

Medicolegal significance of asymmetrical hearing loss in cases of industrial noise exposure

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Abstract

Objective: In Australia, the current guidelines for evaluation of noise-induced hearing loss suggest that, in cases of asymmetrical loss, ‘the worse ear be equated to the better ear’ for purposes of compensation. This study aimed to establish that such a method was prejudicial to the worker (i.e. the plaintiff). In consideration of the legal duty ‘to co-construct the ideas of truth and the ideas of justice in the context of legal proceedings’, our study objectives were (1) to document the incidence of asymmetrical hearing loss in compensation cases seen in our practice, and (2) to provide a reasoned argument for inclusion of the same for compensation considerations.

Study design: Open, retrospective, clinical study.

Setting: Australian plaintiffs with asymmetrical hearing loss (who comprise a significant percentage of industrial hearing loss legal cases) may be excluded from full consideration of their hearing loss as a result of the current guidelines. In contrast to the process of medical diagnosis and treatment, it appeared that the application of accepted probability standards within the legal process may permit inclusion of such clients’ hearing loss in compensation considerations.

Methods: This study included 208 consecutive clients referred by legal practitioners for assessment of hearing loss for compensation purposes.

Results and conclusion: A total of 22.6 per cent of clients (47 of 208) had asymmetrical hearing loss, with the left side having the greater loss in 60 per cent of cases. We believe that asymmetrical hearing loss should be included in compensation considerations, both on medical and legal grounds.

Key words: Hearing Loss; Noise Induced Hearing Loss; Medico-legal; Australia

Introduction

The association between noise and hearing loss has been known for a long time. It appears that noise-induced hearing loss may date back to the Bronze Age, when an alloy of copper and tin was used to fashion weapons and tools in the Near East (circa 6000 to 2000 BC).¹ In the first century AD, Pliny the Elder, in his *Natural History*, noted that persons living near the cataracts of the Nile were ‘stricken deaf’.² This connection was also recognised by the early physicians Ambroise Paré (1510–1590) and Sir Francis Bacon (1561–1626). The discovery of gunpowder (causing acoustic trauma), the Industrial Revolution and its associated mechanisation of factories and transport (resulting in occupational noise-induced hearing loss), and the later development of noisy hobbies and amusements (leading to socioacusis) all contributed to the social incidence of noise-induced hearing loss.

Studies on animals (by Wittmaack)³ and on human volunteers (by Davis *et al.*)⁴ have given insight into the pathology of noise-related hearing damage. In

modern times, Russia was the first government to recognise officially (in 1929) that noise damage to hearing was a (compensable) occupational disorder.⁵ In the US, one-third of hearing impairment is attributable to noise exposure,⁶ and is irreversible.

Unlike presbycusis, noise-induced hearing loss is entirely preventable and does not progress after discontinuation of the noise exposure. Upon ongoing exposure to the same noise, as the severity of resultant hearing loss increases, its rate of progression decreases.

Noise-induced hearing loss is generally accepted by otologists to be symmetrical, except in long gun shooters and single headphone users, for whom exposure is not similar for both ears.

Dobie⁷ has outlined criteria for the diagnosis of occupational noise-induced hearing loss: a slowly progressive, almost always bilateral, sensorineural hearing loss (SNHL) is involved; the loss is always greater at the frequencies of 3–6 kHz; and high frequency losses rarely exceed 75 dB while low frequency losses rarely exceed 40 dB.⁸

Materials and methods

We undertook an open, retrospective study of 208 consecutive clients referred for assessment of hearing loss for compensation purposes, as per the *NSW Workers Compensation Act 1987 Section 69A*. Clients were referred by legal practitioners. The 208 clients were seen between March 2006 and September 2007, and comprised 203 men and five women, aged 36 to 73 years.

Diagnosis was based on the requisite history of substantial noise exposure at work, audiogram results (showing a hearing shift at high frequencies with a typical notch at 4–6 kHz) and elimination of competing diagnoses by an ENT expert.

This study aimed (1) to document the incidence of asymmetrical hearing loss in compensation cases seen in our practice, and (2) to provide a reasoned argument for inclusion of the same in compensation considerations.

Asymmetrical hearing loss was defined as loss of 10 dB or greater for two consecutive frequencies, or of 15 dB for any one frequency between 0.25 and 6 kHz.

Results

Section 69A of the *NSW Workers Compensation Act 1987* does not allow compensation for a hearing loss of less than 6 per cent. We encountered 81 such clients in our study. Although these clients were eligible to apply for reassessment after three years, three moved to quieter work environments and were thus ineligible for compensation despite measurable noise-induced hearing loss.

Of the total 208 clients, 47 had asymmetrical hearing loss as defined (46 men and one woman). Asymmetrical hearing loss was thus encountered in 22.6 per cent of our clients. A history of definite asymmetrical noise exposure (involving sudden impact noise) was volunteered by three clients with asymmetrical hearing loss (6.4 per cent); the remainder ($n = 44$) were unable to recall such exposure, even on close questioning. The 47 clients with asymmetrical hearing loss underwent magnetic resonance imaging (MRI) scanning for central pathology; none was found. Of these 47 clients, 60 per cent had greater hearing loss in the left ear.

Of our clients with hearing loss, 33 were truck drivers. In this subgroup, in contradiction to the findings of Dufresne *et al.*,⁹ 16 clients had symmetrical hearing loss and 17 asymmetrical hearing loss. Of the latter 17 clients, 12 had greater hearing loss in the left ear and five had greater loss in the right ear.

Discussion

The diagnosis of noise-induced hearing impairment is based on assessment of the type and duration of noise exposure. Although the relationships between these factors are set out in various international and national standards, it must be remembered that the noise frequency band, noise impulsiveness and noise kurtosis (i.e. statistical metric measure of the “peakedness” of

the probability distribution of the noise parameters) are also very relevant, as is individual susceptibility. Substantial noise exposure is the sine qua non of diagnosis.

Exposure to organic solvents (e.g. toluene, xylene and styrene) may have a synergistic effect. It may not be possible or necessary to differentiate between the noise and the chemical contributions to hearing loss, as the employer’s scope of liability extends to include both in regard to occupational hearing loss.

In noise-induced hearing loss sufferers, the audiogram typically shows a hearing shift at high frequencies, with a notch at 4–6 kHz.

Clinical history-taking and examination by an ENT expert serves to eliminate other possible diagnoses and complications.

The list of causes of asymmetrical hearing loss is long, and includes cerebello-pontine angle tumours, head injury, viral or vascular deafness, Ménière’s disease and perilymphatic fistula. In the presence of such conditions, ENT experts are often hesitant to declare that asymmetrical hearing loss is noise-induced, hence the Australian guidelines statement. Although it behoves the ENT expert to aid the patient by arriving at a diagnosis and alleviating symptoms, in a legal context the ENT expert is not obligated to the client alone but also to the court. In the latter situation, the ENT expert has an obligation to assist the court by ‘reason of specialized training, study or experience’,¹⁰ and is thus permitted to offer opinions to the court as to the meaning and implications of other evidence, unlike lay witnesses.

The ENT expert must realise that, in the legal arena, he or she is governed by the ‘preponderance of the evidence’ standard. In terms of statistical probability, this can be expressed as a probability of greater than 0.5 that the facts support (in this case) noise as the causative agent of hearing loss. This differs from the accepted medical proof standard, which requires a probability of close to 1. The rules of civil legal procedure do not require that the ENT expert ‘prove’ their case in any real sense. This is expressed by Cohen as follows:

Logical proof is not required. Neither is proof by the exclusion of all other possibilities, nor by the demonstration that the probability equals or closely approaches 1.0 (as in criminal proof).¹¹

Rather, the ENT expert is required only to demonstrate that noise is more likely than not to be the causative agent. In other words, all that is required is a probability of 0.5 or greater.

It is important to appreciate that the significance given to probability values differs vastly in law and medicine. Basically, a probability of up to 0.49 denotes inadequate proof in the legal arena, as regards the ‘preponderance of evidence’ standard; however, in science an error range is applied within which judgement is reserved pending further information. This latter option is not available in the legal sphere.

Both legal and medical proof could more accurately be described in terms of ‘fact finding’ or,

more appropriately, ‘fact inferring’.¹¹ As explained by Cohen:

...both disciplines do not traverse a logical route from premise to conclusion but, rather, traverse a route from individual data points of information — fact evidence — to inferences about the ultimate facts at issue. Thus, in neither empirical science nor in litigation are facts “proven” in any rigorous sense of the word. Rather, facts relevant to the proposition sought to be proved or disproved are considered and, if those facts lead the decision maker to conclude that the probability is sufficiently high (*and each discipline has its own standards*), the matter is pronounced proven (*or demonstrated*). In other words, while those who report the results of medical or legal fact-finding typically speak of “proof,” neither of those disciplines really engage in proof in the more formal (*mathematical*) sense of the term.¹¹ *Italics provided by SVF*

It is ultimately within this ‘proof process’ atmosphere that medical experts must function.

Although both medical and legal ‘proof’ share both superficially similar vocabulary and the basic goal of drawing inference from incomplete data, they approach these endeavours in fundamentally different ways. Associate Justice Harry Blackmun appeared to allude to this in the case of *Daubert versus Merrell Dow Pharmaceuticals*, when he wrote:

[T]here are important differences between the quest for truth in the courtroom and the quest for truth in the laboratory. Scientific conclusions are subject to perpetual revision. Law, on the other hand, must resolve disputes finally and quickly.¹²

In common law negligence claims,¹³ the ‘egg-shell skull rule’¹⁴ maintains that the defendant must take the plaintiff as that party finds the other, with all his or her weaknesses, beliefs, capacities and attributes.¹⁵ (Note that the *Civil Liability Act 2002 (NSW)*, by virtue of section 3B(1)(f),¹³ does not apply to claims made by a worker against his or her employer.) The plaintiff is entitled to recover for the whole loss. The defendant is liable despite any pre-existing susceptibility on the plaintiff’s part which causes the plaintiff’s damage to be far more severe than would otherwise be the case,¹⁶ and also despite any effect of the injury which renders the plaintiff more susceptible to further injury.¹⁷

Obviously, in cases of alleged noise-induced hearing loss the damage is of the same kind as would be reasonably foreseeable.

Equipped with these legal concepts, it is pertinent to examine the available medical evidence supporting noise causation of asymmetrical hearing loss, from a legal perspective.

Sabini and Sclafani¹⁸ undertook a study of asymmetrical hearing loss, involving history-taking, physical examination and extensive investigation; however, no definitive diagnosis was obtained for 19 per cent of 53 patients. Based on their results, these authors recommended only MRI, fluorescent treponemal antibody and erythrocyte sedimentation rate tests to

investigate patients with asymmetrical SNHL. This study made no mention of consideration of noise exposure during patient diagnosis. It is notable that, despite undertaking the recommended investigations, the diagnosis remained unknown in 19 per cent of cases. It is likely that these cases included some patients with noise-induced hearing loss.

Hendrix *et al.*¹⁹ studied 225 patients with asymmetrical SNHL, and were unable to reach a definitive diagnosis in 56 per cent.

A recent study by the UK Medical Research Council involved 48 313 randomly selected volunteers, and provides an indication of the incidence of unexplained asymmetrical hearing loss in the general population.²⁰ The authors state, ‘[t]his is a whole population survey, so the prevalence estimates reflect the general population rather than clinic samples that are inevitably influenced by referral patterns’, and herein lies the merit of this study for medicolegal purposes. This study concluded that only approximately 1 per cent of adults without a history of noise exposure had a defined asymmetrical hearing loss. Furthermore, these individuals had no other potential cause of asymmetrical hearing loss. Therefore, in this study the probability of asymmetrical hearing loss being attributable to noise, in the absence of noise exposure, the attributability to noise exposure is just zero. This recent study provides valuable information on the incidence of asymmetrical hearing loss in a population without a history of noise exposure; the incidence appears to be significantly low.

However, in studies of noise-exposed individuals, the incidence of asymmetrical hearing loss varies from 4.7 to 35 per cent. Our own study found an incidence of 22.6 per cent.

In Barrs and colleagues’ study,²¹ 20 per cent of 246 noise-exposed workers had asymmetrical hearing loss (of 15 dB at one frequency); MRI investigations showed no central lesion.

Segal *et al.*²² evaluated 429 patients with mild to moderate SNHL, and found a 35 per cent incidence of asymmetrical hearing loss together with negative retrocochlear investigation results. They concluded that a correlation existed between noise exposure and asymmetrical hearing loss more frequently affecting the left ear.

Chung *et al.*²³ found a 4.7 per cent incidence of asymmetrical hearing loss in 1461 patients with noise-induced hearing loss; the right ear was less affected in 82.6 per cent cases.

Alberti *et al.*²⁴ found a 15 per cent incidence of asymmetrical hearing loss in 1873 patients referred for compensation assessment. An obvious, treatable diagnosis, based on clinical history and investigation, was established for 54 per cent of patients with asymmetrical hearing loss. The authors concluded that 36 per cent of asymmetrical hearing loss cases were attributable to noise exposure, due to a definitive pattern of hearing loss and a history of noise exposure. These authors proposed the following possible mechanisms: shielding of one ear from noise; unequal recovery after severe noise exposure; and unequal sensitivity of the ears. In 10 per cent of cases, the cause of hearing loss remained unknown.

Some or all of these unknown cases may still have been attributable to noise exposure. From a legal perspective, it is highly improbable that these cases were due to a cause other than noise exposure.

The Stenger effect lends further credibility to our discussion on noise causation. The Stenger test is based on the premise that two tones of equal frequency and quality cannot be perceived simultaneously if one is louder; the subject hears only the louder tone. Conversely, it may be stated that if an environmental sound is perceived subjectively in one ear, the level of sound reaching that ear must be objectively more intense (i.e. of greater dB level). In individuals with normal (symmetrical) hearing, two sounds of equal frequency spectrum and loudness can be perceived simultaneously. However, if such an individual perceives more sound on one side, then the sound level reaching that ear will be more intense. This difference may be related to interaural time difference, or to sound level differences due to 'head shadow'. Hence, when an individual perceives noise as louder on one side than the other, a noise-related causation for asymmetrical hearing loss is more probable than not.

The American College of Occupational and Environmental Medicine position paper on noise-induced hearing loss states:

While noise-induced hearing loss is typically bilateral, asymmetric sources of noise such as sirens or gunshots can produce asymmetric loss. When evaluating cases of asymmetric loss, referral to rule out a retro-cochlear lesion is first warranted before attributing the loss to noise.²⁵

While the latter statement is appropriate for medical assessment, we will argue that in the legal arena such investigation is not necessary.

The Consensus conference paper⁶ on noise-induced hearing loss concedes that 'hearing loss is usually bilateral but some degree of asymmetry is not unusual, especially with lateralised noise sources such as rifles'.

Two types of noise exposure injury are recognised:⁶ acoustic trauma (i.e. exposure to a short duration of sound of sufficient intensity to cause injury) and noise-induced hearing loss (i.e. moderate periods of exposure to repeated, lower intensity sound).

Definitions of noise exposure injury occurring in an industrial context (also known as industrial, occupational and noise-induced hearing loss) must include the following two components.

Firstly, progressive noise-induced hearing loss (often referred to simply as noise-induced hearing loss, a misleading term as hearing loss due to sudden loud exposure also constitutes noise-induced hearing loss), i.e. a slow, symmetrical, sensorineural hearing loss. It is generally agreed that this type of hearing loss occurs with exposures of more than 85 dB(A) over 8 hours per workday, over a period of 10 years or more. It should be noted that a 3 dB increase is equivalent to a doubling of sound intensity. This is incorporated in some standards and regulations.²⁶

Secondly, definitions of industrial noise exposure injury should also include sudden noise-induced

hearing loss (often called acoustic trauma), i.e. immediate SNHL, which may be unilateral depending on the proximity of the source to each ear. Abrupt or explosive sounds can cause damage even with a single exposure. Sudden noise-induced hearing loss involves exposure levels greater than 140 dB. It appears that incidental exposure to such noises may not affect all individuals (e.g. in the case of fireworks, and possibly the type B impulse noise encountered in industry as described by Boettcher *et al.*).²⁷ However, regular such exposure will affect more individuals (e.g. in the case of firearms, termed type A impulse noise by Boettcher *et al.*).²⁷

Due to the protection afforded by the stapedial reflex, it is believed that a combination of simultaneous steady state noise and impulse noise may reduce the exposure risk, compared with the additive risk of either type of noise encountered separately. However, it appears that these two components, when encountered separately, have a cumulative effect which may result in asymmetrical SNHL.

It is the acoustic energy of the sound reaching the ear, not its source, that is important.⁶ In this respect, 'noise studies' of the work environment which aim to refute or document noise exposure in individual cases are of debatable value.

It must be remembered that not all individuals respond to sound in the same manner. There is a remarkably broad range of individual differences in sensitivity to any given noise exposure, and some individuals may be less tolerant of the same sound. Individual differences in ear anatomy and physiology may be responsible. It is also theoretically possible that not all individuals recover to the same extent from a similar exposure. Similarly, the two ears may vary in their response and recovery. Empirical studies documenting such differences are difficult to undertake. To date, there is no practical approach available to predict such differences in susceptibility.

Science may await such findings, but the law cannot wait when dispensing judgement on individual cases. In the real world, such information unfortunately seems unlikely to become available in the near future, as otological research has more pressing priorities, ethical considerations are problematic, and funding is scarce.

The incidence of occult acoustic neuroma (discovered at post-mortem examination) is quoted as 1 per cent.²⁸⁻³⁰ This figure is relatively high compared with the incidence of clinically encountered neuromas.³¹ In the absence of further information (either mitigating or adverse), the probability of non-noise-induced hearing loss remains at about 1 per cent in any individual case. Based on the high level of improbability of a non-noise-related cause in cases of asymmetrical hearing loss, it is possible in the legal arena, even in the absence of an investigative MRI, to state that an asymmetrical hearing loss is noise-induced (i.e. more likely to be so than not), provided the relevant history and clinical findings are available. However, it is prudent to inform the patient of any asymmetrical element to their hearing loss, and of the need for further medical assessment; from a medical perspective, MRI is

mandatory in such cases. Nevertheless, in a legal context the ENT expert can justifiably comment on the causation of hearing loss, as being (more probably than not) noise-induced.

- **Asymmetrical hearing loss due to industrial noise exposure is not uncommon**
- **In Australia, the relevant guidelines suggest that ‘the worse ear be equated with the better ear’ in compensation cases**
- **The prejudiciality of such an approach is discussed in a medicolegal context**

It should be remembered that the liability of any particular employer for the patient’s loss is within the jurisdiction of the court.

Conclusion

The central question is this: ‘Is the asymmetrical hearing loss due to some asymmetrical, unrecognised noise exposure, or to some asymmetrical individual pathology?’²⁰ The incidence of asymmetrical hearing loss due to non-noise-related pathology is approximately 1 per cent. In the absence of other significant clinical history or evidence of otological disease, the ENT expert should conclude that asymmetrical hearing loss is caused by noise exposure, and thus should be included in compensation considerations.

In the object ‘to co-construct the ideas of truth and the ideas of justice in the context of legal proceedings’,³² it is suggested that the current Australian Society of Otolaryngology – Head and Neck Surgery guidelines for evaluation of occupational noise induced hearing loss of gradual process³³ unjustly favour the employer at the cost of the employee in cases of asymmetrical hearing loss, and that these guidelines should thus be subject to revision.

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