TECHNICAL SPECIFICATION

ISO/TS 16976-7

First edition
2013-04-15

Respiratory protective devices — Human factors —

Part 7: Hearing and speech

Appareils de protection respiratoire — Facteurs humains —

Partie 7: Discours et audition

Reference number
ISO/TS 16976-7:2013(E)

Contents

Page

Foreword

ISO (the International Organization for Standardization) is a worldwide federation of national standards bodies (ISO member bodies). The work of preparing International Standards is normally carried out through ISO technical committees. Each member body interested in a subject for which a technical committee has been established has the right to be represented on that committee. International organizations, governmental and non-governmental, in liaison with ISO, also take part in the work. ISO collaborates closely with the International Electrotechnical Commission (IEC) on all matters of electrotechnical standardization.

The procedures used to develop this document and those intended for its further maintenance are described in the ISO/IEC Directives, Part 1. In particular the different approval criteria needed for the different types of ISO documents should be noted. This document was drafted in accordance with the editorial rules of the ISO/IEC Directives, Part 2. www.iso.org/directives

Attention is drawn to the possibility that some of the elements of this document may be the subject of patent rights. ISO shall not be held responsible for identifying any or all such patent rights. Details of any patent rights identified during the development of the document will be in the Introduction and/or on the ISO list of patent declarations received. www.iso.org/patents

Any trade name used in this document is information given for the convenience of users and does not constitute an endorsement.

The committee responsible for this document is ISO/TC 94, *Personal safety — Protective clothing and equipment*, Subcommittee SC 15, *Respiratory protective devices*.

ISO/TS 16976 consists of the following parts, under the general title *Respiratory protective devices — Human factors*:

— *Part 1: Metabolic rates and respiratory flow rates*

— *Part 2: Anthropometrics*

— *Part 3: Physiological responses and limitations of oxygen and limitations of carbon dioxide in the breathing environment*

— *Part 4: Work of breathing and breathing resistance: Physiologically based limits*

— *Part 5: Thermal effects*

— *Part 7: Hearing and speech*

— *Part 8: Ergonomic factors*

The following part is under preparation:

— *Part 6: Psycho-physiological effects*

Introduction

For an appropriate design, selection and use of respiratory protective devices, basic physiological demands of the user must be considered. The function of a respiratory protective device, the way it is designed and used and the properties of its material may affect communications: either speech or hearing or both.

This part of ISO/TS 16976 belongs to a series of documents providing basic physiological and anthropometric data on humans. It contains information about hearing and speech associated with wearing respiratory protective devices.

Respiratory protective devices — Human factors —

Part 7: **Hearing and speech**

1 Scope

This part of ISO/TS 16976 contains information related to the interaction between respiratory protective devices and the human body functions of hearing and speech.

2 Normative references

The following documents, in whole or in part, are normatively referenced in this document and are indispensable for its application. For dated references, only the edition cited applies. For undated references, the latest edition of the referenced document (including any amendments) applies.

ISO 1999, *Acoustics — Estimation of noise-induced hearing loss*

ISO 16972, *Respiratory protective devices — Terms, definitions, graphical symbols and units of measurement*

3 Terms and definitions, and abbreviated terms

3.1 Terms and definitions

For the purposes of this document, the terms and definitions given in ISO 1999, ISO 16972 and the following apply.

3.1.1
hearing
manner in which the brain and central nervous system recognizes and interprets sounds

3.1.2
ototoxicity
damage to hearing from overexposure to drugs or toxic substances

3.1.3
noise
unwanted sound

3.1.4
presbycusis
gradual sensorineural hearing loss due to natural ageing

3.1.5
sound
form of energy that moves through media in waves of pressure

3.1.6
sound pressure
local pressure change caused by the vibration

Note 1 to entry: Measured in pascals (Pa).

3.2 Abbreviated terms

SPL sound pressure level

NIHL noise induced hearing loss

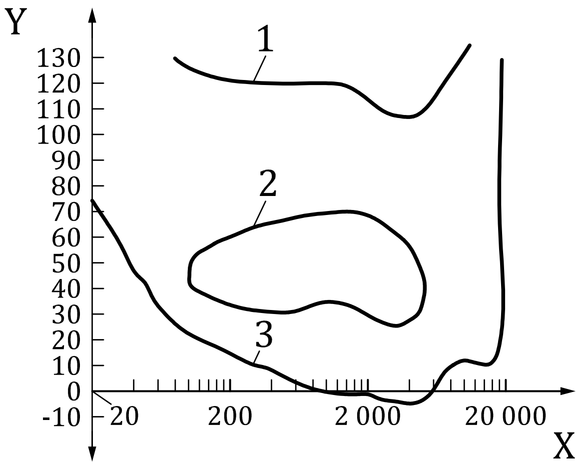
TWA time-weighted average

STI speech transmission index

SII speech intelligibility index

4 Range of hearing and speech

Humans with normal hearing can usually hear sound pressure waves in a frequency range of about 20 Hz to 20 000 Hz, but the ear is most sensitive to frequencies from 500 Hz to around 4 000 Hz and declines dramatically in sensitivity as frequencies drop below 500 Hz. Figure 1 depicts the frequencies response and sound pressure level response of human hearing and speech. The frequency range is affected by ageing as explained further in 7.3.



Key

X logarithmic sale of frequency (Hz)

Y sound pressure level (dB)

1 pain threshold

2 range of speech

3 hearing threshold

Figure 1 — Range of human hearing and speech

5 Measurement of sound pressure

Sound pressure is the local pressure change caused by the vibration and is measured in pascals (Pa). Sound pressure level (SPL) is the logarithmic ratio of the sound pressure to a reference pressure and is expressed in decibels (dB) by the equation

$$L_p = 20 \log_{10}\left(\frac{P_{\mathrm{RMS}}}{P_0}\right)$$

where

L_p is the sound pressure level, in dB;

P_{RMS} is the root mean square (RMS) sound pressure, in Pa;

P_0 is the sound reference pressure, in Pa.

In air, the sound reference pressure is 0,000 02 Pa. That reference is based on the average human threshold of hearing at a frequency of 1 000 Hz.

When measuring sound pressure level as it relates to human perception, weighting factors are used to represent human threshold at different frequencies. The most common is the A-weighted sound measurement which approximates the human threshold at 40 dB and is expressed as dBA. Two other sound measuring weighting factors exist: B-weighted and C-weighted, which approximate the human threshold at 70 dB and 100 dB, respectively.

Examples of some typical sound levels are:

library	40 dBA
normal conversation	60 dBA
traffic noise	80 dBA
metal shop	100 dBA
siren	120 dBA
jet engine	140 dBA

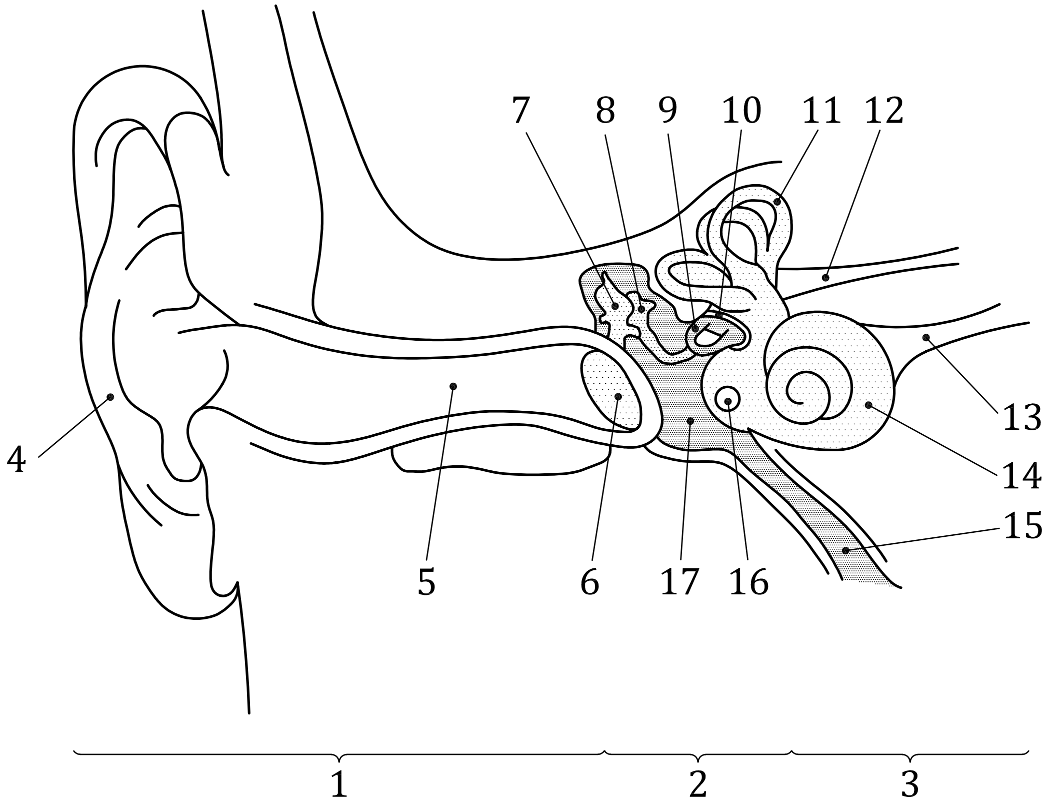
A perceived difference in sound level occurs at approximately 3 dB, and a perceived doubling of sound volume occurs with a 10 dB increase in sound pressure level.

6 Physiology of the ear

6.1 General

The human ear is the sense organ that detects sounds and changes the pressure waves into a signal of nerve impulses that is sent to the brain. The ear not only receives and converts sound but also plays a major role in the sense of balance and body position.

As shown in Figure 2, the ear is usually described in three sections: the outer ear (key 1), middle ear (key 2) and inner ear (key 3).



Key

1	outer ear	10	oval window
2	middle ear	11	semicircular canals
3	inner ear	12	vestibular nerve
4	pinna	13	cochlear nerve
5	external auditory channel	14	cochlea
6	tympanic membrane	15	Eustachian tube
7	malleus	16	round window
8	incus	17	tympanic cavity
9	stapes		

Figure 2 — Physiological ear terms

6.2 Outer ear

The outer ear is the most external portion of the ear. The outer ear includes the pinna (also called auricle), the ear canal, and the very most superficial layer of the ear drum (also called the tympanic membrane). In humans, the only visible portion of the ear is the outer ear. The outer ear does help get sound (and imposes filtering), but the ear canal is very important. Unless the canal is open, hearing will be damped. Ear wax (cerumen) is produced by glands in the skin of the outer portion of the ear canal. The outer ear ends at the most superficial layer of the tympanic membrane. The tympanic membrane is commonly called the ear drum.

The pinna helps direct sound through the ear canal to the tympanic membrane (eardrum).

6.3 Middle ear

The middle ear, an air-filled cavity behind the ear drum (tympanic membrane), includes the three ear bones or ossicles: the malleus (or hammer), incus (or anvil), and stapes (or stirrup). The opening of the

Eustachian tube is also within the middle ear. The three bones are arranged so that movement of the tympanic membrane causes movement of the malleus, which causes movement of the incus, which causes movement of the stapes. When the stapes footplate pushes on the oval window, it causes movement of fluid within the cochlea (a portion of the inner ear).

In humans the middle ear (like the ear canal) is normally filled with air. Unlike the open ear canal, however, the air of the middle ear is not in direct contact with the atmosphere outside the body. The Eustachian tube connects from the chamber of the middle ear to the back of the pharynx.

The arrangement of the tympanic membrane and ossicles works to efficiently couple the sound from the opening of the ear canal to the cochlea. There are several simple mechanisms that combine to increase the sound pressure.

— The first is the “hydraulic principle”. The surface area of the tympanic membrane is many times that of the stapes footplate. Sound energy strikes the tympanic membrane and is concentrated to the smaller footplate.

— A second mechanism is the “lever principle”. The dimensions of the articulating ear ossicles lead to an increase in the force applied to the stapes footplate compared with that applied to the malleus.

— A third mechanism channels the sound pressure to one end of the cochlea, and protects the other end from being struck by sound waves. In humans, this is called “round window protection”.

6.4 Inner ear

The inner ear includes both the organ of hearing (the cochlea) and a sense organ that is attuned to the effects of both gravity and motion (labyrinth or vestibular apparatus). The balance portion of the inner ear consists of three semicircular canals and the vestibule. When sound strikes the ear drum, the movement is transferred to the footplate of the stapes, which presses into one of the fluid-filled ducts of the cochlea. The fluid inside this duct is moved, flowing against the receptor cells of the Organ of Corti, which fire. These stimulate the spiral ganglion, which sends information through the auditory portion of the eighth cranial nerve to the brain.

7 Hearing loss

7.1 Conductive hearing loss

Abnormalities such as impacted ear wax (occlusion of the external ear canal), fixed or missing ossicles, or holes in the tympanic membrane generally produce conductive hearing loss. Conductive hearing loss may also result from middle ear inflammation causing fluid build-up in the normally air-filled space. In some cases conductive hearing loss is reversible.

7.2 Ototoxicity

A number of drugs in clinical use are considered “ototoxic”, having the potential to cause damage to hearing as a side effect. Hearing loss caused by ototoxic drugs can be reversible or permanent.

7.3 Presbycusis

Hearing loss caused by natural aging affects the higher frequencies making word recognition difficult, see Clause 8. It is permanent.

7.4 Noise induced hearing loss (NIHL)

NIHL is caused by exposure to sound levels or durations that damage the hair cells of the cochlea. Initially, the noise exposure may cause a temporary threshold shift, that is, a decrease in hearing sensitivity that typically returns to its former level within a few minutes to a few hours. Repeated exposures lead to a permanent threshold shift, which is an irreversible sensorineural hearing loss. Hearing loss has causes other than occupational noise exposure. Hearing loss caused by exposure to nonoccupational noise is collectively called sociocusis. It includes recreational and environmental noises (e.g. loud music, guns, power tools, and household appliances) that affect the ear the same as occupational noise. Combined exposures to noise and certain physical or chemical agents (e.g. vibration, organic solvents, carbon monoxide, ototoxic drugs, and certain metals) appear to have synergistic effects on hearing loss.[1],[2],[3],[4],[5],[6],[7],[8],[9],[10],[11],[12],[13],[14],[15] Some sensorineural hearing loss occurs naturally because of ageing; this loss is called presbycusis. Conductive hearing losses, as opposed to sensorineural hearing losses, are usually traceable to diseases of the outer and middle ear.

7.5 Other types of hearing loss

For more information on other types of hearing loss, refer to ISO 1999.

7.6 Other effects of noise

Noise exposure is also associated with nonauditory effects such as psychological stress and disruption of job performance[17],[18],[19] and possibly hypertension.[20],[21],[22],[23],[24],[25],[26],[27],[28],[29],[30],[31],[32] Noise may also be a contributing factor in industrial accidents.[33],[34],[35],[36] Nevertheless, data are insufficient to endorse specific damage risk criteria for these nonauditory effects.

8 Noise exposure limits

8.1 Workplace exposure levels and durations

Occupational noise exposure should be controlled so that worker exposures are less than the combination of exposure level (L) and duration (T), as calculated by the following formula (or as shown in Table 1).

$$T(\text{min}) = \frac{480}{2^{(L-\text{TWA})/3}}$$

where TWA (Time-Weighted Average) noise exposure limits vary with jurisdiction, but are usually accepted to be 80 dBA or 85 dBA for an eight hour work shift using a 3 dB exchange rate, i.e. the duration halves for every 3 dB increase in sound pressure level (SPL).

Table 1 — Maximum exposure duration (*T*)

SPL	Maximum duration for TWA = 85 dBA			Maximum duration for TWA = 80 dBA		
dB	h	min	s	h	min	s
80	25	24		8	0	
81	20	10		6	21	
82	16	0		5	2	
83	12	42		4	0	
84	10	5		3	10	
85	8	0		2	31	
86	6	21		2	0	
87	5	2		1	35	
88	4	0		1	16	
89	3	10		1	0	
90	2	31			47	37
91	2	0			37	48
92	1	35			30	0

Table 1 *(continued)*

SPL	Maximum duration for TWA = 85 dBA			Maximum duration for TWA = 80 dBA		
dB	h	min	s	h	min	s
93	1	16			23	49
94	1	0			18	54
95		47	37		15	0
96		37	48		11	54
97		30	0		9	27
98		23	49		7	30
99		18	54		5	57
100		15	0		4	43
101		11	54		3	45
102		9	27		2	59
103		7	30		2	22
104		5	57		1	53
105		4	43		1	29
106		3	45		1	11
107		2	59			56
108		2	22			45
109		1	53			35
110		1	29			28
111		1	11			22
112			56			18
113			45			14
114			35			11
115			28			9
116			22			7
117			18			6
118			14			4
119			11			4
120			9			3
121			7			2
122			6			2
123			4			1
124			4			1
125			3			<1
126			2			<1
127			2			<1
128			1			<1
129			1			<1
130			<1			<1
131			<1			<1
132			<1			<1

Table 1 *(continued)*

SPL	Maximum duration for TWA = 85 dBA			Maximum duration for TWA = 80 dBA		
dB	h	min	s	h	min	s
133			<1			<1
134			<1			<1
135			<1			<1
136			<1			<1
137			<1			<1
138			<1			<1
139			<1			<1

Where the noise exposure is composed of two or more periods of noise at different levels, the total noise dose (D), which is the actual exposure relative to the amount of allowable exposure and for which a dose of 100 and above represent exposures that are hazardous, can be calculated as:

$$D=100\left(\frac{C_1}{T_1}+\frac{C_2}{T_2}+......+\frac{C_n}{T_n}\right)$$

where

C_n indicates the total time of exposure at a specific noise level;

T_n indicates the duration for that level as given in the table above.

Incremental doses can be summed using the following tables.

As an example, calculate the dose for an employee who is subjected to 92 dB for 2 h, 87 dB for 4 h and 82 dB for 2 h.

From Table 1, T_n for 92 dB is 1 h 35 min (1,58 h), for 87 dB, 5 h 2 min (5,03 h), and for 82 dB, 16 h.

Therefore,

$$D = 100\left(\frac{2}{1{,}58}+\frac{4}{5{,}03}+\frac{2}{16}\right) = 100\ (1{,}27 + 0{,}80 + 0{,}13) = 220\ \%$$

Using Table 2 below,

D = 126 + 79 +13 = 218 %.

The difference is caused by rounding.

Table 2 — Percentage of total dose based on 85 dBA TWA

SPL (dBA)	Duration of exposure (h)							
	1/4	1/2	1	2	4	8	10	12
105	318	636						
104	252	504	1008					
103	200	400	800					
102	159	317	635					
101	126	252	504	1008				
100	100	200	400	800				

Table 2 *(continued)*

SPL (dBA)	Duration of exposure (h)							
	1/4	1/2	1	2	4	8	10	12
99	79	159	317	635				
98	63	126	252	504	1008			
97	50	100	200	400	800			
96	40	79	159	317	635			
95	32	63	126	252	504	1008		
94	25	50	100	200	400	800	1000	1200
93	20	39	79	158	316	632	789	947
92	16	32	63	126	253	505	632	758
91	13	25	50	100	200	400	500	600
90	10	20	40	79	159	318	397	477
89	8	16	32	63	126	253	316	379
88	6	13	25	50	100	200	250	300
87	5	10	20	40	79	159	199	238
86	4	8	16	31	63	126	157	189
85	3	6	13	25	50	100	125	150
84	2	5	10	20	40	79	99	119
83	2	4	8	16	31	63	79	94
82	2	3	6	13	25	50	63	75
81	1	2	5	10	20	40	50	60
80	1	2	4	8	16	31	39	47
79				6	13	25	32	38
78				5	10	20	25	30
77					8	16	20	24
76					6	13	16	19
75					5	10	13	15

8.2 Ceiling limit

Exposure to continuous, varying, intermittent, or impulsive noise is regulated by national regulations and is generally accepted not to exceed 137 dBC or 140 dBC.

9 Speech and hearing difficulties

Speech as shown in Figure 1 encompasses a range from about 100 Hz to 5 000 Hz. Various phonemes are comprised of different frequencies even though they may share a similar sound. Plosives like “pa”, “ka”, “tee”, “bee” and “dee” range from low to medium frequencies. Fricatives like “sss”, “vee” and “zee” are mostly high-frequency sounds.

Vowels are mostly medium- to low-frequency sounds. Thus, communication problems due to hearing loss will be dependent on the frequency of the loss. If hearing loss is in the high-frequency range, as it usually is with presbycusis, fricative sounds will be easily confused.

Further affecting the ability to hear speech with high-frequency loss is the fact that there is a difference in the frequency range between male and female voices: males use frequencies down to about 100 Hz while females’ lower limit is about 250 Hz.

The ability to understand speech is compounded by many factors in addition to hearing loss. One major issue is masking due to the background noise. The noise spectrum and speech-to-noise ratio are very important to understanding speech. Another factor is distortion caused by reverberations, muffling, echoes, or just plain variations in pronunciations or dialect. This arena is far too complex for this document.

Some of these confounding factors can be mitigated by the use of mechanical or electronic devices, such as radios, hearing protectors with microphones, hearing aids, and amplifiers.

There are methods to assess the ability of individuals with normal hearing to understand speech under different environmental conditions. The Speech Transmission Index (STI) is one such method. The Speech Intelligibility Index (SII) and modified rhyme test are other examples. With both methods, the environment and the criteria must be established from the needs of the user.

Some respirators can distort or muffle sounds and others can create noise on their own by the use of blowers or just the flow of air. Standards for respirators should consider the needs for communication and the prevention of hearing loss, due to the respirator, in the performance requirements.

Bibliography

[1] Hamernik R.P., & Henderson D.The potentiation of noise by other ototraumatic agents. In: *Effects of noise on hearing*, (Hamernik R.P., Henderson D., Dosanjh D.S., Mills J.H.eds.). Raven Press, New York, 1976, pp. 291–307.

[2] Brown J.J., Brummett R.E., Meikle M.B., Vernon J. Combined effects of noise and neomycin: cochlear changes in the guinea pig. *Acta Otolaryngol.* 1978, **86** pp. 394–400

[3] Gannon R.P., Tso S.S., Chung D.Y. Interaction of kanamycin and noise exposure. *J. Laryngol. Otol.* 1979, **93** pp. 341–347

[4] Brown J.J., Brummett R.E., Fox K.E. Combined effects of noise and kanamycin: cochlear pathology and pharmacology. *Acta Otolaryngol.* 1980, **106** pp. 744–775

[5] Hamernik R.P., Henderson D., Coling D., Slepecky N. The interaction of whole body vibration and impulse noise. *J. Acoust. Soc. Am.* 1980, **67** (3) pp. 928–934

[6] Pryor G., Dickinson J., Howd R.A., Rebert C.S. Transient cognitive deficits and high-frequency hearing loss in weanling rats exposed to toluene. *Neurobehav. Toxicol. Teratol.* 1983, **5** pp. 53–57

[7] Rebert C.S., Sorenson S.S., Howd R.A., Pryor G.T. Toluene-induced hearing loss in rats evidenced by the brainstem auditory-evoked response. *Neurobehav. Toxicol. Teratol.* 1983, **5** pp. 59–62

[8] Humes L.E. Noise-induced hearing loss as influenced by other agents and by some physical characteristics of the individual. *J. Acoust. Soc. Am.* 1984, **76** (5) pp. 1318–1329

[9] Boettcher F.A., Henderson D., Gratton M.A., Danielson R.W., Byrne C.D. Synergistic interactions of noise and other ototraumatic agents. *Ear Hear.* 1987, **8** (4) pp. 192–212

[10] Young S.Y., Upchurch M.B., Kaufman M.J., Fechter L.D. Carbon monoxide exposure potentiates high-frequency auditory threshold shifts induced by noise. *Hear. Res.* 1987, **26** pp. 37–43

[11] Bynes C., Henderson D., Saunders S., Powers N., Farzi F.Interaction of noise and whole body vibration. In: *Recent advances in researches on the combined effects of environmental factors*, (Manninen O.ed.). Py-Paino Oy Printing House, Tampere, Finland, 1988

[12] Fechter L.D., Young J.S.Y., Carlisle L. Potentiation of noise induced threshold shifts and hair cell loss. *Hear. Res.* 1988, **34** pp. 39–48

[13] Johnson A., Juntunen L., Nylén P., Borg E., Höglund G. Effects of interaction between noise and toluene on auditory function in the rat. *Acta Otolaryngol. (Stochh)*. 1988, **105** pp. 56–63

[14] Morata T.C., Dunn D.E., Kretschmer L.W., Lemasters G.K., Keith R.W. Effects of occupational exposure to organic solvents and noise on hearing. *Scand. J. Work Environ. Health*. 1993, **19** (4) pp. 245–254

[15] Franks J.R., & Morata T.C.Ototoxic effects of chemicals alone or in concert with noise: a review of human studies. In: *Scientific basis of noise-induced hearing loss*, (Axelsson A., Borchgrevink H.M., Hamernik R.P., Hellström P.A., Henderson D., Salvi R.J.eds.). Thieme Medical Publishers, Inc, New York, 1996

[16] ISO 1999, *Acoustics — Estimation of noise-induced hearing loss*

[17] Taylor S.M. A path model of aircraft noise annoyance. *Sound Vibrat.* 1984, **96** (2) pp. 243–260

[18] Öhrström E., Björkman M., Rylander R. Noise annoyance with regard to neuro-physiological sensitivity, subjective noise sensitivity and personality variables. *Psychol. Med.* 1988, **18** pp. 605–613

[19] Suter A.H. The effects of noise on performance. Aberdeen Proving Grounds, MD: U.S. Army Human Engineering Laboratory. Technical memorandum 3-89, 1989

[20] Parvizpoor D. Noise exposure and prevalence of high blood pressure among weavers in Iran. *J. Occup. Med.* 1976, **18** (11) pp. 730–731

[21] Jonsson A., & Hansson L. Prolonged exposure to a stressful stimulus (noise) as a cause of raised blood pressure in man. *Lancet*. 1977, **1** pp. 86–87

[22] Takala J., Varke S., Vaheri E., Sievers K. Noise and blood pressure. *Lancet*. 1977, **1** pp. 974–975

[23] Lees R.E.M., & Roberts J.H. Noise-induced hearing loss and blood pressure. *CMAJ*. 1979, **120** (5) pp. 1082–1084

[24] Alchaire J.B., & Mullier M. Occupational exposure to noise and hypertension: a retrospective study. *Ann. Occup. Hyg.* 1979, **22** pp. 63–66

[25] Manninen O., & Aro S. Noise-induced hearing loss and blood pressure. *Int. Arch. Occup. Environ. Health*. 1979, **42** pp. 251–256

[26] Singh A.P., Rai R.M., Bhatia M.R., Nayar H.S. Effect of chronic and acute exposure to noise on physiological functions of man. *Int. Arch. Occup. Environ. Health*. 1982, **50** pp. 169–174

[27] Belli S., Sani L., Scarficcia G., Sorrentino R. Arterial hypertension and noise: a cross-sectional study. *Am. J. Ind. Med.* 1984, **6** pp. 59–65

[28] Delin C. Noisy work and hypertension. *Lancet*. 1984, **2** p. 931

[29] Talbott E., Helmkamp J., Matthews K., Kuller L., Cottington E., Redmond G. Occupational noise exposure, noise-induced hearing loss and the epidemiology of high blood pressure. *Am. J. Epidemiol.* 1985, **121** (4) pp. 501–504

[30] Verbeek J.H.A.M., van Dijk F.J.H., deVries F.F. Non-auditory effects of noise in industry. IV. A field study on industrial noise and blood pressure. *Int. Arch. Occup. Environ. Health*. 1987, **59** pp. 51–54

[31] Wu T.N., Ko Y.C., Chang P.Y. Study of noise exposure and high blood pressure in shipyard workers. *Am. J. Ind. Med.* 1987, **12** pp. 431–438

[32] Talbott E., Findlay R., Kuller L., Lenkner L., Matthews K., Day R. et al. Noise-induced hearing loss: a possible marker for high blood pressure in older noise-exposed populations. *J. Occup. Med.* 1990, **32** (8) pp. 685–689

[33] Cohen A. The influence of a company hearing conservation program on extra-auditory problems in workers. *J. Safety Res.* 1976, **8** (4) pp. 146–162

[34] Schmidt J.A.W., Royster L.H., Pearson R.G. Impact of an industrial hearing conservation program on occupational injuries for males and females [Abstract]. *J. Acoust. Soc. Am.* 1980, **67** p. S53

[35] Wilkins P.A., & Acton W.I. Noise and accidents – a review. *Am. Occup. Hyg.* 1982, **215** pp. 249–260

[36] Moll van Charante A.W., & Mulder P.H.G. Perceptual acuity and the risk of industrial accidents. *Am. J. Epidemiol.* 1990, **131** pp. 652–663

ICS 13.340.30

Price based on 12 pages