

Presbycusis: A Lazy Diagnosis

Presbycusis, or age-related hearing loss, is conventionally defined as a gradual, bilateral sensorineural hearing loss (SNHL) associated with advancing age, initially affecting higher frequencies and subsequently causing difficulty understanding speech, particularly in noisy environments. The pathophysiology of ageing is complex and multifactorial: oxidative stress contributes to progressive loss of cochlear hair cells, atrophy of the stria vascularis, and degeneration of auditory nerve fibres and synapses, as well as central auditory pathway and processing deficits.¹ There is no disputing that the auditory system changes with age, just like any other part of the body. What this article aims to highlight is whether clinicians have the right to make this diagnosis when faced with older patients with SNHL without first taking a thorough history, particularly the often-neglected social history, which should include a detailed occupational history. I would argue that when the diagnosis is made without sufficient enquiry, it becomes something of a lazy diagnosis: age per se is an association, not an aetiology. In my view, presbycusis should scarcely enter the diagnostic vocabulary when considering differentials in patients younger than 60 years. SNHL before then demands an aetiological search rather than the convenient refuge of ageing.

Worsening hearing is more prevalent with advancing age, but this does not mean that hearing loss is inevitable, nor that age alone satisfactorily explains it. Auditory function varies substantially between people of similar chronological age. Some retain good hearing well into advanced age, while others develop significant SNHL much earlier. Concern regarding misuse of the term presbycusis is not new. More than half a century ago, Lowell and Paparella urged clinicians to make every effort to establish the correct diagnosis before attributing SNHL solely to ageing, noting that some patients diagnosed with presbycusis probably had familial or genetic hearing loss.² When faced with a 60-

year-old patient with gradual bilateral SNHL, we should ask not how old the patient's ears are, but what has happened to them over the years. The cochlea of a 60-year-old is not merely a cochlea that has existed for 60 years. It is a cochlea that has experienced 60 years of life. For many Malaysian workers, a substantial part of that life may have been spent in noisy environments.

Noise-induced hearing loss (NIHL) is a well-established consequence of exposure to hazardously loud sound. Workers exposed to chronic loud noise are at risk of permanent SNHL; and because that risk is predictably measurable, this occupational disease is preventable. Under Malaysia's Occupational Safety and Health (Noise Exposure) Regulations 2019, a daily noise exposure level exceeding 82 dB(A) constitutes excessive noise and triggers statutory requirements for assessment and management of occupational noise exposure. A 40-year-old factory worker with bilateral high-frequency SNHL and years of hazardously loud occupational exposure may readily be diagnosed with occupational NIHL following appropriate assessment. The same person with further deterioration in hearing thresholds 20 years later may acquire a new and remarkably convenient diagnosis: presbycusis. This is where indiscriminate use of the term becomes particularly troubling.

Ageing and occupational NIHL are not mutually exclusive processes. Age-related auditory changes superimposed on established noise damage neither render the earlier exposure resolved, nor erase its contribution to the eventual hearing deficit. Turning 60 does not make the preceding 20 years of occupational exposure disappear. Separating the effects of ageing and noise in an individual is undoubtedly difficult. Their effects may be additive, interactive and consequently audiometrically inseparable.³ The typical 4,000 Hz notch associated with NIHL does not obligingly remain

preserved. Continued exposure, progression at neighbouring frequencies and superimposed ageing may flatten or obscure the dip, even alter the audiometric pattern altogether.

By the time a patient presents later in life with a smooth, downward-sloping bilateral SNHL, the audiogram may no longer reveal the history of cochlear injury: it tells us the current pattern and degree of hearing loss, but not its cause. A bilateral sloping high-frequency SNHL may be compatible with presbycusis. It may also be compatible with cumulative occupational noise exposure, ototoxic injury, genetic susceptibility, or combinations of these. Distinguishing NIHL from age-related hearing loss at an individual level is inherently difficult, but that uncertainty should not make exposure history less important. It should make it more important. For older patients, the clinician should establish not merely the present job but a lifetime history of noise exposure, use of personal hearing protection, previous audiograms, documented threshold shifts, co-exposure to ototoxic chemicals, firearm training, and the relation between exposure and hearing deterioration. Age must not become a substitute for taking that exhaustive and yes, tedious history. Nor should the possibility of exposures outside work become an easy means of discounting occupational causation. Hazardous noise, admittedly, no longer belongs only to the workplace. The traditional image of NIHL requires updating, as the acoustic environment of everyday life has changed dramatically. Personal listening devices, earbuds, headphones and gaming devices now accompany almost every waking activity. Many fall asleep listening to music, podcasts, white noise or autonomous sensory meridian response (ASMR) sounds through these devices. Consequently, more than one billion young people may be at risk from unsafe recreational listening.⁴ This should broaden our clinical history, not dilute recognition of occupational NIHL. It tells us that NIHL can occur outside employment; it does not make documented occupational exposure irrelevant. Duration, intensity, intermittency, impulse characteristics, hearing protection, cumulative dose and concurrent exposures

all matter. Recognition of non-occupational exposure should help us understand the totality of cochlear insult. We should be cautious not to shift responsibility for preventable workplace exposure from employer to employee.

This brings us back to presbycusis and the possibility that SNHL reflects noise exposure instead. The term *sociocusis* has been used to describe hearing deterioration associated with the noise of everyday industrialised living rather than biological ageing alone. More recently, Fink challenged the conventional concept more directly. In a 2025 commentary, he argued that SNHL frequently labelled presbycusis or age-related hearing loss may substantially represent cumulative sociocusis and NIHL.⁵ This underscores the uncomfortable but pertinent question: how much SNHL have we attributed to ageing simply because ageing is easier to document than a lifetime exposure history? A diagnosis of exclusion should require some excluding. Presbycusis should never be the first explanation merely because the date of birth seems to fit. Before making the diagnosis, we should consider occupational and non-occupational noise exposure; ototoxic medications and chemical exposure; diabetes and cardiovascular risk; smoking; previous infections and otological disease; head injury; family history; and the rate, symmetry and pattern of progression. Significant asymmetry, unilateral tinnitus, sudden or rapidly progressive loss, fluctuation, vestibular symptoms, neurological findings or disproportionately poor speech discrimination should prompt investigation for alternative pathology. Contemporary clinical guidance similarly recommends further evaluation for asymmetric loss, conductive or mixed loss, or poor word recognition rather than simply treating these findings as manifestations of ageing.

One might argue that none of these matters as the management remains the same. The loss is permanent and irreversible, and the patient will require hearing rehabilitation, hearing aids being foremost regardless of the underlying cause. But calling hearing loss presbycusis suggests inevitability and risks

transforming a potentially preventable disorder into an unavoidable consequence of ageing. It discourages investigation, obscures occupational contribution, weakens prevention and inadvertently normalises disability. There is also something faintly ageist about it. We do not usually explain an older person's dyspnoea simply by saying that lungs age; we investigate cardiovascular and respiratory disease. We do not automatically attribute neurological deterioration to ageing without considering causes. Yet bilateral SNHL in older adults can acquire the presbycusis label with remarkably little resistance. Common is not synonymous with normal, and normal is certainly not synonymous with inevitable. We may never be able to apportion a patient's SNHL precisely between occupational noise, ageing, genetics and other causes. Human biology does not divide itself so obligingly and this uncertainty should not be the justification for giving age all the credit.

So the next time a 60-year-old presents with bilateral SNHL, we should resist the temptation to diagnose the audiogram before diagnosing the patient. Age is crucial, yes, but ask more carefully about the years spent working in hazardous noise. Ask too about ototoxic chemical exposures, medications, illnesses and family history. Only after considering what a lifetime has done to the ears should we decide how much of that damage can be attributed to ageing itself. Presbycusis may well be the correct diagnosis. It should never be the lazy one.

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