

“High Frequency Presbycusis”–Is There an Earlier Onset?

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Abstract The objective of this study is to determine whether presbycusis occurs much earlier than previously believed if the high frequency (above 8 kHz) are included. Tertiary referral center (a teaching University). This is a cross-sectional observational study. Healthy adults from 20 to 49 years of age who had essentially normal hearing were included into the study. They were subjected to high frequency pure tone audiometry (until 16 kHz). Participants were grouped based on age ranges of 10 years (e.g., 20–29, 30–39, and 40–49) and the presence of symmetrical high frequency sensor neural hearing loss were documented. There is a significant presence of symmetrical high frequency sensor neural hearing loss (not attributed to any known risk factors) as early as from the age group of 40 to 49 years. Seven of 43 participants (16%) from age group of 20 to 29 years and 12 of 24 participants (50%) from age group of 30 to 39 years had significant high frequency hearing loss. High frequency hearing loss (high frequency Presbycusis) may occur much earlier than previously believed.

Keywords Presbycusis · Earlier onset

Introduction

Presbycusis is the decline in hearing associated with various types of auditory system dysfunction that accompany aging, and cannot be accounted for, by traumatic, genetic, or pathological conditions [1]. It usually presents after the 5th decade of life, more than 75% are above 55 years [2]. It can be classified into four pathological types which are sensory (degeneration of

outer hair cells), neural (degeneration of neurons and spiral ganglia), metabolic/strial (atrophy of stria vascularis), and mechanical/cochlear conductive (thickening and secondary stiffening of the basilar membrane of the cochlea) [3]. There is an association between advanced age and high-tone deafness as described by Zwaardemaker in 1899. Men are affected more at high frequencies [4]. Systematic decline in the number of outer hair cells starts at birth and continues throughout the lifetime of an individual [5]. Recent studies suggest the role of genetics in this age related hearing loss [6, 13, 14].

Pure tone audiometry (PTA) usually measures hearing thresholds from 125 to 8,000 Hz. However, human hearing ranges from 20 to 20,000 Hz [7]. Hence it is possible that hearing loss in the range of 8,000–20,000 Hz is not being picked up during routine audiometry. The availability of a high frequency PTA machine (MadsonOrbiter 922) producing pure tones of up to 16,000 Hz, enables us to detect high frequencies at a much earlier age.

Materials and Methods

This is a cross-sectional observational study. Healthy adults between 20 and 49 years of age (inclusive of both) were interviewed, excluded using exclusion criteria, and subjected to high frequency pure tone as well as routine audiometry. This was performed in a sound proof room by an experienced audiologist. A Madson Orbiter 922 audiometer was used. This audiometer produces two graphs. One shows the audiogram till 8 kHz with the international symbols and one showing the higher frequencies from 6 to 16 kHz. There were no symbols used for this high-tone graphs. Our search of literature did not reveal symbols used in these high frequency audiographs. However, we have inserted the international symbols manually. Patients were grouped based on

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age ranges of 10 years (e.g., 20–29, 30–39, 40–49) and the presence of symmetrical high frequency sensorineural hearing loss was noted. We used a standard definition of normal hearing range (intensity) between 0 and 20 dB. Thus any participant with hearing loss of more than 20 dB was considered to have a hearing loss. Participants with >15 dB difference in hearing at one frequency or 10 dB hearing difference at two frequencies were defined, as an asymmetrical hearing loss [8].

Examples of high frequency Pure Tone Audiometry results:

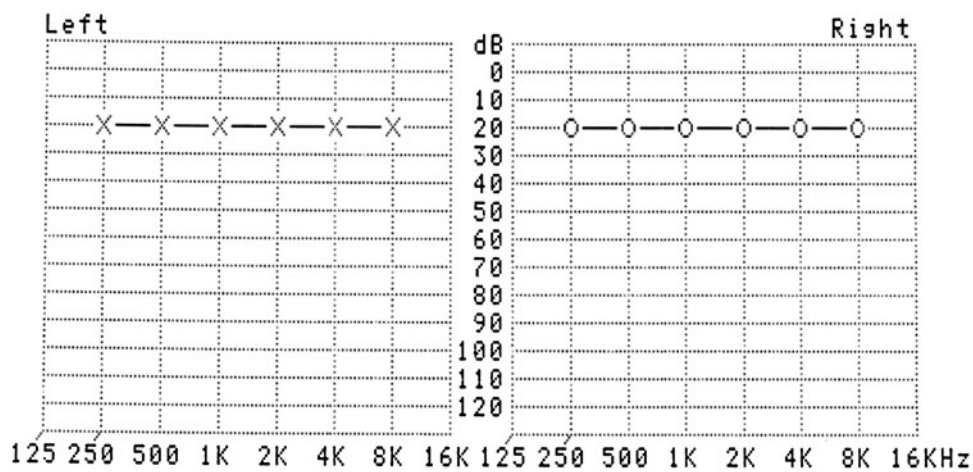
Normal Showing audiographs from 8 to 16 kHz only as the routine audiogram was normal.

Exclusion criteria were as follows

Noise exposure, head/ear trauma, childhood ear disease, vertigo, familial hearing loss, systemic diseases, ototoxic drugs, and radiotherapy to the head and neck region.

Results

A total of 80 healthy adults participated in this study. Any asymmetrical hearing loss was excluded. Out of 80 participants, 43 were in the age group 20–29, 24 in age group 30–39, and 13 in age group 40–49 (Table 1). Thirty-nine were males and 41 were females (Table 2). Racial



Symmetrical high frequency sensorineural hearing loss
Showing audiographs from 8 to 16 kHz only as the routine audiogram was normal.

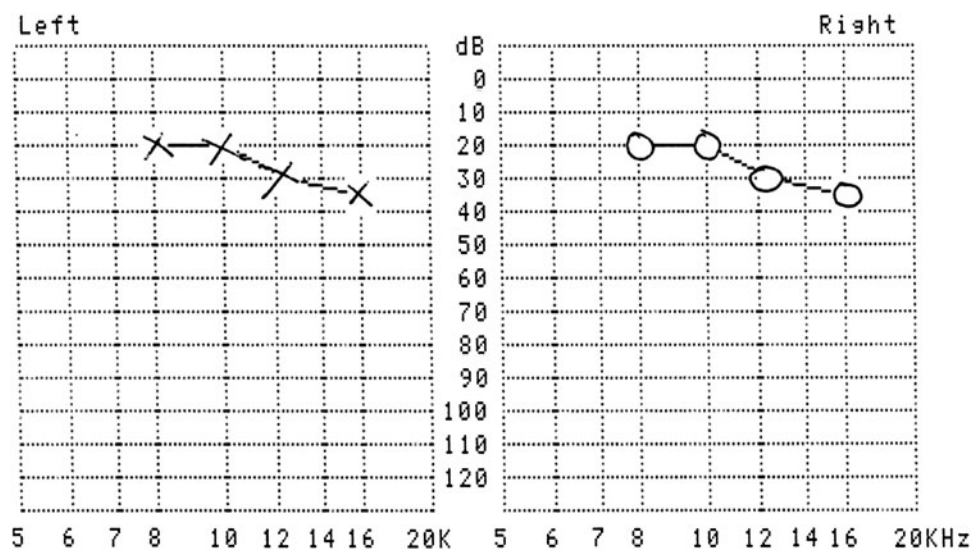


Table 1 Age group of participants

Age group (years)	Participants
20–29	43
30–39	24
40–49	13
Total	80

Table 2 Sex, Age ratio of participants

Age group (years)	Male	Female
20–29	18	25
30–39	16	8
40–49	5	8
Total	39	41

Table 3 Racial distribution of participants

Age group (years)	Malay	Chinese	Indian	Others
20–29	19	13	10	1
30–39	5	5	9	5
40–49	6	3	4	0
Total	30	21	23	6

Table 4 No of participants with high frequency hearing loss in the age groups

Age group (years)	Hearing loss at high frequencies (%)
20–29	7/32 (16)
30–39	12/24 (50)
40–49	13/13 (100)

composition included 30 Malays (37%), 21 Chinese (26%), 23 Indians (29%) and 6 from other ethnic background (one Iban, one Sudanese and four Iranians) (Table 3).

There were 32 participants (40% of total participants) who had high frequency sensorineural hearing loss. Out of the 32 participants, 7 were from the 20 to 29 age group (16% of that age group), 12 from age group 30 to 39 (50% of that age group), and 13 from age group 40 to 49 (100% of that age group) (Table 4). Statistical analysis was performed in each age group between the participants with hearing loss as compared to those without hearing loss.

We also observed that as the age group progresses from the younger to the older groups, the onset of hearing loss begins much earlier i.e., at a lower frequency. For example, most of the participants (with hearing loss) from the age group 20 to 29 had hearing loss from 12 kHz onwards

whereas the participants from age group 40 to 49 started to have hearing loss at a much lower frequency (8 kHz onwards).

Chi-Square Test

The data was analyzed using the Chi-Square test and $P < 0.05$ was taken as significant difference.

Age group 20–29

Hearing loss

	Observed <i>N</i>	Expected <i>N</i>	Residual
Yes	7	21.5	–14.5
No	36	21.5	14.5
Total	43		

	Hearing loss
Chi-Square ^a	19.558
df	1
Asymp. Sig.	0.000

^a 0 cells (0.0%) have expected frequencies less than five. The minimum expected cell frequency is 21.5

$P < 0.0001$ (indicating a significant difference for no hearing loss group compared to hearing loss group). This means that the age group 20–29 does not have significant presbycusis.

Age group 30–39

Hearing loss

	Observed <i>N</i>	Expected <i>N</i>	Residual
Yes	12	12.0	0.0
No	12	12.0	0.0
Total	24		

	Hearing loss
Chi-Square ^a	0.000
df	1
Asymp. Sig.	1.000

^a 0 cells (0.0%) have expected frequencies less than five. The minimum expected cell frequency is 12.0

$P = 1.0$ (no significant difference between the two groups)

Age group 40–49

Hearing loss

	Observed <i>N</i>	Expected <i>N</i>	Residual
Yes	13	13.0	0.0
Total	13 ^a		

^a This variable is constant. Chi –Square test cannot be performed

Unable to perform test as all the patients had hearing loss. There is significant presbycusis in the age group 40–49.

Discussion

Presbycusis is the decline in hearing associated with various types of auditory system dysfunction that accompany aging, and cannot be accounted for by traumatic, genetic, or pathological conditions [1]. It usually presents after the 5th decade of life, more than 75% are above 55 years [2]. The international incidence of presbycusis varies widely among societies. Westernized foreign countries and primitive civilizations have very different patterns of hearing loss. A 1962 study by Rosen and colleagues of a remote tribe of the Sudan called the Mabaans revealed significantly less hearing loss in the elderly population than in similarly aged people of urban societies. Whether this is because of the lack of chronic noise exposure or the paucity of other systemic ailments that are common in industrialized societies (e.g., atherosclerosis, diabetes, reactive airway disease) is not known. In general, most of the world's population experiences some degree of decline in hearing with advancing age.

Characteristically, presbycusis involves bilateral high frequency hearing loss associated with difficulty in speech discrimination and central auditory processing of information. However, other patterns of presbycusis exist. The association between advanced age and high-tone deafness was first described by Zwaardemaker in 1899. Since then, extensive research has been made to determine the pathological changes of presbycusis. However, the exact mechanisms still remain unknown.

Histological changes associated with aging occur throughout the auditory system from the hair cells of the cochlea to the auditory cortex in the temporal lobe of the brain. These changes may correlate with different clinical findings and auditory test results, depending on the severity of the changes and the anatomic level at which they occur. Gacek and Schuknecht [3] identified four sites of aging in the cochlea and divided presbycusis into four types based on these sites. The histological changes are correlated approximately with symptoms and auditory test results.

- Sensory presbycusis: Epithelial atrophy with loss of sensory hair cells and supporting cells in the organ of Corti. This process originates in the basal turn of the cochlea and slowly progresses toward the apex. These changes correlate with a precipitous drop in the high frequency thresholds, which begins after middle age. The abrupt downward slope of the audiogram begins above the speech frequencies; therefore, speech discrimination is often preserved. Histologically, the atrophy may be limited to only the first few millimeters of the basal end of the cochlea. The process is slowly progressive over time. One theory proposes that these changes are due to the accumulation of lipofuscin pigment granules.
- Neural presbycusis: Atrophy of nerve cells in the cochlea and central neural pathways. Schuknecht estimated that 2,100 neurons are lost every decade (of 35,000). This loss begins early in life and may be genetically predetermined. Effects are not noticeable until old age because pure tone average is not affected until 90% of neurons are gone. Atrophy occurs throughout the cochlea, with the basilar region only slightly more predisposed than the remainder of the cochlea. Therefore, no precipitous drop in the high frequency thresholds on audiogram is observed. A disproportionately severe decrease in speech discrimination is a clinical correlate of neural presbycusis and may be observed before hearing loss is noted because fewer neurons are required to maintain speech thresholds than speech discrimination.
- Metabolic (or strial) presbycusis: Atrophy of the stria vascularis, which normally maintains the chemical and bioelectric balance and metabolic health of the cochlea. Atrophy of the stria vascularis results in hearing loss represented by a flat hearing curve because the entire cochlea is affected. Speech discrimination is preserved. This process tends to occur in people aged 30–60 years. It progresses slowly and may be familiar.
- Mechanical (or cochlear conductive) presbycusis: This condition results from thickening and secondary stiffening of the basilar membrane of the cochlea. The thickening is more severe in the basal turn of the cochlea where the basilar membrane is narrow. This correlates with a gradually sloping high frequency sensorineural hearing loss that is slowly progressive. Speech discrimination is average for the given pure tone average.

The changes associated with presbycusis are rarely found exclusively at one site, but typically involves simultaneous changes at multiple sites. This explains the difficulty of associating specific clinical symptoms or signs with specific anatomic locations [9–11]. Much of the current

research focuses on finding underlying genetic abnormalities that may cause, contribute to, or predispose to the development of this disease. One of the most widely investigated potential causes is a genetic mutation in mitochondrial DNA. Reduced perfusion of the cochlea associated with age may contribute to the formation of reactive oxygen metabolites, which may adversely affect the inner ear neural structures as well as cause damage to mitochondrial DNA. Damaged mitochondrial DNA may cause reduced oxidative phosphorylation, which may lead to neural dysfunction in the inner ear. A study by Dai et al. [12] also suggested that damaged mitochondrial DNA may lead to anatomic changes of the inner ear, more severe narrowing of the vaso nervorum in the internal auditory meatus in temporal bones with a mitochondrial DNA deletion. Damaged mitochondrial DNA has also been linked to a greater rate of apoptosis of certain cells in the inner ear in a study by Pickles [13].

Two specific mitochondrial DNA deletions, mtDNA4834 and mtDNA4977, have been linked to age related hearing loss in rodents. Han et al. and Dai et al. [14, 15] have linked the mtDNA4977 deletion with archived human temporal bones from patients with presbycusis.

Although the precise etiology of presbycusis is currently not known, the cause of presbycusis is generally agreed to be multifactorial.

As the routine PTA only tests hearing loss up to 8 kHz, it is generally assumed that the onset of age related hearing loss or presbycusis is generally after the age of 50 or so. With the advent and more extensive usage of high frequency audiograms, it appears that age related hearing loss begins much earlier especially at the higher frequencies although they are only clinically detected once the speech frequencies are involved. Occasionally patients may perceive the lack of appreciation of high frequency sounds at a young age in areas of music. Hence high frequency presbycusis occurring at a much earlier age should be considered.

Conclusions

This study gives us some evidence that age related hearing loss appears much earlier if the high frequency (above 8 kHz) thresholds are taken into consideration. This

can appear as early the 2nd decade (16%) and 3rd decade (50%) It is present in all patients by the 4th decade even if they do not have any clinical hearing loss as shown by a normal conventional audiogram. Thus this is in keeping with the belief that there is a systematic decline in the number of outer hair cells (of the organ of Corti) from birth and progresses as age advances [5].

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