on the OSHA 300 Log form if the absolute average of hearing loss at 2,000, 3,000, and 4,000 Hz is 25 dB (51).

In some cases, the physician must address the contribution of presbycusis to hearing impairment. The OSHA standard includes recommended calculations to determine the contribution of age to hearing impairment (4). More recently, the validity of applying population-derived statistics to individual audiometric results has been challenged, and NIOSH, in its criteria document for a revised standard, has recommended that audiograms no longer be adjusted to account for the effects of presbycusis (8).

Table 85.2 illustrates a case of progressive, albeit subtle, changes that occurred over a 10-year period,

Table 85.2
Example of Audiometric Thresholds Obtained on Worker Who Exhibited Progressive Noise-Induced Hearing Loss

	Frequency (Hz)[a]											
	Left ear						Right ear					
Thresholds[b] on:	500	1,000	2,000	3,000	4,000	6,000	500	1,000	2,000	3,000	4,000	6,000
Reference	5	0	0	10	10	5	0	5	5	10	10	10
1st annual	0	0	0	10	10	10	5	5	5	10	15	10
2nd annual	5	5	0	10	15	10	0	5	5	15	20	15
3rd annual	0	5	5	15	15	15	5	5	5	15	25	10
4th annual	5	5	10	15	20	20	0	5	5	20	25	15
5th annual	0	5	15	25	30	25	10	10	10	15	25	20
6th annual	5	10	20	35	40	30	10	10	15	20	35	25
7th annual	0	10	30	45	50	40	15	15	20	30	40	35
8th annual	5	15	35	50	55	40	15	20	30	45	55	40
9th annual	10	25	40	60	70	50	15	35	45	55	65	50
10th annual	10	35	55	70	85	60	20	40	50	65	80	55
11th annual	15	40	65	80	95	80	10	45	60	75	90	70

[a]Frequency is a measure of the pitch of a sound and is expressed in Hertz (Hz). Higher frequencies (4,000, 6,000 Hz) are usually first affected in noise-induced hearing impairments.
[b]Thresholds are recorded in decibels (dB), and the quantities shown under frequency indicate the softest intensity level at which the person could hear the different test tones. (Note: 0 dB is audiometric "zero," and deviations from optimum normal are recorded in dB hearing levels greater than 0.)

ultimately leading to serious hearing impairment. This case exemplifies the difference between the clinical diagnosis of hearing loss and findings on screening for early signs of noise-induced hearing impairment. The beginnings of a threshold shift became apparent at the third annual hearing examination and meets criteria for STS by the fourth and fifth year, yet because the capacity to hear and understand speech was not yet compromised, no further measures were made to conserve hearing. The occupational physician, in a preventive role, is charged with recognizing the vitally important role of early changes in hearing, before substantial and irreversible impairment develops.

Some additional points that may assist in the evaluation of suspected NIHL include the following (52):

1. Chronic NIHL is usually symmetric; other otologic disorders, especially the more serious as well as treatable types, are often asymmetric. Localization of hearing deficits may depend upon the specifics of exposure, however. Drivers may present with greater hearing loss in the left ear, as a result of heavier exposures through the window of a truck cab.
2. NIHL usually develops gradually; other otologic disorders may progress rapidly.
3. NIHL usually causes proportionatly more higher-frequency threshold shifts, while hearing loss from other sources, such as infections, may result in more uniform loss across the hearing spectrum.
4. Regardless of the cause, a pure tone threshold average in excess of 25 dB in either ear is likely to cause hearing difficulties.

Disposition and Follow-up

After reviewing diagnostic studies, especially the audiometric evaluation, the physician can formulate an opinion as to the cause of hearing loss and whether therapy may be effective. Unfortunately, treatment measures for NIHL tend to be ineffective, since the primary problem is not amplification of sound but distinguishing various types of sounds. There is considerable difficulty hearing conversation in the presence of background noise and differentiating between competing sounds. Thus, amplification devices that correct other types of hearing impairment by increasing transmission of sound in the middle ear are largely ineffective. Nonetheless, the physician is wise to be aware of the need for otologic referral in evaluating hearing loss, if only to assess potentially remediable causes of hearing loss. The American Academy of Otolaryngology—Head and Neck Surgery has published guidance for otologic referral that applies to most hearing conservation programs (HCP) (53). Indications for referral include a threshold average in excess of 25 dB in either ear on testing at 500, 1,000, 2,000, and 3,000 Hz, a mean difference in acuity between the two ears of 15 dB at lower frequencies (500 to 2,000 Hz), or a 30 dB difference at higher frequencies (3,000 to 6,000 Hz) on baseline audiometry. Referral is also recommended for findings of an average change of 15 dB at the lower frequencies or high-frequency loss of 20 dB noted on periodic examination. In addition, an otolaryngologist should be consulted for other medical problems related to the ear that may be outside the expertise of the occupational physician, including problems

with persistent ear pain, drainage, dizziness, severe persistent tinnitus, or sudden, fluctuating, or rapidly progressive hearing loss not explained by a history of noise exposure alone.

Situations may arise where the occupational physician must recommend restrictions, accomodations, or modifications that affect worker assignment in jobs involving potentially hazardous noise exposures. Findings that may lead to such actions include persons with severe unilateral loss in one one ear with an adequately functioning contralateral ear, persons with moderate to severe hearing impairment that appears to be progressive in spite of efforts to control exposures, persons who continue to demonstrate progressive threshold shifts in hearing that are attributable to causes other than noise exposure, and persons with chronic otologic conditions, including chronic otitis media or otitis externa, who are unable to adequately use or wear hearing protection. Decisions on job placement in these individuals should consider the potential likelihood and severity of further injury as well as work modifications that might reduce the hazard to the worker. To be in compliance with the Americans with Disabilities Act, reasonable accommodations that would allow such potentially disabled employees to continue work must be considered, such as the elimination of job tasks with higher exposure or their reassignment to other workers, depending on individual circumstances.

Determination of Impairment

Evaluation of hearing impairment may be requested in cases where hearing loss has become permanent and irreversible. As with impairment and disability evaluations for other organ systems, the physician is requested to make a determination of impairment based primarily upon testing results, which may be problematic in many cases. Pure-tone audiometry, as noted above, may not reflect handicap based on inability to function in areas of daily activity, including work. Modest decrements in speech recognition, for example, may be severely disabling if fine discrimination of sound is a part of the work. Nonetheless, most approaches to impairment evaluation are founded upon standardized testing. The American Academy of Otolaryngology has published a formula for calculating hearing impairment based on pure tone hearing loss at various frequencies (54). The guidelines assign a 1.5% impairment of monaural hearing for every decibel that the average hearing level (the mean thresholds measured at frequencies of 500, 1,000, 2,000, and 3,000 Hz) exceeds a 25 dB threshold. Impairment does not begin until an average hearing loss of 25 dB has been reached and is considered complete at a threshold average of 92 dB. Provision is made for correction for the effects of aging and presbycusis, although NIOSH has argued against this approach in the individual worker (8). These criteria have been adopted by the American Medical Association in the *Guides to the Evaluation of Permanent Impairment*, in which the sum of thresholds at these four frequencies is used to calculate impairment (55). Additional impairment may be assigned for the presence of tinnitus, although such a determination is necessarily subjective. Evaluation should be made without regard to the use of hearing aids or other assistive devices, as these will not permit an evaluation of the possible extent of impairment. Other approaches have been recommended based upon job-specific functions; one designed for army personnel uses a mathematical model to evaluate a soldier's ability to hear when engaged in certain required or frequently performed tasks (56).

REGULATION AND CONTROL OF NOISE EXPOSURE

Occupational Safety and Health Administration Regulations

A standard to help prevent NIHL in American industries was issued by OSHA in 1983 (4). This regulation requires employers to assess the level of noise in a facility; to reduce noise when it exceeds certain levels; and to provide employees with appropriate medical testing, education, training, and hearing protection devices. The OSHA standard requires employers to implement noise control measures when levels exceed 90 dB [expressed as an 8-hour time-weighted average (TWA_8)] and to establish a HCP when levels are beyond 85 dB. Revised criteria for a recommended standard were published by NIOSH in 1998 based upon its evaluation of the state of the science (8). The NIOSH recommendations differ from current OSHA requirements in their proposal to reduce the permissible 8-hour exposure limit to 85 dB, a level that would be protective of greater numbers of workers based on their estimation of the decreased risk of lifetime hearing loss (8% compared with 25% at 90 dB) at the lower intensity. In addition, NIOSH recommends reduction in exposure by cutting the exchange rate at which exposure time must be halved from the current 5 dB increase to 3 dB, a figure that better reflects the logarithmic scale of noise intensity. At the present time, however, these recommendations have not been promulgated in a new standard, although they represent a scientifically valid approach to hearing protection in the workplace.

Permissible Exposure Limits and Identification of Exposed Employees

According to the current OSHA standard, the permissible daily exposure limit for noise is 90 dB, TWA_8. An exchange rate of 5 dB for every doubling or halving of the exposure time is used to modify the permissible

TWA for louder noise exposures. For example, workers are permitted only a 4-hour exposure to noise at 95 dB and a 2-hour exposure at 100 dB. The ceiling or short-term exposure limit (STEL) is 115 dB for no more than a 15-minute period; this is the maximum value beyond which noise exposure is never permitted. The European Union uses an exchange rate of 3 dB for every halving of the exposure time. This is also the value recommended in the NIOSH revised criteria for a new standard, and has a firmer mathematical foundation because, as a logarithmic measurement, an increase of 3 dB represents a doubling of sound wave pressures.

OSHA requires the employer to institute an HCP when workers are exposed to sound levels at or above the action level of 85 dB TWA_8. Noise exposures in workers must be calculated without regard to the attenuation that may be provided by personal protective equipment. The fundamentals of an HCP include the following measures: noise level assessment, noise control measures, hearing protection devices, audiometric monitoring, and education and training.

Noise Level Assessment

The first step in assessing the need for an HCP is to measure the ambient noise level. Measurements performed in the occupational setting usually consist of overall levels that are obtained either through a sound level meter or a noise dosimeter (57). OSHA requires monitoring of areas that might reasonably be expected to expose employees to noise in order to identify those who need to be enrolled in the HCP or who will need hearing protection. These measurements can also be effective in determining the amount of attenuation required of the hearing-protection devices that may be used. Generally, OSHA allows area surveys to assess individual exposure if the workforce is located in the same general area and the noise levels are relatively uniform throughout the work shift. When area surveys are not appropriate, individual measurements must be made with a personal dosimeter. This particular approach, although capable of yielding more accurate results, tends to be more time consuming and complicated. Accurate measurements depend on reliable calibration of the monitoring device. When area or personal exposure measurements are felt not to give an accurate picture of exposure, it is worthwhile to assess "noise at the ear." An approach to monitoring noise exposure in workers who wear communication headsets has also been introduced (58).

Once noise levels are determined, they need to be re-evaluated at intervals, especially if new processes or plant equipment are introduced into an operation. Periodic measurement may serve as a method of evaluation of the efficacy of preventive controls. The occupational physician or health service should also, if possible, obtain results of noise level assessments, the date of the measurements, and whether they reflect normal operations; these data may prove useful in the clinical evaluation of noise-exposed workers.

Noise Control Measures

In work settings where noise levels exceed 90 dB TWA_8, engineering controls should be employed as the principal measure for noise reduction. Machinery design, enclosure of noisy machinery, installation of sound-absorbent surroundings, and noise control products such as baffles or mufflers can be effective in reducing noise at its source. Improved maintenance of machinery may serve to lessen ambient noise. In most existing settings, noise control measures must be retrofitted onto existing equipment, and such measures should involve participation by engineers, safety personnel, and workers who operate the machinery in order to establish their acceptability and appropriateness (8). A proactive approach to noise reduction is emphasized by the "buy quiet" policies adopted by some corporations. This involves identification and targeting of machinery and processes for noise reduction through new equipment purchases and inclusion of noise level criteria in bidding and purchasing procedures. Despite apparent high initial costs, substantial savings may be realized by using this approach; at its most effective, it may obviate the need for many elements of an HCP, such as personal protective equipment and annual audiometric examinations, if noise levels are reduced below the action level.

Administrative procedures may also become necessary if engineering controls fail to limit noise exposures to acceptable levels. Rotation of workers from exposed to nonexposed areas and limitation of working hours in areas with hazardous levels of noise are the main methods by which these controls are effected. The exchange rate proposed by NIOSH, which halves allowable exposure time for every 3 dB increase in sound intensity, may be used as guidance for administrative reduction of noise exposures.

Hearing Protection Devices

The fundamental approach to reducing the risk of NIHL is to control noise at its source. However, this approach may be inadequate or not feasible, so it is essential to provide hearing protection devices. These are of three basic types: (a) insert, devices placed directly into the ear canal; (b) semi-insert, devices that cover entry into the ear canal; and (c) muffs, which completely encapsulate the ear itself. Hearing protection devices provide various levels of attenuation, usually expressed as a noise reduction rating (NRR) that represents the manufacturer's assessment of testing under optimum conditions. Actual efficacy of these devices in the workplace, however, is dependent on many variables, and attenuation

of noise under normal working conditions may be 25% to 75% of the labeled NRR. Most hearing protection devices provide 15 to 30 dB attenuation if they are fitted properly and used in accordance with their instructions. When insert plugs are combined with muffs, an additional 10 to 15 dB protection can be obtained.

No one type of hearing protection can be considered the single best choice for all users; different workers will choose different devices due to such factors as personal comfort and variations in the anatomic structure of the ear. Thus, it is essential to offer employees a variety of hearing protection devices to ensure that all can comfortably wear them. During the audiometric evaluation, it is worthwhile to acquaint or re-educate the employee in the proper use of the hearing-protection device.

Noise cancellation technology has received increasing interest as a possible means by which high ambient noise levels can be reduced at the ear. Such devices operate by registering immediate noise levels and "blocking" them by generation of a canceling waveform relayed back to the ear. Problems remain with the use of this technology in most workplace settings; it is best adapted to low-frequency noise in confined spaces, where noise usually originates from a single direction. It performs less well in worksites where higher-pitched noise, which presents a greater hazard to hearing, is transmitted from a variety of sources and directions. Its use in the workplace should be considered experimental, and noise cancellation instruments should not be used in place of more generally accepted methods of hearing protection (59).

Audiometric Monitoring

The principles of audiometric testing have been outlined above. Systematic and regular monitoring is essential to preventing NIHL. Periodic audiometric examination is a notable example of an effective screening tool that can reduce the likelihood of occupational illness, because workers with early decrements on audiometric tests usually do not describe hearing difficulties.

Occupational physicians who participate in HCPs are often responsible for interpreting audiometric test results. Although most monitoring is performed in response to OSHA mandates, general principles of medical surveillance apply to these testing programs. Among other tasks the occupational physician must (a) determine the acceptability of the results; (b) assess the results for evidence of alterations in hearing, both for individuals and in aggregate; (c) counsel workers as to the results of testing and recommend additional evaluation for hearing abnormalities; and (d) communicate aggregate results to management, worker representatives, and others with a need to know. Physicians should ensure that the audiometric equipment is properly calibrated according to criteria of the Council for Accreditation in Occupational Hearing Conservation. These guidelines also stipulate training requirements for the person performing the audiometric test, the proper calibration of the audiometer, and the efficacy of the sound control booth. Occupational hearing tests are conducted in many settings, including in the plant, at clinical facilities, and in mobile vans. It is essential that the results be reliable and based on proper testing procedures with well-functioning equipment.

For employees covered under the OSHA standard, baseline audiometric testing is required within 6 months of hire. The OSHA standard mandates yearly audiometric examinations for employees exposed at or above the action level of 85 dB. Periodic test results must be compared to the baseline values. If any abnormalities are noted in this evaluation, the worker should be retested after a 14-hour period without exposure to noise. The presence of an STS, even in the absence of clinical symptoms of hearing loss, should be recorded in the OSHA log (51). More importantly, it should trigger a comprehensive audit of the HCP. An abnormal finding may represent a sentinel event, indicating a failure of primary noise controls or the presence of unanticipated exposures. Reassessment of ambient noise levels and the extent of compliance with the use of hearing protection devices are indicated, both for the affected individual and for coworkers. Although there is no requirement for outside referral in the OSHA regulations, the reviewing or examining physician may find it appropriate to help workers obtain more detailed audiometric evaluations. Records of audiometric testing must be maintained by the employer for the duration of the affected worker's employment.

Although audiometric screening is effective among occupationally exposed groups, this testing has not been valuable as a screening tool in the general population due to the low prevalence of hearing loss in younger cohorts (60).

Education and Training

Under the OSHA standard, education of employees in hearing protection and the adverse effects of exposure to noise must be undertaken in workplaces where ambient noise levels exceed 85 dB. Workers should understand the means by which noise damages hearing and the consequences of prolonged unprotected exposure to high levels of noise. The importance of participation in HCPs and the benefits of wearing hearing protection devices and participating in annual audiometric monitoring programs should be reinforced. The insidious nature of hearing loss tends to encourage a relaxed attitude toward compliance, as the consequences are often not recognized for many years. In order to combat this tendency, employees and supervisors must develop the motivation and discipline to assure the success of a long-term HCP. Employee participation in the planning and development stages can help to

assure that rewards and disciplinary procedures are appropriate and effective and not viewed as a "top-down" effort at control by management (61). Physician participation in educational and training programs designed to acquaint managers and employees with the health implications of long-term exposure to high noise levels can be of great benefit and will send a message that hearing conservation is taken seriously by the occupational health service and the organization. A variety of materials are available for training purposes and may be used to tailor educational programs to the specific needs of the workforce; a useful compendium of films, computer software, and videotapes is available through NIOSH (62).

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EDITED BY

WILLIAM N. ROM, MD, MPH

Sol and Judith Bergstein Professor of Medicine
Director
Division of Pulmonary and Critical Care Medicine
Departments of Medicine and of Environmental Medicine
New York University School of Medicine
New York, New York

ASSOCIATE EDITOR

STEVEN B. MARKOWITZ, MD

Professor and Director
Center for the Biology of Natural Systems
Queens College, City University of New York
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Department of Community and Preventive Medicine
Mount Sinai School of Medicine
New York, New York

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