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NOISE-INDUCED TINNITUS

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Tinnitus has recently been defined[1] as "the sensation of sound not brought about by simultaneously applied mechano-acoustic or electrical signals". It has many causes and associated factors, probably the most common respectively being noise exposure and the degenerative processes associated with ageing[2,3].

Cause(s) of Tinnitus. There are very many possible causes. In the majority of cases we are at present unable to identify the cause. In particular, just because a person has noise-induced hearing loss does not necessarily mean that the tinnitus is also noise induced, although, as will be discussed below, tinnitus is more frequent in persons who have been noise-exposed.

The most useful classification[1] is not by cause but by probable site:
(i) Peripheral: external ear, middle ear, cochlea or eighth cranial nerve
(ii) Central: in the central auditory neural pathways,with their subdivisions
(iii) Extra-auditory: e.g. vascular, nasopharyngeal, muscular (extratympanic)
(iv) Unknown: includes any suspected site where the topographical probability is assessed at 50% or less. At the present state of the art, even the site of tinnitus in the majority of cases will not be known with sufficient probability to justify any classification other than unknown.

It is uncommon to find a case of troublesome tinnitus without some other identifiable hearing disorder, most commonly sensorineural hearing loss. Tinnitus is a frequent part of the sympomatology of the pathological disorder in the cochlea. In many ways it can be likened to pain in the hearing system. There is some evidence for its presence being due to an increase in the rate of spontaneous discharge of some auditory nerve fibres[4] and this certainly accords with a widely held concept of what tinnitus is. It seems unlikely that external or middle ear disorders per se often cause tinnitus. The tinnitus commonly associated with them probably amounts to an enhancement by occlusion effects or reduction of masking by ambient noise of (a) sensorineural tinnitus either secondary to the middle-ear disorder or coincident, (b) physiological noise, usually of a humming or pulsating type, or possibly (c) spontaneous acoustic emissions from the cochlea[5].

Epidemiology of Tinnitus. In the pilot study of the Institute of Hearing Research's current National Study of Hearing (NSH), a questionnaire on hearing difficulties and tinnitus was sent to a random sample of 6804 persons living in Cardiff, Glasgow, Nottingham and Southampton. This procedure, called Tier A, enabled stratification of respondents by age groups and reported impairments, and thereby permitted appropriate sub-samples from those strata to be invited to attend the clinics for (Tier B) clinical and audiological investigation. Response rates were around 80% at Tier A and 50% at Tier B; domiciliary follow up showed the biases in non-responders and non-attenders to be minimal. The percentage of respondents in each city who reported tinnitus, hearing impairment, or both are shown in Table 1.

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	Cardiff	Glasgow	Nottingham	Southampton
T	9.6	9.2	9.5	8.3
HIT	8.6	9.4	8.6	7.2
HI	15.2	18.9	18.5	16.7
N	66.5	62.4	63.5	67.7

Table 1. Percentages of respondents in each city reporting tinnitus (T), hearing impairment (HI), both (HIT) or neither (N).

In a pre-pilot study carried out in Glasgow, people were asked if they had ever experienced a buzzing or ringing in the head or ears: the positive response rate was 39% not unlike the 21-39% prevalences in various age groups reported by Hinchcliffe[6] for a similar question. For the pilot study the question was altered to exclude those people who had experienced only an occasional whistling or ringing in the ears of less than 5 minutes duration or where it occurred only just after very loud sounds, e.g. discos, shooting or noise at work. Table 1 indicates areported tinnitus figure over all four cities of about 17%. This is taken to represent the experience of material tinnitus in the adult population of this country at a severity for the tinnitus being of potential relevance for audiological diagnosis (though not necessarily as the primary basis for seeking help).

A total of 272 persons were seen in the outstation clinics at Glasgow and Southampton in the pilot study at Tier B, and consequently the analyses have been largely restricted to points needed to guide further work. Nevertheless, some useful data have resulted. Table 2 shows that the questionnaire produces sufficiently divergent audiometric profiles at Tier B for its strata to be used as the bases of our sampling strategy for the main study. In both N and HI strata, and in both better and worse ears, the average hearing level increases quite markedly when there is a present or past history of tinnitus (T). It would seem that, in the N stratum, tinnitus is an early sign of auditory disorder in which the associated hearing loss is still subclinical. Just why the presence of tinnitus in addition to hearing difficulty is an indicator of a more severe or advanced state of disorder deserves further examination.

Stratum	BEA	WEA	mean s.d. •
N	11	15	9
T	17	25	14
HI	26	33	18
HIT	40	53	23

No, no complaint; T, reported tinnitus; HI, reported hearing impairment; HIT reported hearing impairment and tinnitus. BEA and WEA = better and worse ear hearing thresholds averaged across 0.5, 1, 2 and 4 kHz. • mean s.d. is that for the better and worse ear standard deviations.

Table 2. Mean hearing levels (dB re ISO 389: 1975) for each stratum.

Table 3 gives further details of the effects of tinnitus as reported at Tier A. It can be seen that about 5% of the sample reported moderate or severe annoyance from tinnitus, and a similar proportion reported some degree (not quantified) of sleep disturbance by tinnitus.

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	Cardiff	Glasgow	Nottingham	Southampton
Tinnitus	17.9	18.6	18.1	15.5
Annoyance : moderate	4.1	4.1	4.4	3.8
severe	0.7	2.8	0.4	0.7
Sleep disturbance	3.8	7.3	5.4	4.4
Effect on life:severe	0.4	0.5	0.4	0.5

Table 3. Percentages of adult population reporting tinnitus and its effects.

Cross-tabulations of annoyance, effects on life as a whole and sleep disturbance showed that, although these effects are related, the association is far from perfect. This has been observed previously in patients complaining of tinnitus (J W P Hazell 1980, personal communication), and noted also in the clinical work of this Institute. About 20% of patients who are severely affected by tinnitus report little or no interference with sleep. The importance of this is that if one relies solely on reported sleep disturbance as a yardstick of severity of a patient's tinnitus, 20% of serious cases may be missed.

	Age (years) 18-39	40-60	over 60
No noise	11.1	13.2	18.2
Noise	21.9	21.4	32.8

Table 4. Percentage of population of four cities (combined) who reported tinnitus, as a function of reported occupational noise exposure and age.

Table 4 shows the influences of age and noise in the determination of tinnitus. Presence or absence of noise exposure was derived from one simple question in Tier A: 'Have you ever worked in a place for more than 6 months which was so noisy that you had to raise your voice to be heard?' Hinchcliffe[6] used a similar question on noise exposure: he found an age effect, but not a noise effect. It is apparent that, within each age group, a history of noise exposure almost doubles the risk of tinnitus. Analysis of more recent NSH data suggests that this is true of continuous tinnitus, but the increase in risk of intermittent tinnitus is rather less (35-50%). A doubling of risk is generally regarded as within the range meriting preventative measures in occupational and environmental health. However, there is a similar trend for higher prevalence of tinnitus with increasing age and increasing age must inevitably correlate with the number of years of unprotected exposure to noise. The relevance of this is not negated by the existence of a similar trend in the no-noise group, since the Tier A question did not cover military and other shooting noises, acoustic accidents, etc. It seems likely that both noise and age are main determinants of population tinnitus, but whether they are interactive or are additive remains to be determined from the main study with its detailed assessment of noise immission ratings at Tier B.

It has several times been reported from tinnitus clinics that left-sided tinnitus is more common that right-sided. We found a similar trend in our pilot study[2,3]. However, analysis of population-based data from Phases I and II of the NSH showed no consistent evidence that when unilateral tinnitus occurs it is more commonly on one side than the other. This is most definitely the case where noise exposed men are concerned, although where there had been a

clearly asymmetric noise exposure tinnitus was more commonly found in the more exposed ear.

Management of Tinnitus. The present study has already yielded an important indication of the potential size of the clinical problem: around 200 000 persons in this country are likely to have had their lives severely affected by tinnitus, and many more are affected to a lesser degree. This points to the need for allocation of corresponding research and clinical resources. Tinnitus maskers are beginning to enter clinical practice. When supported by expert patient counselling, they can give excellent results in a significant, if minority, proportion of tinnitus patients and worthwhile results in the majority.

Tinnitus self-help groups are now widespread and many patients are obtaining much benefit from membership of the British Tinnitus Association (inaugurated in 1979, and administered by the Royal National Institute for the Deaf) and from reading its regular newsletter and attending meetings of local branches of the Association. In the absence of elaborate rehabilitative services, making patients aware of these groups is an essential part of present management of the patient with tinnitus. Other lines of management include use of drugs. These may simply be to counter the secondary effects of tinnitus, e.g. use of sedatives, tranquillisers or antidepressants, or to attempt to treat a suspected vascular cause, e.g. with vasodilator drugs, or as a palliative. Of the latter, the best yet available seems to be tocainide, although unfortunately only a minority combine benefit from the drug with freedom from prohibitive side effects. In a few cases, the cause of the associated hearing loss is found or a general bodily disorder discovered; the appropriate medical or surgical treatment is given and the tinnitus may also be reduced or abolished thereby. Other treatments under evaluation include psychotherapy with or without bio-feedback, physiotherapy along lines sometimes used for cervical vertigo, and even direct electrical stimulation. The field of tinnitus research, clinical investigation and therapy is now an expanding one that is beginning to dispel the sense of ignorance and hopelessness that has surrounded the subject for so long.

Noise-Induced Tinnitus. It is too early yet to think in terms of this as a condition that can be diagnosed and quantified in such a way as to be usable as an additional damage risk criterion for noise exposure. But it is an added handicap in cases of noise-induced hearing loss that deserves greater attention in occupational medicine and hygiene than it has received to date, and should act as a further spur towards institution of effective hearing conservation procedures. Probably about 50% of well defined cases of noise-induced hearing loss have some degree of noise-induced tinnitus. In perhaps 10% if causes significantly increased handicap. In occasional cases, they are so severely affected by tinnitus that the tinnitus becomes by far the greater disability.

In the compensation field, severe tinnitus can be counted as a "greater disablement" under the present governmental (DHSS) scheme for Occupational Deafness. Noise-induced tinnitus per se is not compensatable unless the result of an acute accident, acoustic or other. The problem is one of diagnosis and quantification; both are most imprecise, and moreover there is no means of objective verification. To compensate fully for it would be to invite gross abuse of the system.

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In civil legal actions, there is greater flexibility and cases of noise-induced tinnitus are coming forward with increasing frequency. In several instances they have, rightly, attracted greater compensation than have the more severe cases of noise-induced hearing loss. But it is not easy to prove or disprove. Apart from the usual history taking of noise exposure and its temporary and/or permanent effects (hearing loss and tinnitus), and the audiological investigation of the hearing loss, there are some useful psycho-acoustic tests of tinnitus. The relation of the tinnitus pitch-match frequency can help to indicate whether the tinnitus is more or less likely to be noise-induced. The final diagnosis depends on the combination of all evidence available, but with tinnitus of unknown cause being so common it is often very difficult - especially so when the troublesome tinnitus comes on years after the last noise exposure. Quantification largely depends on general assessment of the claimant's credibility, which can be much helped by objective checking of the hearing thresholds, coupled with his description of the loudness of the tinnitus and of its effects on his general well-being.

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