OCCUPATIONAL HEARING LOSS AND VIBRATION INDUCED DISORDERS

C M Jones

Hearing loss

Incidence of occupational hearing loss

- Department of Social Security estimated that 13 000 workers received benefits in 1992
- It is the third commonest assessed claim (after hand-arm vibration syndrome and tenosynovitis)
- The OPCS Disability Survey (1985-8) estimated that 52 500 people were affected in England and Wales
- The 1990 Labour Force survey estimated that 103 100 people had deafness, tinnitus, or other ear conditions caused by work and a further 18 300 thought that their ear condition had been made worse by work

Causes of deafness

Sensorineural loss

- Congenital deafness (associated with maternal rubella or flu or prenatal medication)
- Familial deafness
- Birth trauma
- Childhood illnesses (such as measles (usually bilateral deafness), mumps (unilateral), encephalitis, meningitis, cerebral abscess, typhus)
- Ototoxic drugs
  - Streptomycin and some other antibiotics (such as gentamicin and neomycin)
  - Anti-rheumatic drugs
  - Diuretics
  - Quinine, nicotine, alcohol, and aspirin
- Fracture of base of skull
- Acoustic neuroma (unilateral deafness)
- Ménière's disease
- Presbycusis

Conductive loss

- Impacted earwax
- Ruptured eardrum (blow to head or explosion)
- Blockage of eustachian tube
- Ossicular dysfunction
  - Dislocation
  - Otitis media or fluid
  - Otosclerosis

Audiometric characteristics of noise induced hearing loss

- Bilateral notch in hearing threshold at 3, 4, or 6 kHz with recovery at 8 kHz
- Progressive deepening and widening of notch with increasing exposure to noise
- Notch due to shooting is narrow and asymmetrical (in right handed people it is deeper on left side because right ear is protected by gun stock)
- The 6 kHz frequency is the most variable tested, and an isolated notch at 6 kHz is usually of no clinical importance

In 1908 the annual report of the Chief Inspector of Factories stated: "men employed in certain trades are liable to have their sense of hearing seriously impaired, if not entirely destroyed in the course of time, as a result of long continued exposure to loud noise." It is only in recent decades that this situation has begun to change.

This irreversible sensorineural deafness is caused by damage to the hair cells of the organ of Corti in the cochlea. It can be the cause of accidents due to failure to hear warning signals. It reduces the quality of life and, especially in elderly people, produces social isolation. If tinnitus is prominent psychiatric symptoms can occur.

Clinical presentation

Noise induced hearing loss develops insidiously. A gradual loss of clarity in perceived speech occurs, which is often attributed to inattention or to others not speaking clearly because when the sufferer looks at speakers he or she can understand them. Difficulty in understanding others in a crowd is, in the same way, presumed to be due to competition with background noise (perceptual rivalry). Eventually, the sufferer realises that others do not have this problem. This realisation may come suddenly, such as when a telephone with an electronic bleep is bought to replace one with a bell.

A high pitched tinnitus, initially intermittent, becomes continuous in up to 20% of cases and can be a presenting symptom. The other feature (usually revealed on direct questioning) is loudness recruitment: at a certain volume perceived sound suddenly becomes more intense.

Diagnosis

As well as asking about current symptoms, the doctor taking a patient's history should cover other possible causes of sensorineural and conductive deafness. Both social and work related exposure to noise should be reviewed, including the firing of guns (military and sporting), playing in pop groups, and listening to amplified music. To help assess the level of noise at work, a useful rule of thumb is that voices need to be raised to communicate over a distance of one metre at 90 dB(A).

A check should be made of the present and past use of hearing protectors, their type, and how often they are used.

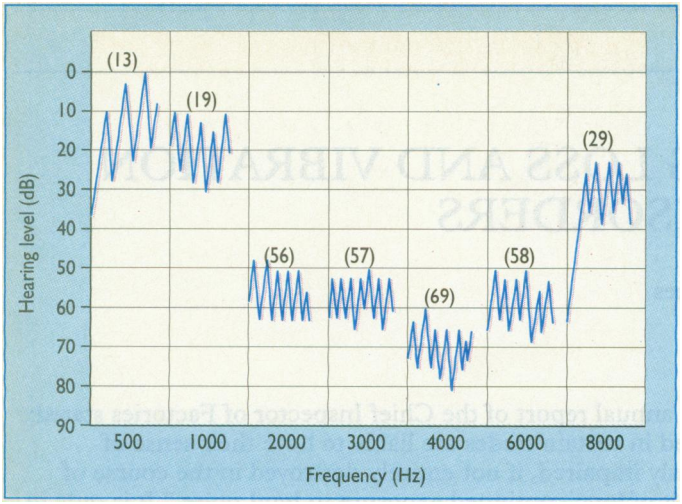
Examination by otoscopy and tuning fork tests can exclude other causes of deafness. The diagnosis is usually made by air and bone conduction, audiometry being compatible with exposure to noise, although other tests may be advisable in some cases.

Audiometry

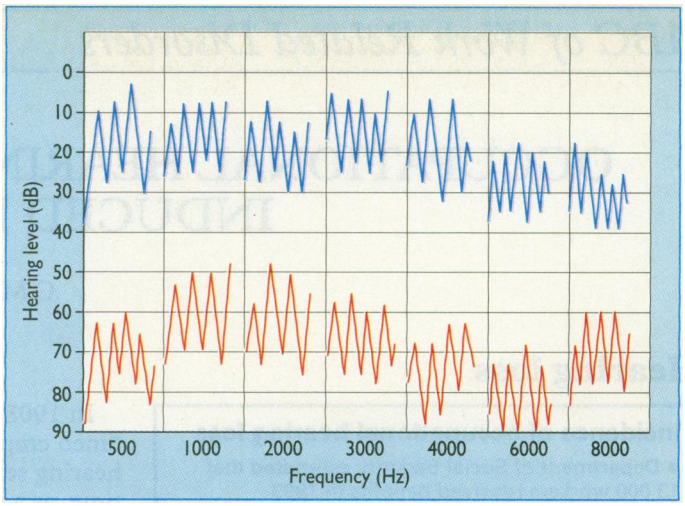
Audiometry should be performed when the subject has not been exposed to loud noise for at least 16 hours (or has worn high efficiency muffs before the test). This minimises the temporary lowering of the hearing threshold caused by noise (temporary threshold shift).

There is a learning effect with audiometry. This can result in the thresholds for the second ear tested (conventionally the right ear) being better than the first. The learning effect can also extend to the next two audiometric tests. It may be up to 10-15 dB for each frequency.

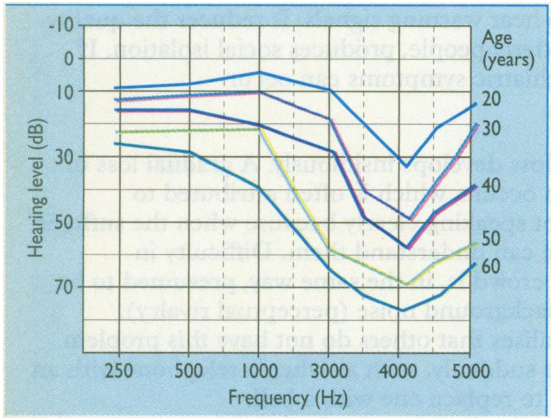
Audiometric screening in industry is usually done by discrete frequency, pulsed tone, self recording, air conducting audiometry according to the Bekesey or Hughson-Westlake procedure. Accuracy is related to the background level of noise during testing, and so a soundproof booth is usually required (EN 26189 gives the criteria).



Audiogram of 50 year old man with disability of 44 dB. Pattern is typical of noise induced hearing loss.



Audiogram showing conductive pattern of hearing loss after acute perforation of eardrum (red) compared with normal conductive pattern for a 54 year old man (blue).



Development of noise induced hearing loss with time (for exposure to 101 dB(A) and including presbycusis).

A loss of less than 20 dB in all frequencies can be considered normal. A similar reduction in all frequencies or one where the threshold improves in the higher frequencies indicates a conductive loss. A notch in the region of 1-3 kHz indicates a familial cause of hearing loss. Classic noise induced hearing loss occurs at 4 kHz. Presbycusis produces a smooth pattern of increasing loss in higher frequencies.

The population varies considerably in its susceptibility to the effect of noise (as shown by differences in men's hearing threshold for 1-3 kHz). The rate of hearing loss due to noise is greatest at initial exposure and reduces progressively with continued exposure. In contrast the changes due to presbycusis increase progressively and must always be considered when audiograms are interpreted. The progression is less in women and in people living in rural areas. As presbycusis affects the higher noise frequencies, most of its effect on the calculation of disability can usually be ignored during working life.

Average correction (in dB) for presbycusis

Age (years)	Noise frequency (kHz) 0.5	1	2	3	4	6	8
30	1	1	1	2	2	3	3
40	2	2	3	6	8	9	11
50	4	4	7	12	16	18	23
60	6	7	12	20	28	32	39
70	10	11	19	31	43	49	60

Average hearing threshold (in dB) for 1-3 kHz for men exposed to 90 dB(A)

Age (years)	Population centiles 10	30	50	70	90
20	11	6	3	0	–3
30	23	15	10	6	2
40	33	22	16	11	6
50	42	29	22	16	10
60	51	38	30	24	16

Calculation of disability for compensation awarded by Department of Social Security

Monaural: Mean hearing loss for 1 kHz, 2 kHz, and 3 kHz

Binaural: $\frac{\text{(Mean hearing loss of better ear)} \times 4 + \text{(worse ear)}}{5}$

Disability and compensation

Disability is calculated from the hearing loss in the main speech frequencies (1-3 kHz). Tinnitus adds to this. Disability should be calculated only from an audiogram produced after 48 hours without noise exposure (to exclude the temporary threshold shift) and preferably one that is not the first audiogram recorded for the patient (to allow for the learning effect). If the impairment is about the same in both ears and there is no tinnitus, it is unusual for the sufferer to experience important handicap with less than 20 dB of disability. At 50 dB the handicap is substantial, and lip reading is required for comprehension of speech. The Department of Social Security awards compensation when disability exceeds 50 dB but calculates the size of award from a disability of 30 dB. Many sufferers do not apply for compensation because they know the conditions are stringent.

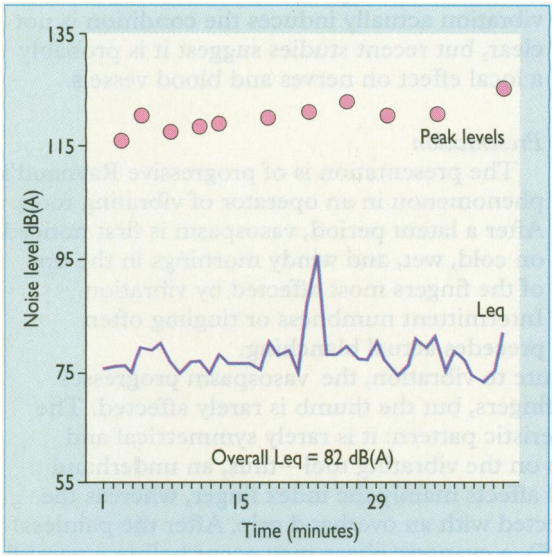
In recent years, because of the high cost of litigation, most compensation has been provided by agreements between unions and insurance companies, and impairments as low as 10-14 dB have been compensated. However, the trend is now against this because of the increasing value of awards given in the civil courts.

Noise in the workplace

Decibels (dB)

dB = 20 log (P/Po)
where P = Measured pressure
Po = Reference pressure (2×10^{-5} Pa)

Reference pressure is equivalent to threshold of hearing



Relation between short term Leqs, period Leq, and peak sound level in an industrial situation.

Ear muffs attached to a safety helmet.

1989 Noise at Work Regulations

Employers have a general duty to
- Make, review, and record assessments of noise
- Reduce damage to hearing of employees
- Reduce exposure to noise by methods other than use of ear protectors
- Provide hearing protection
- Create "ear protection zones"
- Use and maintain equipment for reducing exposure to noise
- Provide information, instruction, and training

Manufacturers are required to provide noise levels of equipment that they have supplied

Assessing noise exposure

Because of the ear's range of sensitivity, noise is measured on the logarithmic decibel scale, in which 0 is the limit of detection and about 130-135 dB is painful. As the ear has varying sensitivity to different sound frequencies, weighting factors are applied to each frequency to derive the dB(A) scale, in which all frequencies sound equally loud. An increase of 3 dB(A) is only just detectable to the human ear, though this is equivalent to a doubling of the sound intensity.

Hearing damage is related to the total noise dose received at the ear—a product of the noise level and its duration. Explosive noise is more damaging than continuous noise as the stapedial reflex (which reduces transmission of intense sound) takes 10 milliseconds to activate.

The Leq (the equivalent continuous sound level) is a single value that has the same energy level as the fluctuating levels normally experienced (the time weighted average). It can be calculated over any period of time, but modern sound level meters can measure it directly.

Legislation is formulated on the basis of an Leq for an eight hour day (the $L_{EP,d}$) and the maximum instantaneous (peak) noise level.

Noise in the workplace is assessed with sound level meters of varying complexity. A personal dosimeter may be attached to a person, or tape recordings can be taken for later analysis. A doctor needs to know the $L_{EP,d}$, the peak level, and the frequency distribution of the noise.

Preventing hearing loss

The use of ear protectors should be considered as a last resort when the engineering control of noise is inadequate. In reality, however, ear protectors are commonly needed because fully controlling noise at source can be very difficult and expensive.

Various types of protectors are available. Ear muffs, with oil or foam filled seals, are attached to head bands or helmets. Ear plugs—which may be disposable, reusable, or made to measure—and ear caps attached to stethoscope bands are also available. Manufacturers provide the mean and standard deviation of their ability to attenuate the standard frequencies or a single number rating (SNR). The level of protection is conventionally considered to be the "mean–(1 SD)" per frequency.

The selected ear protector should be appropriate for both the frequency and level of the noise and should attenuate sounds to about 80 dB(A). Any greater attenuation can cause social isolation. There is increasing evidence that some ear plugs are not as effective in practice as test figures suggest, and some companies prohibit their use for high noise levels. Protectors must be worn continuously to be effective: unprotected exposure to 100 dB(A) for 15 minutes is equivalent to eight hours of exposure at 85 dB(A), and even the best protector worn for half of the time will reduce the $L_{EP,d}$ by only 3-5 dB(A).

Legal considerations

Until the 1989 Noise at Work Regulations were introduced after the 1986 European Commission directive, only a voluntary code of practice published by the Department of Employment (1972) and the 1974 Woodworking Regulations applied.

The current regulations state that

- At the first action level ($L_{EP,d}$ of 85 dB(A)) an assessment should be made by a competent person, advice be given to employees, ear protectors be made available, and noise reduction be effected if reasonably practicable
- At the second action level ($L_{EP,d}$ of 90 dB(A)) the full requirement of the legislation are invoked (see box), and ear protection must be worn
- If the peak sound pressure exceeds 200 pascals (that is, 140 dB relative to 20 μPa) the full requirements apply regardless of the $L_{EP,d}$.

Many employers implement a comprehensive hearing conservation programme with routine audiometry.

Hand-arm vibration syndrome

Classification of hand-arm vibration syndrome (Stockholm scale)

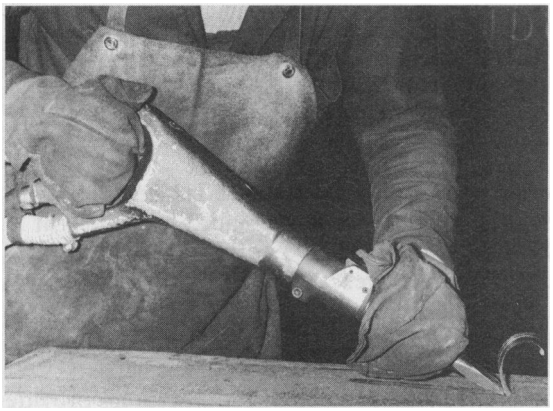
Vascular component

Stage (grade)	*Description*
0	No attacks
1V (mild)	Occasional attacks affecting only tips of one or more fingers
2V (moderate)	Occasional attacks affecting distal and middle (rarely also proximal) phalanges of one or more fingers
3V (severe)	Frequent attacks affecting all phalanges of most fingers
4V (very severe)	As in stage 3 but with trophic changes in fingertips

Sensorineural component

Stage	*Description*
0SN	Exposed to vibration but no symptoms
1SN	Intermittent numbness with or without tingling
2SN	Intermittent or persistent numbness, reduced sensory perception
3SN	Intermittent or persistent numbness, reduced tactile discrimination or manual dexterity

Staging is made separately for each hand. Grade of disorder is indicated by stage and number of affected fingers on both hands.



Using a chipping hammer with overhand grip.

Differential diagnosis of hand-arm vibration syndrome (most are rare)

Vascular
- Trauma
- Polyarteritis nodosa
- Scleroderma
- Thoracic outlet syndrome
- Cold agglutinins
- Systemic lupus erythematosus
- Dermatomyositis
- Rheumatoid arthritis

Neurological
- Peripheral nerve entrapment
- Trauma to arm or neck
- Peripheral neuropathy
- Drugs and other toxic effects
- Syringomyelia
- Spinal cord compression
- Multiple sclerosis

Further reading

- *The noise at work regulations.* London: HMSO, 1990. (Noise guides Nos 1-8.)
- *Management of health and safety at work regulations and approved code of practice.* London: HMSO, 1992.
- *Hand transmitted vibration clinical effects and pathophysiology.* Parts 1 and 2. London: Faculty of Occupational Medicine Royal College of Physicians of London, 1993.

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The ABC of Work related disorders is edited by David Snashall, clinical director of Occupational Health Services, Guy's and St Thomas's Hospitals NHS Trust, London.

Until recently this was known as vibration induced white finger (VIWF), reflecting its mode of presentation. It has been renamed to reflect the wider range of its effects. The Taylor-Pelmear classification is also being replaced by the Stockholm Workshop scale.

Though originally described in the mining, metallurgical, and engineering industries, forestry may now be the main source of cases because of widespread use of chainsaws. How vibration actually induces the condition is not clear, but recent studies suggest it is probably a local effect on nerves and blood vessels.

Presentation

The presentation is of progressive Raynaud's phenomenon in an operator of vibrating tools. After a latent period, vasospasm is first noticed on cold, wet, and windy mornings in the tips of the fingers most affected by vibration. Intermittent numbness or tingling often precedes actual blanching.

With continued exposure to vibration, the vasospasm progresses proximally and to other fingers, but the thumb is rarely affected. The vasospasm has a characteristic pattern: it is rarely symmetrical and reflects the subject's grip on the vibrating tool—thus, an underhand grip on a vibrating chisel affects mainly the index finger, whereas the little finger is mainly affected with an overhand grip. After the painless vasospasm ("dead finger"), a cyanotic phase may occur before a painful hyperaemia ("hot aches"). In some cases only cyanosis occurs with vasospasm. In the most severe cases the fingers are permanently cyanosed and trophic changes of the fingertips can occur. The intermittent neuropathy that accompanies the vasospasm becomes continuous and deteriorates with further exposure. It affects most modalities and results in clumsiness.

Unless it is associated with cold, vibration itself rarely induces vasospasm. As the condition develops, the amount of cold required to induce vasospasm is less and the condition can occur in the summer, though usually in association with wet hands or wind.

Diagnosis

There is no single diagnostic test. Though it is often difficult, an attempt should be made to induce vasospasm by cooling the hand. Neurological testing shows most sensations to be dulled. Carpal tunnel syndrome (reportable under RIDDOR for workers exposed to vibration) should be excluded by Tinel's sign or Phalen's test.

Prognosis

The vascular component can slowly improve if exposure to vibration is stopped early in stage 2, and a change of job should be encouraged. It is debatable whether the neurological symptoms can improve.

Assessing risk

Risk of the condition is related to the vibration dose received by the hand, the strength of grip used, the type of vibration, the ratio of work and rest periods, and factors affecting the circulation such as body warmth and smoking. There is great individual variation. The condition seems to be more common with low frequency vibrations but has been reported with frequencies up to 1500 Hz. Accelerometer readings can be taken on a vibrating tool or the interface between hand and tool to evaluate the potential risk.