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Clinical records

Hearing loss due to noise trauma

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Abstract

THREE different types of noise-induced hearing loss have been reported in the literature. The two less common types are described here. Three cases of these two types from our clinic are reported. Since these 2 types of noise-induced hearing loss often involve low frequencies it is important to recognize them in compensation cases.

The most commonly recognized noise-induced hearing loss (NIHL) is the insidious type caused by chronic noise exposure. It is manifested generally as a high-frequency sensorineural hearing loss with a notch in the audiogram at the 3 to 6 kHz area. Two other types of NIHL are also known but occur much less frequently. One of them is a sudden hearing loss due to a single exposure to an intense sound stimulus. Hearing loss mostly involves high frequencies but it may also occur at low frequencies. This is termed explosive noise-induced hearing loss (ENIHL) here. The third type of NIHL is also a sudden hearing loss but is distinguished from ENIHL in that it occurs abruptly after a certain period of noisy work at an intensity level no higher than that previously exposed to. The period of noisy work varies from days to years. This type of NIHL is denoted here as accidental noise-induced hearing loss (ANIHL). Becker and Matzker (1961) described two such cases and referred them as 'akustischen unfall (acoustic accident)'. This is the reason that the term ANIHL is chosen here. Audiograms of this type usually have flat or U-shaped configurations.

Numerous reports can be found on the insidious type of NIHL. It will not be elaborated further here. However, ENIHL and ANIHL are less common and are the types that we are concerned with here.

In a study reported by Ziv *et al.* (1973) both ears of 77 soldiers and sailors who had suffered blast injuries were examined. 41·1 per cent had some kind of hearing loss. 5·8 per cent of these acquired flat or sloping sensorineural hearing loss and 21·9 per cent suffered high-tone hearing loss only. The ones with the flat hearing loss are interesting since they deviate from the more common type of high-frequency notched NIHL. The mechanisms of injuries for producing these two types of hearing loss are likely to be different. The high-frequency notched hearing loss is probably the type described by Spoendlin (1976): ruptures of the reticular lamina and detachment of the organ of Corti from the basilar membrane. The hearing loss acquired in this manner remains more or less localized at the high-frequency areas. However, if the injury involves the rupture of Reissner's membrane, the oval window, and/or the round window, it may cause a hearing loss also at low-frequencies (Simmons, 1968; Goodhill, *et al.*, 1973; Lyons *et al.*, 1978). Symptoms of this latter type could be very similar to those of

Menière's disease. One case has been reported by Pulec (1972) in which a sergeant after being exposed to an Air Force jet of about 160 dBA developed a classic Menière's disease in one ear two weeks later.

Twenty cases of ANIHL were reported by Kawata and Suga (1967). Most of them had asymmetrical U-shaped or flat audiograms. Only two cases demonstrated abnormal vestibular function. Cases with this type of hearing loss also have been described elsewhere although they were not identified as ANIHL (Pulec, 1972; Lyons *et al.*, 1978). In one case it was actually depicted as Menière's disease caused by noise. Pulec feels that the incidence of Menière's disease due to loud noise exposure is about 2 per cent.

In this communication 3 cases are reported. Two of them can be considered as the type ENIHL and one as ANIHL.

Report of cases

Case 1

This is a 36-year-old man who was sent to the Hearing Branch of the Workers' Compensation Board for a claim investigation after he was exposed to a small blast of nitroglycerine detonation about eight feet away in a large room on 6 July 1973. Following this explosion, he noticed a sudden onset of ringing tinnitus, a plugged feeling and a hearing loss, all in his left ear. He did not experience any pain. The tinnitus and hearing loss lasted for several hours. They were not accompanied by any dizziness or disequilibrium. By the next day, the tinnitus had completely disappeared and the hearing had improved. On 8 July 1973, he developed hearing loss and tinnitus exactly as he had experienced on the previous Wednesday. He began to feel dizzy as he got up from the chair and felt as though he was leaning and staggering towards the left side. He also had some whirling rotating vertigo but no nausea or vomiting. This persisted for about two hours. Since then he has had repeated attacks of a similar nature and has noticed that on a few occasions his dizziness would recur when exposed to loud noise. Gradually his symptoms became less frequent until November 1973 when he had a severe attack much worse than any of the others he had experienced, with vertigo, tinnitus and hearing loss in the left ear accompanied by nausea lasting approximately two hours. Since then he had not had any hearing test until July 1976 when he was shown to have a flat loss of about 50 dB in the left ear. He was seen again in August 1976 when his hearing showed some improvement. When he was last seen in December 1976, he reported no further dizzy spells and fluctuating hearing loss. Electronystagmographic tests showed normal results.

Case 2

This 36-year-old man was investigated at the Hearing Branch of the Workers' Compensation Board for a hearing loss on 3 March 1976 and again on 21 April 1976 at which latter time he also received vestibular testing.

He first had his hearing tested in 1972, when he had an audiogram for a check-up while having a septal reconstruction done on his nose by an ENT specialist. At that time, he had a bilateral notched 4000 Hz hearing loss with normal thresholds at low frequencies. A repeated audiogram in September 1974 was virtually identical to that obtained in 1972.

Apparently, this patient went to see an ENT specialist on 4 November 1975 because he thought the hearing in his right ear 'seemed to have slipped'. An audiogram was done and showed that he had the same notched 4000 Hz hearing loss in the left ear but an additional 50 dB hearing loss at the low frequencies in the right ear. He was then sent to the Audio-Vestibular Unit at Vancouver General Hospital for investigation for a possible retro-cochlear lesion. When tested on 21 November 1975 it was found that he had a notched high-frequency sensorineural hearing loss somewhat worse in the right ear than the left, but the low frequencies were again normal and symmetrical in both ears.

In January 1976, this man was standing beside a compressed air-operated vibrator when it was turned on suddenly. There was no blast of air but there was a sudden onset of very loud noise and the patient felt a sudden pain in his right ear. He rushed away from the noise and realized that he had an intense ringing tinnitus in his right ear with severe hearing loss, about 50 dB HL at low frequencies. This time his hearing loss did not recover. His hearing loss was still the same when he was tested again in May 1977. Impedance audiometry showed normal middle-ear function. He still has tinnitus in his right ear which changes in pitch.

Electronystagmographic tests showed responses within normal limits. He also reported no dizziness or balance problems.

Case 3

This 56-year-old man was examined at the Hearing Branch on 30 August 1977. Between 2 April and 10 April 1965 he was working inside a lime rock tower with a jackhammer. He was in there for at least five to six days and, although he was wearing ear plugs, they kept falling out because of the vibration. At the end of each day's shift he would have a ringing tinnitus and a temporary hearing loss which would usually clear up by the next day. However, on the sixth day, he got up in the morning to find that his right ear was still ringing but his left ear had recovered. He also realized, at that time, that he had a hearing loss in his right ear. Audiometric tests later confirmed a flat sensorineural hearing loss (50 dB) in his right ear. He did not have any vestibular symptoms either at the time of the incident or following it.

Electronystagmography showed responses within normal limits. He also had a history of syphilis.

Discussion

Case 1 clearly demonstrates an example of ENIHL with Menière's symptoms. Case 2 is also a type of ENIHL but it suggests that the noise aggravated an existing endolymphatic problem. The third case should be considered as ANIHL and it is very similar to those reported by Kawata and Suga (1967). The timing of the occurrence of the sudden low-frequency hearing loss in all three cases with the episodes of acoustic trauma is too much of a coincidence for one to dismiss noise as the cause of the hearing loss.

There are various theories on the mechanisms of acoustic damage to hearing and they have been reviewed by Bohne (1976). The evidence which these theories are based on is mainly obtained from animals and humans with the high-frequency notched hearing loss. Using this type of theory to explain the cause of the sudden low-frequency hearing loss by noise may not be appropriate.

The causes secondary to acoustic trauma of the ENIHL and ANIHL of the low-frequency type have been attributed to round window rupture and endolymphatic hydrops formation (Pulec, 1972; Lyons *et al.*, 1978). The authors who found the round window ruptures suggest that very low frequency sound is responsible for the rupture and susceptibility increases with the size of the helicotrema. At very low frequencies fluid flow through the helicotrema would increase its effect, thereby exerting greater pressure on the round window membrane.

Although the possibility of noise trauma inducing the formation of endolymphatic hydrops seems somewhat far-fetched, the fact that it does occur cannot be ignored. Perhaps in the few cases reported in the literature, noise was just a potentiating agent which interacts with an already malfunctioning endolymphatic system. The theory of rupture for endolymphatic hydrops postulates an inadequacy of reabsorption of the normally produced endolymph in the endolymphatic system (Schuknecht, 1975). As a result of this, there is a progressive accumulation of endolypmh with distension of part of the membranous labyrinth which eventually ruptures causing the leakage of endolymph into the perilymph. The appearance of high concentration of potassium ions in the perilymph impedes the function of hair cells and neurons which, in the vestibule, provokes acute episodes of vertigo and, in the cochlea, induces fluctuating hearing loss. Also, Lipscomb (1975) hypothesized that continuous, intense noise would cause an elevation of endolymphatic fluid pressure which, in effect, would by creating an inner-ear conductive hearing loss biasing against the low-frequency sound due to the increase in stiffness of the system. If all these are correct, noise exposure can aggravate an already existing endolymphatic problem, or incite the malfunction of the endolymphatic system.

Concluding remarks

The effect of noise is more far reaching than is generally recognized. Since it affects the cochlea as many other ear pathologies do, it is likely that it interacts with them and aggravates the situation. This has been shown in noise and ototoxic drugs (Gannon *et al.*, 1979). Noise of very high intensity can also cause membrane ruptures other than hair-cell damage. Therefore, in considering compensation cases with NIHL, low-frequency hearing loss should not always be treated as non-occupational.

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