Occupational hearing loss

Sultan T. Al-Otaibi, *MBBS, FRCPC.*

ABSTRACT

Occupational hearing loss is a common work related problem that can be attributed to an offending agent in the workplace. This paper describes the different causes of occupational hearing loss and its compensation. Physicians should be aware of this preventable medical condition.

Keywords: Occupational, hearing loss.

Saudi Medical Journal 2000; Vol. 21 (6): 523-530

With the increasing complexity of our industrial society, the exposure to chemical and physical agents in the workplace poses a serious threat to the hearing system. In the United States of America (USA), hearing loss affects about 28 million people, 10 million of whom have hearing loss related to noise exposure. The National Institute of Safety and Health (NIOSH) estimate that 14% of workers are exposed to hazardous noise greater than 90 dB(A).[1]

Anatomy of the cochlea. The membranous cochlea is divided into 3 compartments called scalae; the central compartment, scala media, contains endolymph, while the other 2 compartments, scala vestibuli and scala tympani, contain perilymph. Scala media is triangular and its base is known as the basilar membrane. The oblique side of the triangle is called Reissner's membrane (2 cells thick), and the 3rd side is known as stria vascularis (bed of capillaries). The organ of Corti is composed of inner hair cells (one row of cells), and outer hair cells (3 rows of cells) and sits on the basilar membrane of scala media. The hair cells are flanked with nerve fibers (CN VIII) and are in contact with the tectorial membrane. About 95% of auditory nerves terminate in the inner hair cells, while 5% go to the outer hair cells. The hair bundles at the top of hair cells are called stereocilia. (Figure 1).

Occupational hearing loss. The term 'occupational hearing loss' can be misleading because it does not imply difficulty in hearing, but rather, difficulty in understanding speech. Relevant literature on occupational hearing loss was obtained through a Medline search. Information was also located in bibliographic databases such as Toxline, Toxnet, and NIOSHTIC. The literature on occupational hearing loss was reviewed. Occupational hearing loss can be attributed to exposure to offending agents in the workplace, and these include the following:

Chemicals hazards. Organic solvents. These agents cause hearing loss, tinnitus and vertigo, in addition to neuro-behavioral effect. The mechanism of action is thought to be that the solvents can injure sensory cells and peripheral nerve endings of the cochlea, and a retrochoclear action has also been proposed.[2-4] Solvents include the following:
a. Hexane: An organic solvent used in many industrial settings, including shoe factories. Exposure to hexane in rats was found to cause high frequency hearing loss.[5] The same findings were reported in workers chronically exposed to hexane.[6]
b. Xylene: A solvent used in paint, varnish and thinners. Rats exposed to xylene were found to have alteration in auditory function.[7] Workers exposed to xylene did not show any significant hearing loss.[8]
c. Styrene: A solvent used in the production of plastics, rubber and resins. It was found to affect the

From the Dhahran Health Center, Saudi Aramco, Dhahran, Kingdom of Saudi Arabia.

Address correspondence and reprint request to: Dr. S. T. Al-Otaibi, Saudi Aramco, PO Box 11606, Dhahran 31311, Kingdom of Saudi Arabia. Tel. +966 (3) 877-8506. Fax. +966 (3) 877-8841. E-mail: Otaibist@aramco.com.sa

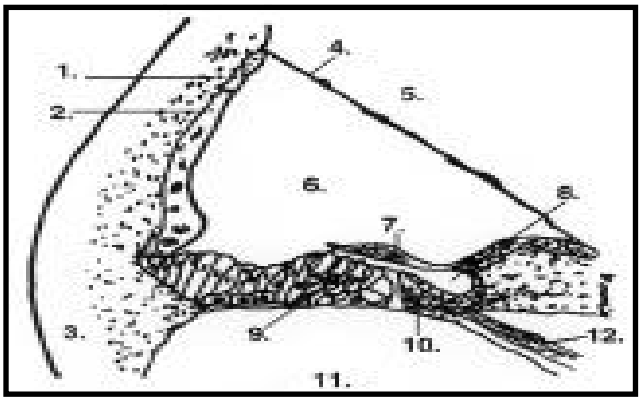

Figure 1 - Normal cochlear anatomy. 1. Spiral ligament. 2. Stria vascularis. 3. Bone. 4. Reissner's Membrane. 5. Scala Vestibuli (perilymph). 6. Scala Media (endolymph). 7. Tectorial Membrane. 8. Inner Sulcus. 9. Outer Hair Cells. 10. Inner Hair Cells. 11. Scala Tympani (Perilymph). 12. Nerve Fibers (CN VIII).

hearing systems in experimental animals but did not appear to have any affect on humans.[3,4,7] d. Toluene: This solvent is used in the manufacture of chemicals, paints, lacquers, rubber and printing materials. Several studies reported high frequency hearing loss in rats exposed to toluene.[9,10] In humans, exposure to toluene caused high frequency sensorineural hearing loss.[11,12] e. Trichloroethylene: A solvent that is used as a grease remover in paints, waxes, in dry cleaning and as an ingredient in other cleaning solutions. Rats exposed to trichloroethylene were found to have mid to high frequency hearing impairment.[13,14] In humans, chronic exposure to trichloroethylene led to bilateral, symmetrical high frequency sensorineural hearing loss with dips at 2 or 3 kHz.[8,15] f. Carbon Disulfide: This is used as a solvent or insecticide and in viscose, rayon and other chemical processes. Animal experiments showed an effect on Brainstem Auditory Evoked Response (BAER) by carbon disulfide which indicates a retrocochlear defect.[16]

Sensorineural hearing loss and associated central vestibular disorders have been reported in workers chronically exposed to carbon disulfide.[17] Both trichloroethylene and carbon disulfide are associated with facial numbness due to their effect on the facial nerve, hence a defect in the stapedial muscle which attenuates up to 30 dB when workers are exposed to noise.[18] In summary, the ototoxic effects of solvents on the auditory system are reported mainly from animal studies and in case reports of substance abusers. Of these solvents, 3 are proven ototoxic (toluene, trichloroethylene and carbon disulfide), and 2 are probably ototoxic in humans (styrene and xylene). Furthermore, chronic occupational exposure to solvents in occupational settings where noise is often present, has an additive toxic effect on hearing.[3,4]

Heavy metals. (i) Lead: Used in batteries, leaded gasoline and others. Lead workers were reported to have vertigo and sensorineural deafness.[19] In animal experiments, lead caused demyelination of the 8th nerve.[20] (ii) Mercury: In 1953, a critical neurological disorder, known as Minamata Disease with severe sequelae leading to death was reported in Japan after the consumption of fish and shellfish contaminated with mercury.[21] The mercury exposure caused hearing loss, ataxia, weakness and sensory changes.[22-24] (iii) Arsenic: Occurs naturally in soils and ores. Animal studies of sodium arsenic caused changes in the organ of corti and stria vascularis of the cochlea. It led also to degeneration of the Reissner's membrane.[25] Hearing loss in humans has been reported from exposure to airborne arsenic.[24,26] (iv) Tin: Used as heat stabilizers for polyvinyl chloride in piping and window casings. Also used as a catalyst for polyurethane foam and rubber. Animal studies have shown that Trimethyltin (TMT) causes damage to the central auditory system while Triethyltin (TET) causes a decreased myelin content in the central nervous system.[27,28] In humans, organotin has been linked to hearing impairment following occupational exposure.[24,29] (v) Manganese used in ferrous metal alloy, electroplating and battery factories. Manganese toxicity in the workplace caused low and high frequency sensorineural deafness that was exacerbated by exposure to noise, as compared to those exposed to manganese alone.[30]

Others. Which include: (i) Carbon monoxide has been reported to cause bilateral sensorineural hearing loss in animal experiments and in humans.[24,31,32] (ii) Butyl Nitrite, which is used as an ingredient in room deodorizers has been reported to cause high frequency hearing loss in rats.[33]

From the above literature review, there is some evidence that chemical exposure in the workplace is associated with hearing loss. Workers exposed to these agents, especially solvents, should therefore be screened audiometrically. Furthermore, epidemiological human studies are needed to explore the effect of these chemicals on the auditory system and to investigate the synergistic effect with noise.

Physical hazards. Noise. Hearing loss as a result of exposure to industrial noise in the coppersmith industry was described by Ramazzini in 1713.[34] A. Type of industrial noise: (i) Impulse noise occurs most commonly from gunfire and by the banging of metal on metal objects. Here noise ranges from 100 to 140 dB. This type of noise causes direct damage to the organ of Corti and tympanic membrane. (ii) Continuous noise is more common in industry than is impulse noise. One example is the noise emitted from a turbine engine. Industries in which there are dangerous noise levels include underground mining, oil drilling, paper, food, textile, rubber, plastic and utility industries. Impulse noise produces a permanent threshold shift at 4 and 6

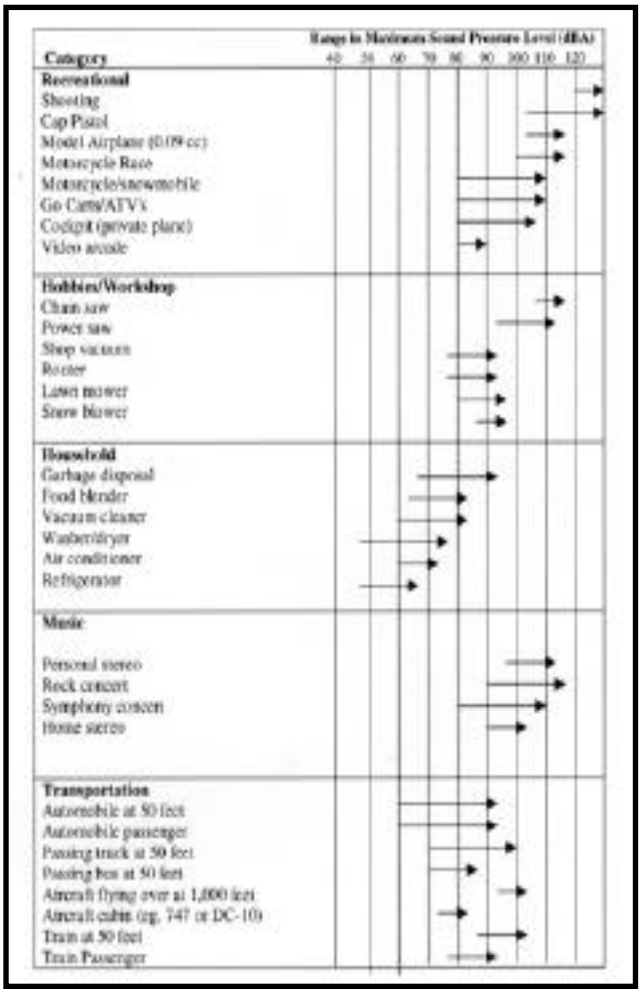

Figure 2 - Range of noise in non-occupational item (adopted from reference 38).

kHz after a shorter duration of exposure than continuous noise.[35] Combined exposure to steady, continuous industrial noise and impulse noise does not increase the risk of Noise Induce Hearing Loss (NIHL) as long as neither exceeds 85 dB.[36,37]

Non-occupational noise exposure: This includes hunting and target shooting (causing asymmetrical NIHL whereby a right-handed person will have left ear sensorineural hearing loss), chain saw use, motorcycles, racing cars, speed boats, and loud music (especially at rock concerts).[38,39] The range of noise in non-occupational items is shown in Figure 2.

B. Individual susceptibility to NIHL: I. Non-auditory factors: a. Age: extreme age (older and neonates) at risk of NIHL.[40,41] b. Gender: are men at higher risk of NIHL than women?[42] c. Eye color: are blue-eyed people more susceptible to NIHL?[42] d. Smoking: has been found to increase NIHL, most likely due to CO in smoke. On the other hand, smoking is reported to increase NIHL in conjunction with noise exposure.[43] e. Health status: Hypertension, heart disease, diabetes mellitus, hypolipoproteinemia, hypercholesterolinemia and hyperlipidemia will increase the vulnerability of the cochlea to noise.[37,42,44-46] II. Auditory factors: 1. Acoustic reflex: The muscle of the middle ear plays a protective role against loud sound by attenuating it during speech production. Patients with Bell's palsy, therefore, are at a higher risk for developing NIHL.[42,47] 2. Efferent auditory nervous system: Some investigators believe that activation of the efferent system causes an inhibition or reduction of the 8th nerve response. However, this role is still not clear and further studies are needed to explain its relation to NIHL.[42] 3. Previous history of noise exposure: Believed to increase individual susceptibility to NIHL depending on the level and frequency of exposure.[42] 4. Outer ear resonance: Has been shown to play a role in the development of high frequency (4 kHz dip) hearing loss.[48]

C. Mechanism of NIHL depends upon the level of noise exposure as follows:[39,49] 1. Mechanical damage: If noise exceeds 140 dB, such as in gunfire and detonation of explosives, it causes direct damage to the hair cells and tearing of the delicate basilar membrane. The organ of Corti is replaced afterwards by a single layer of squamous epithelial tissue. Metabolic damage: If noise is between 90 and 140 dB, metabolic damage develops slowly over years of exposure. Here, sensory cells are killed by noise through metabolic and electrolyte disturbance. The outer hair cells are affected first followed by the inner hair cells. The cells do not regenerate, but are replaced by scar tissue. Blood vessels, secretory cells and nerve cells are also damaged by exposure to loud noise.

D. Interaction of noise and other oto-traumatic agents: 1. Aminoglycoside antibiotics: include kanamycin and gentamicin. The ototoxicity mechanism is due to damage to sensory hair cells and stria vascularis of the cochlea.[50] Animal and human studies revealed a positive interaction between exposure to noise and aminoglycoside antibiotics.[36,51] 2. Loop inhibiting diuretics: include frusemide and ethacrynic acid. The ototoxicity mechanism at stria vascularis causes edema of the marginal cells, but does not damage hair cells.[36] It is believed there is no interaction between loop inhibiting diuretics and exposure to noise, because noise affects the cochlea at hair cell level.[36] 3. Salicylate: The ototoxicity mechanism damages the mitochondria of cells of stria vascularis. Also, the metabolic mechanism, through inhibition of prostaglandin synthesis, affects electrolyte balance of the cochlear fluids.[36,52] If salicylate is taken concurrently with noise, there seems to be no interaction because of the different actions of both at the cochlea. Generally, the data available in this issue is contradictory.[36] 4. Cisplatinum: The

ototoxicity mechanism through the stria vascularis, also affects hair cells as is the case with diuretics, such as aminoglycoside.[53,54] Most studies indicate that individuals on chemotherapy are at increased risk of developing NIHL when exposed to noise.[36] 5. Vibration: Most studies report that vibration alone does not affect hearing.[36] However, exposure to a combination of vibration and noise in the workplace increases the risk of NIHL among workers exposed to both offending agents. This interaction between noise and vibration is more commonly reported with whole body vibration.[55,56] 6. Solvents: Styrene and toluene have been shown to have a synergetic effect when associated with noise in animal studies.[3,4,9,10]

E. Health effects of noise: I. Non-auditory: Possible hypertension, heart disease, and deafness in children of pregnant women exposed to noise. The evidence in epidemiological studies is not strong. Other effects of noise include sleep disturbance, psychological effect (annoyance, irritability, fear), higher accident rates, and lack of communication between workers. Noise is a factor of annoyance in the industrial workplace which precludes human capabilities and work satisfaction.[57-60] II. Auditory: 1. Acoustic trauma: Sudden loss of hearing by an intense single incident noise such as a blast or an explosion, which can also result from a non-noise cause such as diving when there is no noise exposure. It leads to conductive, sensorineural or mixed type deafness. Sensorineural deafness results from mechanical damage to the organ of Corti.[39,61] Acoustic trauma is reported to be associated with Meniere's disease.[62,63] 2. Temporary threshold shift (TTS): Temporary hearing reduction of 10dB at high frequency 3000-6000 Hz after noise exposure. It occurs at the end of each workday and at weekends. Symptoms resolve after removal from noise, and hearing recovers within hours.[39,64,65] The mechanism of action is believed to be due to fatigability of the organ of Corti following noise exposure.[49] In animal experiments, chronic TTS shows no abnormalities of the sterocilia, while in permanent threshold shift, a complete absence of the organ of Corti was reported and this determines the reversibility of the threshold shift.[66] Temporary threshold shift can progress to permanent threshold shift (PTS) if noise exposure continues, but cannot be used to predict the risk of PTS from TTS.[42] 3. Permanent Threshold shift: Permanent and irreversible sensorineural hearing loss after repeated exposure to loud noise over a number of years. It is usually bilateral, symmetrical and accompanied by high frequency tinnitus. It occurs gradually and the patient is unaware of any hearing loss until it involves speech comprehension. It is believed that NIHL does not progress after removal from further noise exposure.[39] 4. Tinnitus: A high frequency ringing sound, frequently accompanying NIHL. It is a subjective complaint that can be intermittent or continuous, and increased by further exposure to noise. It is more pronounced in quiet environments and can interfere with sleep.[39]

Characteristics of NIHL: The American College of Occupational and Environmental Medicine have published criteria to aid in diagnosing NIHL as follows:[67] 1. It is always sensorineural, affecting the hair cells of the inner ear. 2. It is almost always bilateral. Audiometric patterns are usually similar. 3. It almost never produces a profound hearing loss. Low frequency limits are usually about 40 dB and high frequency limits about 75 dB. 4. Once the exposure to noise is discontinued, there is no further substantial progression of hearing loss as a result of noise exposure. 5. Previous NIHL does not make the ear more sensitive to future noise exposure. As the hearing threshold increases, the rate of loss decreases. 6. The earliest damage to the inner ear reflects a loss at 3000, 4000, and 6000 Hz. There is always far more loss at 3000, 4000, 6000 Hz than at 500, 1000, and 2000 Hz. The greatest loss usually occurs at 4000 Hz. The higher and lower frequencies take longer to be affected than in the range 3000 to 6000 Hz. 7. Given stable exposure conditions, losses at 3000, 4000 and 6000 Hz will usually reach a maximal level in about 10 to 15 years. 8. Continuous noise exposure over the years is more damaging than interrupted exposure to noise that permits the ear to have a rest period.

4000 Hz Audiometric Dip: Permanent hearing loss occurs in frequencies centered at 4000 Hz despite the difference in spectral and temporal characteristics of noise. The mechanism is unclear but is believed due to the outer ear properties in people exposed to noise.[48]

Other causes of 4 kHz notch are as follows: 1. Viral infection: Viral URTI, rubella, measles, CMV and herpes virus. 2. Skull trauma. 3. Hereditary deafness. 4. Ototoxicity; aminoglycoside, diuretics, Acoustical Society of America (ASA) and Cisplatinum. 5. Acoustic neuroma. 6. Unknown. 7. Multiple sclerosis. 8. Bacterial meningitis. 9. Neonatal Rh incompatibility. 10. Presbycusis

Asymmetrical NIHL:[37] 1. If the worker is right-handed or left-handed and tries to adjust his/her position. 2. Truck drivers. 3. Military personnel (rifle shooting). Non-occupational causes of hearing loss, such as acoustic neuroma, must be excluded among these workers.

Causes of bilateral sensorineural hearing loss: 1. Presbycusis (the most common cause of sensorineural deafness). 2. The sensorineural aspect of otosclerosis. 3. Effects of hearing aid amplification. 4. Unknown cause. 5. Ototoxic drugs. 6. Heredity. 7. Others: head trauma, viral infection, Meniere's disease and non-organic hearing loss. 8. Non-occupational noise exposure e.g., hunting and target shooting.

Table 1 - Hearing disability in relation to hearing threshold level (adopted from reference 69).

Hearing threshold level (dB ANSI)	% hearing disability	Hearing threshold level (dB ASA)
More than 26 to 31	5	More than 16 to 21
More than 31 to 36	10	More than 21 to 26
More than 36 to 41	15	More than 26 to 31
More than 41 to 46	25	More than 31 to 36
More than 46 to 51	35	More than 36 to 41
More than 51 to 56	50	More than 41 to 46
More than 56 to 66	70	More than 46 to 56
More than 66 to 76	90	More than 56 to 66
Over 76	100	Over 66
ANSI -		ASA -

Table 2 - Duration of noise exposure as measured by sound level meter in Canada (adopted from reference 71).

Column 1 Sound level in decibels	Column 2 Duration - hours per 24 hour day
90	8
92	6
95	4
97	2
100	2
102	1.5
105	1
110	0.5
115	0.25 or less
Over 115	No exposure

Pressure changes. In diving and aviation, a defect in eustachian tube function leads to conductive deafness (middle ear serous and infected otitis media), also barotrauma of the inner ear with rupture of the round and oval windows, and inner ear decompression sickness (nitrogen bubble in endolymph or perilymph).[23,68]

Hearing Loss Compensation. A. USA: This is based on American Medical Association (AMA) guidelines for the evaluation of permanent impairment.[69,70] To calculate hearing impairment, use audiometers calibrated to American National Standards Institute (ANSI) specifications.

To determine decibels of hearing at 500, 1000, 2000 and 3000 Hertz: If the hearing loss is less than 25 dB at these frequencies, there is no impairment. If it is greater than 76 dB, then the impairment is considered to be 100% as shown in Table 1. In this case, add the decibels determined for the 4 frequencies in each ear separately.

Example: Sample audiogram and calculation of impairment.

	500	1000	2000	3000	4000	6000	8000
Right ear (dB)	25	35	35	45	50	60	45
Left ear (dB)	25	35	40	50	60	70	50

Unilateral Impairment: (Average dB at 500, 1000, 2000 and 3000 Hz) - 25 dB (low fence) x 1.5% = percentage of unilateral impairment. e.g., Right ear = (25+35+35+45 divided by 4) - 25 x 1.5% = 15% Left ear = (25+35+40+50 divided by 4) - 25 x 1.5% = 18.8%.

Bilateral Impairment: (Percentage of unilateral impairment in better ear x 5) + (percentage of unilateral impairment in poorer ear) divided by 6 = percentage of bilateral impairment. e.g. (15 x 5) + (18.8%) divided by 6 = 15.6%

Using the decibel sum of the hearing threshold levels, determine the impairment loss on Table 1, page 225, AMA Guidelines. Determine the binaural impairment by plotting the worst ear loss against the better ear on Table 2, page 226, AMA Guidelines. Use Table 3, page 228, AMA Guidelines, to convert this loss to a whole person rating. According to the AMA, tinnitus accompanied by NIHL is compensated as indicated on page 228.

The Occupational and Safety Health Association (OSHA) of the USA requires that when noise levels are more than 85 dB, a hearing conservation program be implemented as follows: 1. Monitoring to assess and record noise levels. 2. Periodic audiometry. 3. Noise control. 4. Education and record keeping.

B. Canada: Similar to the USA, Canada adopted the AMA guide for NIHL and its accompanying tinnitus (up to 5% of impairment attributed to tinnitus in Ontario and Alberta if it had been present for more than 2 years), to evaluate hearing impairment.[71] Some differences in the provinces of Canada are as follows: 1. The length of time that workers were not exposed to noise prior to the hearing for pension varies, e.g., in Ontario 48 hours, British Columbia (BC) 14 hours, Yukon 1 month. 2. There were no guidelines as to who should perform hearing tests in Ontario. 3. The award size

Table 3 - Occupations covered by prescribed disease in the UK (adopted from reference 72).

Any occupation involving:
- The use of powered grinding tools on cast metal, or on billets or blooms.
- The use of pneumatic percussive tools on metal.
- The use of pneumatic percussive tools for drilling rock in quarries, underground, or in mining coal.
- Work wholly or mainly in the immediate vicinity of a plant engaged in forging.
- The use of machines engaged in weaving fibers or high-speed false twisting of fibers.
- The use of machines engaged in cutting, shaping, or cleaning nails.
- The use of specific machines engaged in the working of wood and circular sawing machines.
- The use of chain saws in forestry.

differed from province to province; in BC the award on average was given for 500, 1000, 2000 Hz hearing loss, while in other provinces for 500, 1000, 2000, 3000 Hz hearing loss. 4. British Columbia judges the better ear 4:1, while in other provinces, the better ear is judged at 5:1. 5. Alberta, Newfoundland, and Nova Scotia have not applied presbycosis correction, while others provinces have 0.5% dB/year above the age of 60.

Noise regulations, per se, are not available in Ontario, so the Workers' Compensation Board (WCB) has it's own policy. The Occupational Health and Safety Act and Regulations for Industrial Establishments of Canada require that the daily noise exposure for a particular sound should not exceed the specified levels, as described in Table 2. In addition, hearing protection must be worn when the daily exposure is more than that permitted for the particular sound level. Where hearing protection is required in Canada, the protection shall be sufficient to reduce the sound level below that in column 1. No periodic audiometry screening is required for workers according to the act.

Table 4 - Calculation of hearing disability as per UK regulations (adopted from reference 72).

dB Hearing loss (averaged 1, and 3 kHz)	**Disability %**
50 - 53	20
54 - 60	30
61 - 66	40
67 - 72	50
73 - 79	60
80 - 86	70
87 - 95	80
96 - 100	90
106	100

C. United Kingdom (UK): The UK follows a different system specified in the legislation as NIHL Prescribed Diseases, as shown in Table 3. According to UK regulations, sensorineural hearing loss amounts to at least 50 dB in each ear, being the average hearing loss at 1, 2 and 3 kHz frequencies, causing deafness due to occupational noise in at least one ear.[72] The disability is calculated according to Table 4. The industrial disability benefit rate is calculated according to a specific table. The UK has noise regulations similar to OSHA regulations. United Kingdom legislation is very rigid when it comes to compensation, and certain occupations are excluded, such as military personnel. Eligibility for compensation requires longtime employment and workers should apply within 5 years of ceasing work. No compensation is available for tinnitus.

D. Saudi Arabia: Noise induced hearing loss is compensated by the General Organization for Social Insurance (GOSI). This includes deafness of various degrees as a result of all operations, occupations and industries which are associated with the generation of noise and loud sounds that are liable to affect hearing.[73]

The percentage of Binaural hearing impairment derived as follow: 1. First calculate the monaural impairment for each ear: a. Average threshold values of 500, 1k, 2k and 3k. b. Subtract 25 from the average. c. Multiply reminder (if > 0) by 1.5. Calculate the percentage binaural loss as follows: (5 x % of loss of better ear + % of loss of poorer ear) / 6.

References

1. Rees T, Duckert L. Hearing loss and other otic disorders. In: Textbook of Clinical Occupational and Environmental Medicine. 1st ed. Toronto: WB Saunders; 1994.
2. Barregard L, Axelsson A. Is there an ototraumatic interaction between noise and solvents? Scand Audiol 1984; 13: 151-155.
3. Morata TC, Dunn DE, Kretschmer LW, Lemasters GK, Keith RW. Effects of occupational exposure to organic solvents and noise on hearing. Scand J Work Environ Health 1993; 19: 245-254.
4. Morata TC, Dunn DE, Sieber WK. Occupational exposure to noise and ototoxic organic solvents. Arch Environ Health 1994; 49: 359-365.
5. Rebert CS, Houghton PW, Howd RA, Pryor GT. Effects of hexane on the brainstem auditory response and caudal nerve action potential. Neurobehav Toxicol Teratol 1982; 4: 79-85.
6. Huang CC, Chu NS. Evoked potentials in chronic n-hexane intoxication. Clin Electroencephalogr 1989; 20: 162-168.
7. Pryor GT, Rebert CS, Howd RA. Hearing loss in rats caused by inhalation of mixed xylenes and styrene. J Appl Toxicol 1987; 7: 55-61.
8. Rybak LP. Hearing: the effects of chemicals. Otolarngol Head Neck Surg 1992; 106: 667-686.

9. Rebert CS, Sorenson SS, Howd RA, Pryor GT. Toluene-induced hearing loss in rats evidenced by the brainstem auditory-evoked response. Neurobehav Toxicol Teratol 1983; 5: 59-62.
10. Pryor GT, Dickinson J, Howd RA, Rebert CS. Transient cognitive deficits and high-frequency hearing loss in weanling rats exposed to toluene. Neurobehav Toxicol Teratol 1983; 5: 53-57.
11. Benignus VA. Neurobehavioral effects of toluene: a review. Neurobehav Toxicol Teratol 1981; 3: 407-415.
12. Metrick SA, Brenner RP. Abnormal brainstem auditory-evoked potentials in chronic paint sniffers. Ann Neurol 1982; 12: 553-556.
13. Jasper RM, Muijser H, Lammers JH, Kulig BM. Mid-frequency hearing loss and reduction of acoustic startle responding in rats following trichloroethyelene exposure. Neurotoxicol Teratol 1993; 15; 407-412.
14. Rebert CS, Day VL, Matteucci MJ, Pryor GT. Sensory-evoked potentials in rats chronically exposed to trichloroethyelene: predominant auditory dysfunction. Neurotoxicol Teratol 1991; 13: 83-90.
15. Szulc-Kuberska J, Tronczynska J, Latkowski B. Oto-neurological investigation of chronic trichloroethylene poisoning. Minerva Otorinolaringol 1976; 26: 108-112.
16. Rebert C, Becker E. Effect of inhaled carbon disulfide on sensory-evoked potentials of Long-Evans rats. Neurobehav Toxicol Teratol 1986; 8: 533-541.
17. Magos L. The clinical and experimental aspects of carbon disulphide intoxication. Rev Environ Health 1975; 2: 65-80.
18. Phaneuf R, Hetu R. An epidemiological perspective of the causes of hearing loss among industrial workers. Otolaryng 1990; 19: 31-40.
19. Ciurlo E, Ottoboni A. Variation of the internal ear in chronic lead poisoning. Meinerva Otolaringol 1955; 5: 130-132.
20. Gozdzik T, Moszynski B. Eighth nerve in experimental lead poisoning. Acta Otolaryngol, 1969; 68: 85-89.
21. Mizukoshi K, Watanabe Y, Kobayashi H, Nakano Y, Koide C, Inomata S et al. Neurotological follow up studies upon Minamata disease. Acta otolaryngol Suppl 1989; 468: 353-357.
22. Gerstner HB, Huff JE. Clinical toxicology of mercury. J Toxicol Environ Health 1977; 2: 491-526.
23. Shusterman DJ, Sheedy JE. Occupational and environmental disorders of the special senses. Occupational Medicine 1992; 7: 515-542.
24. Hetu R, Phaneuf R, Marien C. Non-acoustic environmental factor influences on occupational hearing impairment: a preliminary discussion paper. Canad Acoustics 1987; 15: 17B-31.
25. Anniko M, Wersall J. Damage to the stria vascularis in the guinea pig by acute atoxyl intoxication. Acta Otolaryngol 1975; 80: 167-179.
26. Bencko V, Symon K. Health aspects of burning coal with a high arsenic content. I. Arsenic in hair, urine and blood in children residing in a polluted area. Environ Res 1977; 13: 386-395.
27. Ruppert PH, Dean KF, Reiter LW. Trimethyltin disrupts acoustic startle responding in adult rats. Toxicol Lett 1984; 22: 33-38.
28. Amochaev A, Johnson RC, Salamy A, Shah BN. Brain stem auditory evoked potentials and myelin changes in triethyltin induced edema in young rats. Exp Neurol 1979; 66: 629-635.
29. Besser P, Kramer G, Thumler R, Bohl J, Gutmann L, Hopf HC. Acute trimethyltin limbic cerebellar syndrome. Neurology 1987; 37: 945-950.
30. Nikolov Z. Hearing reduction caused by manganese and noise. J Fr Otorhinolaringol Audiophonol Chir Maxillofac 1974; 23: 231-234.
31. Fechter LD, Thorne PR, Nuttall AL. Effects of carbon monoxide on cochlear electrophysiology and blood flow. Hear Res 1987; 27: 37-45.
32. Wright G, Shepard R. Carbon monoxide exposure and auditory duration discrimination. Arch Environ Health 1978; 33: 226-235.
33. Fechter LD, Richard CL, Mungekar M, Gomez J, Starthern D. Disruption of auditory function by acute administration of 'room odorizer' containing butyl nitrate in rats. Fundam Appl Toxicol 1989; 12: 56-61.
34. Ramazzini B. Diseases of worker. Translation of Latin text. New York: Hafner; 1964.
35. Mantysalo S, Vuori J. Effects of impulse noise and continuous steady state noise on hearing. Br J Industr Med 1984; 41: 122-132.
36. Boettcher FA, Henderson D, Gratton MA, Danielson RW, Byrne CD. Synergistic interactions of noise and other ototraumatic agents. Ear Hear 1987; 8: 192-212.
37. Touma JB. Controversies in noise-induced hearing loss (NIHL): Review. Ann Occup Hyg 1992; 36: 199-209.
38. Clark W. Noise exposure from leisure activities: review. J Acoust Soc Am 1991; 90: 175-181.
39. Clark WW. Hearing: the effects of noise. Otolaryngol Head Neck Surg 1992; 106: 669-676.
40. Lalande NM, Hetu R, Lambert J. Is occupational noise exposure during pregnancy a risk factor of damage to the auditory system of the fetus? Am J Ind Med 1986; 10: 427-435.
41. Pyykko I, Pekkarian J, Stark J. Sensory-neural hearing loss during combined noise and vibration exposure. An analysis of risk factors. Int Arch Occup Environ Health 1987; 59: 439-454.
42. Henderson D, Subramanian, Boettcher FA. Individual susceptibility to noise-induced hearing loss: an old topic revisited. Ear Hear 1993; 14: 152-168.
43. Barone JA, Peters JM, Garabrant DH, Bernstein L, Krebsbach R. Smoking as a risk factor in noise-induced hearing loss. J Occup Med 1987; 29: 741-745.
44. Touma JB. Noise and hearing - Review. W V Med J 1994; 90: 327-329.
45. Susmano A, Rosenbush SW. Hearing loss and ischemic heart disease. Am J Otol 1988; 9: 403-408.
46. Gibbin KP, Davis CG. A hearing survey in diabetes mellitus. Clin Otolaryngol 1981; 6: 345-350.
47. Zakrisson J, Borg E, Liden G, Nilsson R. Stapedius reflex in industrial impact noise: fatiguability and role for temporary threshold shift. Scand Audiol 1980; (Suppl 12): 326-334.
48. Pierson LL, Gerhardt KJ, Rodriguez GP, Yanke RB. Relationship between outer ear resonance and permanent noise-induced hearing loss. Am J Otol 1994; 15: 37-40.
49. Spoendlin H. Histopathology of noise deafness - Review. J Otolaryngol 1985; 14: 282-286.
50. Schact J. Biochemistry of cochlear function and pathology. Sem Hear 1986; 7: 101-104.
51. Gannon RP, Tso SS, Chung DY. Interaction of kanamycin and noise exposure. J Laryngol Otol 1979; 93: 341-347.
52. Silverstein H, Bernstein JM, Davies DG. Salicylate ototoxicity: A biochemical and electrophysiological study. Ann Otol Rhinol Laryngol 1967; 76: 118-127.
53. Wright CG, Schaefer SD. Inner ear histopathology in patients treated with cisplatinum. Laryngoscope 1982; 92: 1408-1413.
54. Nakai Y, Konishi K, Chang KC, Ohashi K, Morisaki N, Minowa Y et al. Ototoxicity of the anticancer drug cisplatinum: An experimental study. Acta Otol Laryngolica 1982; 93: 227-232.
55. Iki M, Kurumatani N, Hirata K, Moriyama T. An association between Raynaud's phenomenon and hearing loss in forestry workers. Am Ind Hyg Assoc J 1985; 46: 509-513.
56. Crutchfield CD, Sparks T. Effects of noise and vibration on farm workers. Occup Med 1991; 6: 355-369.
57. Dijki F. Non-auditory effects of noise in industry: a review of literature. Int Arch Occup Environ Health 1986; 58: 325-338.

58. van Dijki FJ, Ettema JH, Zielhuis RL . Non-auditory effects of noise in industry: evaluation, conclusion and recommendation. Int Arch Occup Environ Health 1987; 59: 147-152.
59. Neiemtzow RC. Loud noise and pregnancy. Mil Med 1993; 158: 10-12.
60. Hetu R. Mismatches between auditory demands and capacities in the industrial work environment: Review. Audiology 1994; 33: 1-14.
61. Segal S, Harell M, Shahar A, Englender M. Acute acoustic trauma; dynamics of hearing loss following cessation exposure. Am J Otol 1988; 9: 293-298.
62. Roitman R, Talmi YP, Finkelstein Y, Silver S, Sadov R, Zohar Y. Acoustic trauma-induced Meniere's syndrome (Review). Otorhinolaryngology 1989; 51: 246-250.
63. Clark SK, Rees TS. Post-traumatic endolymphatic hydrops. Arch Otolaryngol 1977; 103: 725-726.
64. Catlin FI. Noise induced hearing loss. Am J Otol 1986; 7; 141-149.
65. Melnick W. Human temporary threshold shift and damage risk. J Acoust Soc Am 1991; 90: 147-154.
66. Gao WY, Ding DL, Zheng XY, Ruan FM, Liu YJ. A comparison of changes in the stereocilia between temporary and permanent hearing loss in acoustic trauma. Hear Res 1992; 62: 27-41.
67. Orgler G, Brubaker W, Crane D, Glorig A, Hatfield T, Hanson R et al. American occupational medicine association noise and hearing conservation committee guidelines for the conduct of an occupational hearing conservation program. J Occup Med 1987; 29: 981-982.
68. Talmi Y, Finkelstein Y, Zohar Y. Barotrauma induced-hearing loss. Scand Audiol 1991; 20: 1-9.
69. American Medical Association. Guides to the evaluation of permanent impairment. 4th ed. Chicago (USA): AMA; 1993.
70. Staloff R, Staloff J. Occupational hearing loss. 2nd ed. New York (USA): Marcel Derkker Inc; 1993.
71. Association of Workers Compensation Boards of Canada. Benefit Comparisons as of July 1, 1990. Edmonton, Alberta: AWCBC; 1990.
72. Raffle PA, Adams PH, Baxter PJ, Lee WR. Hunter's Diseases of Occupations. 8th ed. London (UK): Edward Arnold Publisher; 1994.
73. General Organization for Social Insurance, Riyadh. Regulations for rules and procedures for implementation of the occupational hazards branch and implementing decisions. 1st Ed. Kingdom of Saudi Arabia: 1404 H. - 1984 G.