Article published online: 2023-05-26

Evaluation of Differential Sensitivity for Frequency, Intensity, and Duration in Individuals with Hypertension

Saranya Mahendran[1] Prashanth Prabhu[1]

[1]All India Institute of Speech and Hearing, Mysore, Karnataka, India

Address for correspondence Prashanth Prabhu, PhD, Department of Audiology, All India Institute of Speech and Hearing, Mysore, Karnataka, 570006, India (e-mail: prashanth.audio@gmail.com).

Ann Otol Neurotol ISO 2022;5:78–85.

Abstract

Objective Hypertension is a condition in which the blood vessels have persistently raised pressure. The damage of the cochlea is due to the loss of sensitive hair cells in the inner ear or the damage to the eighth cranial nerve. When the cochlea is damaged, the functioning abilities such as coding, differentiation, and temporal processing abilities will be affected. Hence, there might be deficits in differential sensitivity in individuals with hypertension. The aim of this article was to study the effect of hypertension on differential sensitivity as there is limited literature in this area.

Method Thirty participants were included in the study and classified into two groups: group I as individuals with hypertension and group II as individuals with normal blood pressure in the age range of 25 to 45 years. Psychophysical tests like frequency, intensity, and time discrimination tests were performed using the maximum likelihood procedure (MLP) toolbox, which implements a maximum likelihood procedure for threshold estimation in MATLAB.

Results In all the three test conditions, the scores were significantly poorer in individuals with hypertension compared with individuals of the normal at all the frequencies such as 500 Hz, 1,000 Hz, 2,000 Hz, and 4,000 Hz.

Conclusion This could be because of reduced frequency selectivity and poor temporal coding as well as due to difficulty responding to rapid change in the envelope of sound over time because of cochlear and neural damage in individuals with hypertension.

Keywords
- hypertension
- differential sensitivity
- blood supply
- temporal coding

Introduction

Hypertension (HTN), commonly referred to as increased blood pressure, is a condition in which the blood vessels have persistently raised pressure. Blood is supplied to all parts of the body through the vessels from the heart. Blood pressure is produced by the force of blood pushing against the walls of blood vessels (arteries) as it is pumped by the heart. If the blood pressure is high, it is made harder for the heart.[1] HTN is popularly called the “silent killer” because it has no obvious signs or symptoms as a warning, and many people are not even aware of their problem. Eventually, the increased pressure overload due to HTN causes accumulating damage that the circulatory system can handle, leading to profound health conditions.[2]

HTN is a major risk factor for cardiovascular disorders, especially in India.[3] The most important cause of premature death globally is HTN that is rated by the World Health Organization (WHO).[1] South Asia's diseases of burden high blood pressure rank HTN as the third most vulnerable disease.[4] HTN is directly responsible for 57% of all stroke deaths and 24% of all coronary heart disease (CHD) deaths

DOI https://doi.org/10.1055/s-0043-1768495
ISSN 2581-9607

Thieme Medical and Scientific Publishers Pvt. Ltd. A-12, 2nd Floor, Sector 2, Noida-201301 UP, India

in India.[2] The global burden of HTN worldwide analysis revealed 20.6% of men and 20.9% of women in India were suffering from HTN. The rates for HTN in 2025 may go up to 22.9 and 23.6% for men and women in India.[5] Studies on the Indian population have shown a prevalence of HTN to be 25% in urban and 10% in rural areas.[6] However, noncommunicable diseases country profiles[7] revealed an increase in the prevalence of HTN among Indians at a rate of 33.2% in men and 31.7% in women. The relation between damaged hearing and high blood pressure is not complicated to comprehend. High blood pressure results in damaged blood vessels not centered only to one area of the body; somewhat, the entire body is affected, including the ears. The hearing could be affected due to the deposition of fatty plaque in the blood vessels of the ear.[2]

The damage of the cochlea is due to the loss of sensory hair cells in the inner ear or the damage to the eighth cranial nerve. This occurs naturally or as a result of aging, degenerative disease, injury, etc. There are few research works that report cochlear damage leading to hearing loss in individuals with HTN. Agarwal et al[8] reported a significant relationship between HTN and hearing loss by estimating the hearing threshold on HTN group and compared it with non-HTN group. de Moraes Marchiori et al[9] conducted a study on 308 individuals in the age range of 45 to 64 years, out of which 154 were individuals with HTN and 154 were controls. They revealed a significant relationship between HTN and hearing loss by using a questionnaire on hearing loss and correlated it with blood pressure reading and a poorer pure tone threshold. Soares et al[10] examined the audiological profile of adults with and without HTN using transient-evoked otoacoustic emissions (TEOAEs) and reported a lower TEOAE amplitude in individual with HTN.

Goyel et al[11] studied the impact of severity of hypertension on auditory brainstem responses in 50 healthy age and sex-matched controls (group I) and 50 hypertensive patients (group II). The latter group was further subdivided into group IIa (grade 1 HTN), group IIb (grade 2 HTN), and group IIc (grade 3 hypertension), as per WHO guidelines. They reported that the absolute latency of wave I was observed to be significantly increased in group IIa and IIb hypertensives, while wave V absolute latency was significantly prolonged among Group IIb and IIc, as compared with that of the normal control group.

Thus, all the above studies suggest cochlear or neural damage in individuals with HTN. The cause for hearing impairment in HTN individuals is explained as the reduction in oxygen transport, which increases the blood viscosity that reduces blood flow in the capillaries. This causes a reduction in oxygen, that is, hypoxia, thus causing hearing loss in the individual with HTN.[2] High pressure in the vascular system causes the inner ear to get damaged. This can lead to progressive hearing loss.

It is well known that normal cochlear/neural functioning is important for differential sensitivity or limen, which plays a vital role in speech perception. Since there is cochlear or neural damage reported in individuals with HTN, it is important to study the differential sensitivity in them. Pitch, loudness, and duration play an important role in the perception of speech, language, and music. The talker's emotion will be identified based on the pitch, loudness, and duration in the case of speech. Traditionally, psychophysical methods, which are done in humans, depend mainly on the auditory system. Pure tones are used as principal stimuli to judge the difference between the two sounds.[12] The auditory processing is a fundamental component of most auditory tasks, which is seen at different stages, starting from the onset of a stimulus process by neurons and response to speech, a higher-level function controlled by the cortex. Hence, when the cochlea is damaged, functioning abilities such as coding, differentiation, and temporal processing will be affected. Previous studies have also reported sensory and neural damage in individuals with HTN.[2] Therefore, there might be deficits in differential sensitivity in individuals with HTN. There is a need to study the effect of HTN on differential sensitivity as there is limited literature in this area.

Methods

The study was performed using a standard group comparison design. The participants were selected using a purposive sampling technique. Thirty participants in the age range of 25 to 45 years were included in the study and classified into two groups. Group I included individuals with HTN, and Group II included individuals with normal blood pressure. All the audiological tests were performed in an acoustically treated suite that met the specification of ANSI S3.1–1999 (R2013). Additionally, the test room had optimum temperature and lighting and was free of distraction. Approval was taken from the ethical approval committee of the institute, and the testing was done using noninvasive procedures. Informed consent was taken from all the participants regarding the procedures and the objectives of the study. The equipment used in the study is the audiometer, immittance meter, otoscope, and laptop, in which MATLAB software was installed.

Procedure for Participant Selection

The detailed case history was taken as a closed set interview to rule out the auditory and vestibular disorder symptoms, the sensation of tinnitus, exposure to hearing harmful agents, such as occupational or leisure noise, drugs that are ototoxic general health conditions. The otoscopic examination was performed. It is done to visually inspect the status of the external ear canal and tympanic membrane. This procedure preceded the audiological evaluation. Normal hearing was ensured using pure tone audiometry, and all the participants had hearing thresholds within 25dBHL at octave frequencies between 250 and 8,000 Hz. They had a type "A" tympanogram with the presence of acoustic reflexes indicating normal middle ear functioning.

Assessment of Difference Limen Abilities

All the tests were performed using the MLP toolbox, which implements a maximum likelihood procedure for threshold estimation in MATLAB.[13] In all of the psychophysical tests, stimuli were presented monoaurally at an intensity of 60 dB

HL. Further, the psychometric function that gives the maximum likelihood was used to decide the stimulus to be presented in the upcoming trial. The MLP usually meets the quite stable approximation of the most probable psychometric function, which can be used to approximate thresholds[13–15]

Frequency discrimination: In this, the minimum frequency difference necessary to discriminate between two closely spaced frequencies was assessed. Frequency difference limen was measured at 500 Hz, 1,000 Hz, 2,000 Hz, and 4,000 Hz. The standard and variable stimuli were 250 milliseconds long pure tones with the onset and offsets of 10 milliseconds raised cosine ramp.[13,16] On each trial of three blocks, two blocks had a standard frequency of a pure tone and another block of variable frequency of a pure tone, which is always higher than the standard frequency. The variable block was identified by the participant. The psychometric function of 79.4% was achieved by using the three down, one up rule to estimate the difference limen for frequency.

Intensity discrimination: In this, the minimum intensity difference necessary to discriminate between two otherwise same sounds was assessed. Intensity difference limen was measured at 500 Hz, 1,000 Hz, 2,000 Hz, and 4,000 Hz. Both the standard and variable stimuli were of 250 milliseconds long, pure tones with 10 milliseconds rise and fall time.[13,16] The minimum and maximum intensity deviations used were at 0.99 and 10 dB. On each trial of three blocks, two blocks had a pure tone at a standard intensity. Another block selected at random contained a pure tone of variable intensity, which is always higher than the standard intensity. The participant identified the variable block. The psychometric function of 79.4% was achieved by using the three down, one up rule to estimate the difference limen for intensity.

Duration discrimination: In this, the minimum time difference necessary to discriminate between two otherwise same sounds was assessed. Time difference limen was measured at 500 Hz, 1,000 Hz, 2,000 Hz, and 4,000 Hz. Both the standard and variable stimuli were of 250 milliseconds long, pure tones with 10 milliseconds rise and fall time.[13,16] On each trial of three blocks, two blocks had a pure tone at a standard duration. Another block selected at random contained a pure tone of variable duration, which is always different from the standard duration. The participant's task was to identify the variable block which will be of 0 to 25 milliseconds difference in duration between the two stimuli. The psychometric function of 79.4% was achieved by using the three down, one up rule to estimate the difference limen for time.

Analyses

Statistical Package for the Social Sciences (SPSS) was used to analyze the data. Shapiro–Wilk test of normality was performed to determine whether the data were normally distributed or not. The test scores of the difference limen abilities for individuals with HTN and with normal blood pressure were statistically analyzed.

Compliance with Ethical Standards

In this study, all of the tests were conducted on humans using noninvasive techniques, in accordance with guidelines provided by the Ethics Approval Committee at the institute. All the procedures were explained to the participants, and they were asked for their informed consent prior to participation in the study. The authors declare no conflicts of interest.

Results

In the study, two groups of participants were included. Group I included individuals with hypertension, and group II included individuals with normal blood pressure. In each group, 15 participants (30 ears) were considered for the study. For both the groups, differential limen for frequency, intensity, and time were assessed using the "MLP" toolbox, which implements a maximum likelihood procedure in MATLAB.

Comparison of Frequency Discrimination in Individuals with Hypertension and with Normal Blood Pressure

The mean, median, and standard deviation (SD) of frequency discrimination across frequencies such as 500Hz, 1,000 Hz, 2,000 Hz, and 4,000 Hz in individuals with and with normal blood pressure are shown in ►**Table 1**. The data showed the difference between both the groups, which indicated poorer mean scores for the HTN group compared with with normal blood pressure group.

Shapiro–Wilk test of normality was administered to check whether the data are normally distributed or not and was found to be not normally distributed ($p < 0.05$). Hence,

Table 1 Mean, median, and standard deviation (SD) scores of frequency discrimination across frequencies such as 500Hz, 1,000 Hz, 2,000 Hz, and 4,000 Hz in individuals with hypertension and with normal blood pressure

		Mean (Hz)	SD	Median (Hz)
500 Hz	Normal blood pressure	59.0	23.5	65.0
	Hypertension	98.0	18.2	95.9
1,000 Hz	Normal blood pressure	73.5	23.5	77.5
	Hypertension	99.4	27.9	99.4
2,000 Hz	Normal blood pressure	75.3	14.9	78.0
	Hypertension	102.3	27.6	99.0
4,000 Hz	Normal blood pressure	75.4	31.5	78.0
	Hypertension	117.7	30.5	110.5

nonparametric inferential statistics were done. Mann–Whitney U test was administered for between-group comparison, and the effect size was also calculated. The results of the Mann–Whitney U test are shown in ►**Table 2** for frequency discrimination. The data indicates the scores are significantly poorer for the individuals with HTN when compared with the individuals with normal blood pressure, as shown in ►**Fig. 1**. The effect size of this difference was determined using the formula r = Z/ formula_57968_1. The effect size is 0.95, 0.73, 0.84, and 0.98 for 500, 1,000, 2,000, and 4,000 Hz, indicating that the effect size is strong across all the frequencies.

Comparison of Intensity Discrimination in Individuals with Hypertension and with Normal Blood Pressure

The mean, median, and SD of intensity discrimination across frequencies 500 Hz, 1,000 Hz, 2,000 Hz, and 4,000 Hz in individuals with HTN and with normal blood pressure are shown in ►**Table 3**. The data showed the difference between both the groups, which indicated poorer scores in the HTN group compared with with normal blood pressure group. Shapiro–Wilk test of normality was administered to check whether the data are normally distributed or not and was found to be not normally distributed ($p < 0.05$). Hence, nonparametric inferential statistics were done. Mann–Whitney U test was administered for between-group comparison, and the effect size was also calculated. The results of the Mann–Whitney U test are shown in ►**Table 4**. The results showed that the scores were significantly poorer for individuals with HTN compared with the control group, as shown in ►**Fig. 2**. The effect size of this difference was determined using the formula r = Z/ formula_57968_2 . The effect size is 0.71, 0.82, and 0.71 for 500, 1,000, and 4,000 Hz, respectively, and it indicates that the effect size is strong across all these frequencies and the effect size is 0.42 for 2,000 Hz, which indicates the effect size is weak.

Comparison of Duration Discrimination in Individuals with Hypertension and with Normal Blood Pressure

The mean, median, and standard deviation (SD) of intensity discrimination across frequencies such as 500Hz, 1,000 Hz,

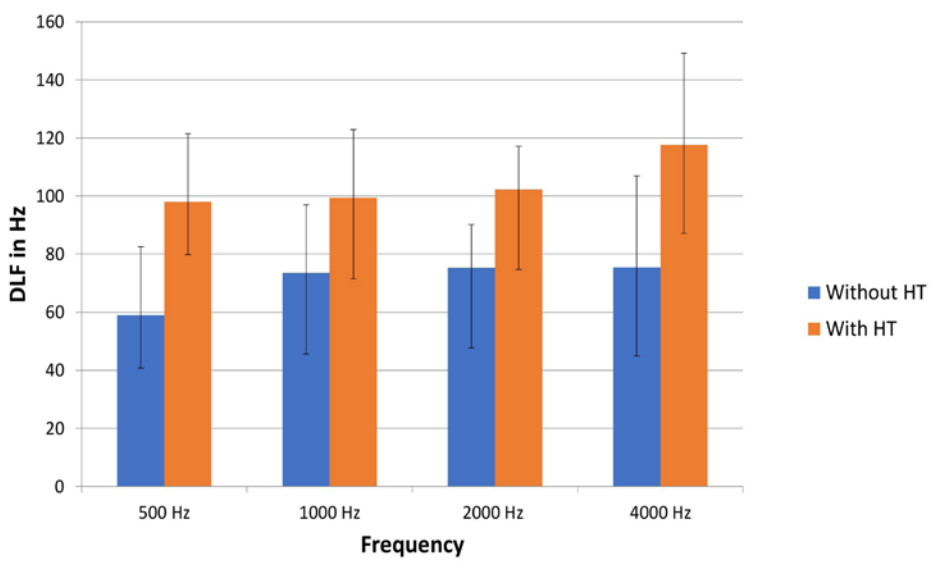

Fig. 1 Mean and standard deviation of frequency discrimination for 500 Hz, 1,000 Hz, 2,000 Hz, and 4,000 Hz frequencies in individuals with hypertension (HTN) and with normal blood pressure. DLF, difference limen for frequency; DLI, difference limen for intensity; DLT, difference limen for time.

Table 2 Results of Mann–Whitney U test and effect size for frequency discrimination test

	U	Z	*p*-Value	Effect size
500 Hz	98.0	5.2	< 0.01	0.95
1,000 Hz	176.0	4.0	< 0.01	0.73
2,000 Hz	133.0	4.6	< 0.01	0.84
4,000 Hz	81.5	5.4	< 0.01	0.98

2,000 Hz, and 4,000 Hz in individuals with and with normal blood pressure are shown in ►**Table 5**. The data showed the difference between both the groups, which indicated poorer mean scores for the HTN group compared with with normal blood pressure group.

Shapiro–Wilk test of normality was administered to check whether the data are normally distributed or not and was found to be not normally distributed ($p < 0.05$). Hence, non-parametric inferential statistics were done. Mann–Whitney U test was administered for between-group comparison, and the effect size was also calculated. The results of the Mann–Whitney U test are shown in ►**Table 6**. The results showed that the scores were significantly lower in individuals with HTN, as shown in ►**Fig. 3**. The effect size of this difference was determined using the formula r = Z / formula_57968_3 . The effect size is 0.80, 0.82, 0.85, and 0.87 for 500, 1,000, 2,000, and 4,000 Hz, respectively, and it indicates that the effect size is strong across all the frequencies.

In all the three test conditions, such as frequency, intensity, and duration discrimination, the scores were significantly poorer in individuals with HTN compared with individuals of the normal at all the frequencies such as 500 Hz, 1,000 Hz, 2,000 Hz, and 4,000 Hz.

Discussion

Our human body depends on a proper blood supply for the normal functioning and survival of the living cells in the body. The most common vascular disease is HTN that may create various changes such as in the heart and vessels. This may cause hemorrhage to the inner ear, which is supplied by the anterior and inferior cerebellar artery.[17–19] Sudden and progressive hearing loss is also caused due to HTN.[20] HTN can damage the ear in numerous ways. One of the possible mechanisms could be related to the increase in blood viscosity, which reduces capillary blood flow and ends up reducing oxygen transport, causing tissue hypoxia, thus causing hearing complaints and hearing loss in patients.[21] In addition to this, arterial HTN may also cause ionic changes in the cochlea's cell potentials, which could lead to hearing impairment.[22]

Table 3 Mean, median, and standard deviation (SD) scores of intensity discrimination across frequencies 500Hz, 1,000 Hz, 2,000 Hz, and 4,000 Hz in individuals with hypertension and with normal blood pressure

		Mean (dB)	SD	Median (dB)
500 Hz	Normal blood pressure	3.3	1.4	2.9
	Hypertension	6.4	2.9	6.9
1,000 Hz	Normal blood pressure	3.1	1.5	2.
	Hypertension	6.9	2.6	7.8
2,000 Hz	Normal blood pressure	2.8	1.5	2.7
	Hypertension	5.4	3.1	6.4
4,000 Hz	Normal blood pressure	3.0	1.4	2.9
	Hypertension	6.6	2.9	7.5

Table 4 Results of Mann–Whitney U test and effect size for intensity discrimination test

	U	Z	*p*-Value	Effect size
500 Hz	182.0	3.9	< 0.01	0.71
1,000 Hz	145.0	4.5	< 0.01	0.82
2,000 Hz	286.5	2.4	< 0.05	0.42
4,000 Hz	185.5	3.9	< 0.01	0.71

Table 5 Mean, median, and standard deviation (SD) scores of duration discrimination across frequencies 500 Hz, 1,000 Hz, 2,000 Hz, and 4,000 Hz in individuals with and with normal blood pressure

		Mean (ms)	SD	Median (ms)
500 Hz	Normal blood pressure	43.5	12.8	40.2
	Hypertension	75.4	29.4	70.0
1,000 Hz	Normal blood pressure	60.6	36.6	50.4
	Hypertension	110.9	34.9	127.0
2,000 Hz	Normal blood pressure	63.0	23.6	59.4
	Hypertension	114.2	36.4	128.0
4,000 Hz	Normal blood pressure	53.2	20.3	52.1
	Hypertension	116.1	38.5	133.5

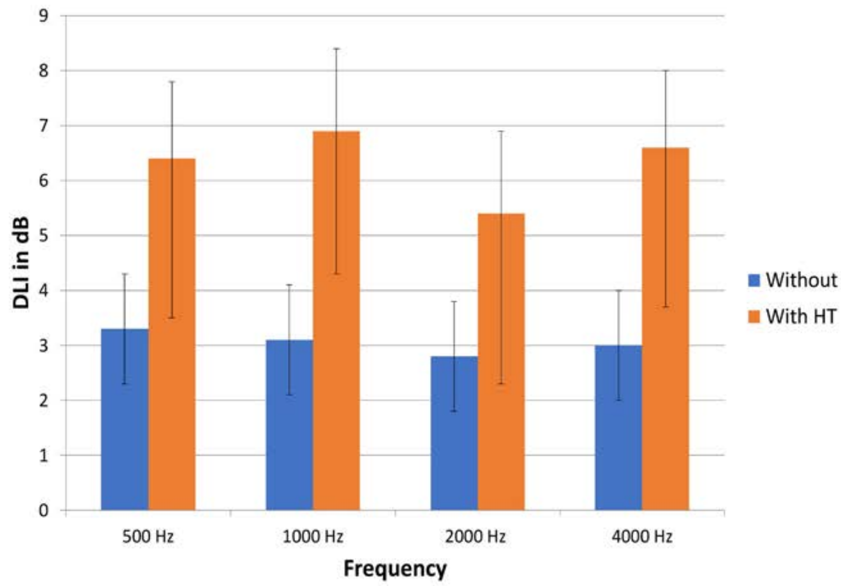

Fig. 2 Mean and standard deviation of intensity discrimination for 500 Hz, 1,000 Hz, 2,000 Hz, and 4,000 Hz frequencies in individuals with hypertension (HTN) and normal blood group.

Khullar et al[23] reported significant prolongation of absolute peak latencies of waves I, II, and V and interpeak latency III to V in individuals with HTN. They conclude that there was a significant correlation of a rise in systolic and diastolic blood pressure with absolute peak latencies of ABRs in hypertensive patients. However, no significant difference in nerve conduction velocity was seen. They suggested that neural damage was seen in individuals with HTN. Goyal et al[11] studied to correlate and to assess the degree of involvement of peripheral and central regions of brainstem auditory pathways with increasing severity of HTN among the patients of essential HTN. The results revealed that all the hypertensives, that is, Group IIa, IIb, and IIc patients, were found to have highly significantly prolonged III to V inter peak latency compared with normal healthy controls. Hence, there has been greater involvement of pontomesenchymal region with the increasing severity of the disease.

Thus, all the above studies suggest cochlear or neural damage in individuals with HTN. The reason for hearing impairment in individuals with HTN is explained as the increase in blood viscosity that reduces blood flow in the capillaries, thereby reducing oxygen transport. This causes tissue hypoxia (reduced oxygenation), thus causing hearing loss in patients.[2] Inner ear damage can occur due to high pressure in the vascular system. This can cause progressive hearing loss. Arterial HTN may cause ionic changes that can lead to hearing loss. HTN causes degeneration of the inner ear due to alteration in microcirculation, thus accelerating the aging process in the inner ears.[2]

It is well known that normal cochlear/neural functioning is important for differential sensitivity or limen, which plays a vital role in the perception of speech. Since cochlear or neural damage is reported in individuals with hypertension, this could have led to poor performance in differential sensitivity in the present study. Pitch, loudness, and duration play an important role in the perception of speech, language, and music. Traditionally, psychophysical methods, which are done in humans, depend mainly on the auditory system. The auditory processing is a fundamental component of most auditory tasks, which is seen at different stages, starting from the onset of a stimulus process by neurons and response to the speech, a higher-level function controlled by the cortex.

Table 6 Results of Mann–Whitney U test and effect size for duration discrimination test

	U	Z	*p*-Value	Effect size
500 Hz	147.5	4.474	< 0.01	0.80
1,000 Hz	141.5	4.564	< 0.01	0.82
2,000 Hz	128.5	4.761	< 0.01	0.85
4,000 Hz	120.0	4.881	< 0.01	0.87

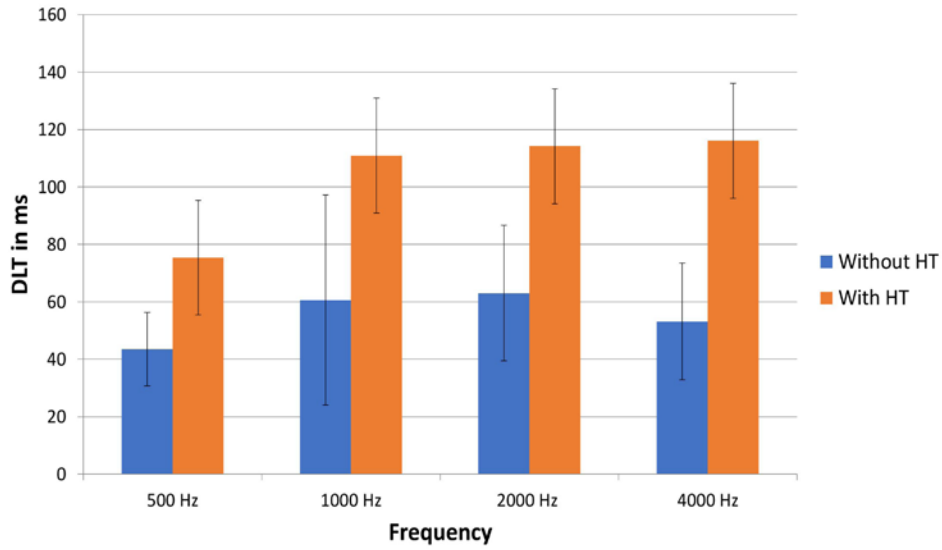

Fig. 3 Mean and standard deviation of duration discrimination for 500 Hz, 1,000 Hz, 2,000 Hz, and 4,000 Hz frequencies in individuals with hypertension (HTN) and normal blood group.

Hence, when the cochlea is damaged, functioning abilities such as coding, differentiation, and temporal processing will be affected. Previous studies have also reported sensory and neural damage in individuals with HTN.[2] In this study, the results revealed significantly poorer scores in differential limen for frequency, intensity, and duration in individuals with hypertension. This might be because of reduced frequency selectivity, poor temporal coding, and difficulty responding to rapid change in the envelope of sound over time because of cochlear and neural damage in individuals with HTN. However, the effect of HTN on the functioning of the central auditory nervous system should be further studied.

Conclusion

The results revealed a significant difference between the two groups in all three tests. HTN causes degeneration of the inner ear due to alteration in microcirculation, thus accelerating the aging process in the inner ears. It is well known that normal cochlear/neural functioning is important for auditory processing, like sensitivity limen abilities. The differential limen is an essential component of most auditory processing capacity, which can be seen at several levels, ranging from neuronal sensitivity to the effects of stimulus onset time to cortical processing of auditory information such as speech stimuli. When the cochlea is damaged, functioning abilities such as coding, differentiation, and temporal processing will be affected. Previous studies have also reported sensory and neural damage in individuals with HTN. These could be the possible reasons for deficits in differential sensitivity among individuals with HTN.

Authors' Contributions

Saranya Mahendran was involved in study design, data collection, analysis of the results, interpreting, and writing of the manuscript; Prashanth Prabhu was involved in study design, data collection, analysis of the data, interpretation and writing of the manuscript.

Conflict of Interest

None declared.

Acknowledgments

The authors acknowledge with gratitude Prof. M. Pushpavathi, Director, All India Institute of Speech and Hearing, Mysore, affiliated to the University of Mysore for permitting to conduct the study at the institute. The authors also like to acknowledge the participant for co-operation.

References

1 Mackay J, Mensah GA. The Atlas of Heart Disease and Stroke. Geneva: World Health Organization; 2004
2 Gupta R. Trends in hypertension epidemiology in India. J Hum Hypertens 2004;18(2):73–78 Gupta R. Trends in hypertension epidemiology in India. J Hum Hypertens 2004;18(2):73–78
3 Srinath Reddy K, Shah B, Varghese C, Ramadoss A. Responding to the threat of chronic diseases in India. Lancet 2005;366(9498):1744–1749 Srinath Reddy K, Shah B, Varghese C, Ramadoss A. Responding to the threat of chronic diseases in India. Lancet 2005;366(9498):1744–1749

4 Lim SS, Vos T, Flaxman AD, et al. A comparative risk assessment of burden of disease and injury attributable to 67 risk factors and risk factor clusters in 21 regions, 1990-2010: a systematic analysis for the Global Burden of Disease Study 2010. Lancet 2012;380(9859):2224–2260
5 Kearney PM, Whelton M, Reynolds K, Muntner P, Whelton PK, He J. Global burden of hypertension: analysis of worldwide data. Lancet 2005;365(9455):217–223
6 Das SK, Sanyal K, Basu A. Study of urban community survey in India: growing trend of high prevalence of hypertension in a developing country. Int J Med Sci 2005;2(2):70–78
7 World Health Organization (WHO). Noncommunicable Diseases Country Profiles, 2018. Geneva: WHO; 2018
8 Agarwal S, Mishra A, Jagade M, Kasbekar V, Nagle SK. Effects of hypertension on hearing. Indian J Otolaryngol Head Neck Surg 2013;65(Suppl 3):614–618
9 de Moraes Marchiori LL, de Almeida Rego Filho E, Matsuo T. Hypertension as a factor associated with hearing loss. Rev Bras Otorrinolaringol (Engl Ed) 2006;72(4):533–540
10 Soares MA, Sanches SGG, Matas CG, Samelli AG. The audiological profile of adults with and without hypertension. Clinics (São Paulo) 2016;71(4):187–192
11 Goyal G,Mittal A,Chaudhary C,Bachhel R,Grewal S,Rai M. The impact of severity of hypertension on auditory brainstem responses. Int J Med Public Health 2014; 4 (3):218–221
12 Shofner WP. Representation of the spectral dominance region of pitch in the steady-state temporal discharge patterns of cochlear nucleus units. J Acoust Soc Am 2008;124(5):3038–3052
13 Grassi M, Soranzo A. MLP: a MATLAB toolbox for rapid and reliable auditory threshold estimation. Behav Res Methods 2009;41(1):20–28
14 Green DM. Stimulus selection in adaptive psychophysical procedures. J Acoust Soc Am 1990;87(6):2662–2674
15 Green DM. A maximum-likelihood method for estimating thresholds in a yes-no task. J Acoust Soc Am 1993;93(4 Pt 1):2096–2105
16 Jain C, Mohamed H, Kumar AU. Short-term musical training and pyschoacoustical abilities. Audiology Res 2014;4(1):102 10.4081/audiores.2014.102
17 Giles TD, Berk BC, Black HR, et al. Expanding the definition and classification of hypertension. J Clin Hypertens (Greenwich) 2005;7(9):505–512
18 Falkner B, Lurbe E, Schaefer F. High blood pressure in children: clinical and health policy implications. J Clin Hypertens (Greenwich) 2010;12(4):261–276
19 Bachor E, Selig YK, Jahnke K, Rettinger G, Karmody CS. Vascular variations of the inner ear. Acta Otolaryngol 2001;121(1):35–41
20 Nagahara K, Fisch U, Yagi N. Perilymph oxygenation in sudden and progressive sensorineural hearing loss. Acta Otolaryngol 1983;96(1-2):57–68
21 Ohinata Y, Makimoto K, Kawakami M, Haginomori S, Araki M, Takahashi H. Blood viscosity and plasma viscosity in patients with sudden deafness. Acta Otolaryngol 1994;114(6):601–607
22 Rarey KE, Ma YL, Gerhardt KJ, Fregly MJ, Garg LC, Rybak LP. Correlative evidence of hypertension and altered cochlear microhomeostasis: electrophysiological changes in the spontaneously hypertensive rat. Hear Res 1996;102(1-2):63–69
23 Khullar S, Gupta N, Babbar R. Auditory brainstem responses & nerve conduction velocity in essential hypertension. Vasc Dis Prev 2009;6(1):51–55 https://benthamopen.com/ABSTRACT/VDP-6-51