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ASSESSING HEARING IMPAIRMENT: PRESBYACUSIS IN THE PRESENCE OF NOISE-INDUCED HEARING LOSS

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INTRODUCTION

Glorig and Nixon's (1960) assertion that ageing and noise exposure are the only factors that influence hearing levels in the general population is commonly subscribed to and this is supported by the recent data obtained by Burns and Robinson in this country. However, as has been demonstrated from population studies in Jamaica (Hinchcliffe, 1968), this assumption may not be tenable for populations outside Europe or North America. Moreover, in assessing to what extent occupational noise is responsible for impaired hearing in a given individual as opposed to a population, many other factors must be considered. Furthermore, where the question of compensation arises we shall have to determine how much of the hearing loss is functional (non-organic). Our experience indicates that, in cases where compensation is involved, it is not a question of whether or not there is a functional component, but to what extent there is a functional component. Finally, the multi-causality of presbyacusis (Weston, 1964; Hinchcliffe, 1968), let alone the question of whether it exists as a separate entity (Schmidt, 1967), poses considerable problems for the physician who is confronted with an older person who has spent a lifetime working in one or more noisy industries.

PRESBYACUSIS

Zwaardemaker (1893) coined the term "presbyacusia" to denote the poorer hearing of elderly people. Although Glorig and Nixon (1960) would restrict the use of the term to hearing losses due to physiological changes with age, others would apply the term to any sensorineural hearing loss occurring in old age. However, the audiogram in presbyacusis characteristically shows a gradual fall-off of the hearing level with positive acceleration towards the higher frequencies. Population studies, e.g. in Britain (Hinchcliffe, 1959a) and in the U.S.A. (Glorig et al., 1957), have consistently shown that, with increasing age, the hearing levels of negligibly noise-exposed and otherwise otologically normal people show a corresponding deterioration which is positively accelerated and is more marked for the higher frequencies. Thus average values are available for the purposes of making "presbyacusis" corrections to data for noise-exposed populations. Robinson (1968) has indeed pointed out that analysis has indicated that subtraction of a standard "presbyacusis" correction was beneficial to the data reduction.

Further analysis (Hinchcliffe, 1962a) of these average hearing levels on negligibly noise exposed and otherwise otologically normal

populations showed that, when the intensity of the threshold stimulus was expressed in terms of a quantity $(\emptyset-\emptyset_k)$, where $\emptyset$ is the physical intensity of the threshold stimulus and $\emptyset_k$ is the physical intensity of some reference level, an exponential decrement with age was exhibited. Moreover, a single curve seemed to fit the data for all frequencies from 250 Hz to 4000 Hz. Furthermore, all sensory threshold sensitivities appeared to exhibit this exponential decrement with age. Thus this ageing phenomenon of threshold sensitivity is not confined to hearing. However, not only do the majority of older people have poorer hearing levels than younger people, but they also have other auditory deficits, which have previously been listed (Hinchcliffe, 1962b). In particular, there may be a marked loss in the discrimination of both undistorted speech (Gaeth, 1948) and of distorted speech (Bocca, 1958). The latter condition, particularly, is indicative of impaired temporal lobe function so that, not unnaturally, it has been argued that many of the features of the auditory disorders with which the elderly are afflicted are primarily of central, rather than of peripheral origin (Hinchcliffe, 1962b). Nevertheless, degenerative changes have been described in various parts of the auditory mechanism so that a pathological basis for degenerative auditory disorders (perhaps a better, more descriptive, term than presbyacusis) would appear to exist. Beginning peripherally and moving centrally, the following changes have been reported: changes in pinna size (Hsi-Kuei et al., 1958); atrophy of the walls of the external acoustic meatus (Babbitt, 1947); calcification of the basilar membrane (Mayer, 1920); spiral organ degeneration - primary (Crowe et al., 1934) or secondary (Fieandt and Saxen, 1937); stria vascularis atrophy (Fleischer, 1952); spiral ganglion degeneration (Crowe et al., 1934); hyperostosis senilis progressiva meatus acoustici interni (Sercer and Krmpotić, 1958); degenerations in the auditory neurones in the brain stem (Kirikae et al., 1964); and changes in the cerebral hemispheres (Hansen and Reske-Nielsen, 1965). The question, however, arises as to whether these degenerative changes in the auditory subsystem are primary or secondary. Korenchevsky (1961) has endeavoured to discriminate between physiological and pathological processes of ageing and between physiological and pathological causes of ageing. He also points out that overwhelming evidence has accumulated to prove that present day old age is an abnormal pathological syndrome in which physiological processes of ageing are complicated and aggravated by the various so-called degenerative diseases of old age.

It could be therefore argued that, with respect to hearing, the principal causes of presbyacusis, viewed as a clinical diagnosis, are pathological ones. Indeed Stein (1928) considered that arteriosclerosis was the principal aetiological factor in presbyacusis and our analysis of Fabinyi's (1931) data indicates that the severity of the hearing loss in the elderly is related to the degree of degenerative arterial disease. Moreover, the importance of degenerative arterial disease in the aetiology of presbyacusis was reasserted at the last (IX) International Congress of Audiology both by Rosen (1969a) and by Bochenek and Jachowska (1969). Furthermore, Rosen and Olin (1965) stated that a longitudinal study in Finland had shown that institution of a non-atherogenic diet is associated with better hearing levels, and these trends are reversed when the diets of the control and the experimental groups are interchanged (Rosen, 1969b). Notwithstanding the assertion of Loeper and his associates (1961) that lipid infiltration is the consequence and not the cause of the vascular degenerative changes in atherosclerosis, Stamler (1960) points out that, almost without exception, experimental athero-

sclerosis close to resembling that in man has been associated with hypercholesterolaemia. Although it is possible to measure and to quantify the level of cholesterol in the blood, and to measure arteriosclerosis by external recordings (Cooper et al., 1967), it is still questionable to what extent one can apportion a person's hearing loss to vascular degenerative changes, acting either directly on the auditory apparatus or indirectly by accelerating the physiological ageing. However, as Robinson (1968) has shown, utilizing data on essentially non-noise exposed populations, it is possible to utilize "presbyacusis" corrections and, with the knowledge of the dispersion of the data, it is possible to assess the probability with which a given hearing loss is likely to be due to the "ageing process", having, of course, adequately assessed the person's audiological status and being cognisant that presbyacusis type curves are not characterized by notching. The pathological basis of presbyacusis - as a degenerative disorder which generally involves the whole auditory subsystem, but with variable emphasis in different people, including non-involvement of the spiral organ (Hallpike, 1962) - probably affords an explanation for the simple additivity of "presbyacusis" and noise-induced hearing loss, the lesions of which are typically and discretely located in the basal turn of the spiral organ.

ASSESSMENT

The audiological assessment of an individual where noise-induced hearing loss is suspected is very much the same as that where any other hearing loss is under investigation, with the exception that, certainly where compensation is involved, a non-organic component in the hearing loss must be excluded. Since malingerers usually follow equal-loudness contours for their simulated auditory thresholds, a sharp high tone notch or an abrupt cut-off in the threshold audiogram almost certainly precludes a functional component, which would, however, be indicated by a type V Békésy audiogram (Jerger and Herer, 1961). In such an event, evoked response audiometry must be conducted to quantify the non-organic component (Beagley and Knight, 1968).

Abnormal compliance versus excess intrameatal pressure curves on tympanometry indicate a conductive component in the hearing loss which can only be quantified by combined air and bone conduction audiometry.

The occurrence of temporary threshold drift, either as Carhart's (1957) tone decay on manual audiometry, or a disparity between pulsed test tone and continuous test tone recordings on Békésy audiometry, indicates a neuronal lesion. Since noise-induced hearing loss is essentially a spiral organ lesion (Igarashi et al., 1964), demonstration of any of the previously cited phenomena indicates that factors other than noise have been, or are being, responsible for the hearing loss. Noise damage preferentially produces hair cell damage at a point located 10 mm. distant from the basal end of the spiral organ, so that audiograms showing other than a high tone notch or an abrupt cut-off at high frequencies, e.g. low tone notches or predominantly low frequency hearing loss (as in endolymphatic hydrops), again indicate factors other than noise in the aetiology of the hearing loss. Even when one has found that a person who has been exposed to occupational noise has, after a "presbyacusis" correction, a high frequency loss with no conductive or non-organic component, and no evidence of a neuronal lesion, one is still not justified in attributing the loss to the noise since there are again many factors which can produce

degeneration of the basal turn of the spiral organ (assuming that the more specialised audiometric tests indicate that this is the locus of the lesion). Aside from congenital aplasias (Scheibe's cochleo-saccular test), there is the possibility of infections, tumours of the stato-acoustic nerve (admittedly rare) and a consequence of ototoxic drugs. Gannon's (1969) study is particularly relevant to the latter factor. Even though noise exposure levels or drug blood levels may, in the absence of the other factor, be insufficient to cause a hearing loss, the two together may summate to produce hearing loss in what otherwise would have been a condition where no hearing loss would have resulted. Hearing loss due to head injury is a special case of stimulation hearing loss and is attributed to generation of a high-intensity transient that reaches the cochlea by bone conduction (Schuknecht and Tonndorf, 1960).

Even when we find that a man, who has been working for an appreciable length of time in a noisy industry, has a classical high tone traumatic notch, it may not be justifiable to attribute this to the noise. The majority of these people would have used fireworks in childhood and/or fired guns, in connection with military service or as a hobby, prior to engaging in the occupation. Although, in random samples of the general population, it was found that measured hearing levels correlated with both the number of rounds of 0.303 ammunition fired (Hinchcliffe, 1959b) and the number of times a 12 bore gun was used (Hinchcliffe, 1961), Atherley and Noble (1969) failed to demonstrate, on a number of occupational groups, a relationship between a history of previous exposure to military gunfire and present hearing level. This discrepancy is yet to be resolved. Otopathologists would, however, be loath to attribute the latter finding to complete recovery of the hearing from the effects of acoustic trauma. It is possible that, in these samples, the effects of gunfire have been obscured by the many other factors which influence hearing level. This would also probably explain the finding that, in an unselected sample of patients attending a neuro-otology clinic, a history of previous, or contemporary, exposure to either acoustic trauma and/or occupational noise is not correlated with the occurrence of high tone notches in the auditory threshold. Or must we conclude that not only gunfire but also occupational noise does not have any long term effects on hearing?

CONCLUSIONS

Because of the uncertainty of the degree to which other factors, whether they be in the nature of noises, noise-equivalents or otherwise, have influenced a hearing level before a person enters a given occupation, one can only say, with any reasonable degree of certainty, that occupational noise has influenced a man's hearing by being in possession of both pre- and post-employment audiograms. Even this safeguard does not enable us to exclude other factors which might damage hearing and have operated during the period of the man's employment.

SUMMARY

It would appear that attempts to attribute any hearing loss to noise exposure, and to apportion the loss to this factor, is not a simple exercise and is fraught with difficulties. Nevertheless, an audiological assessment is possible but conclusions must rest, as elsewhere in medical diagnosis, on a question of probabilities.