ORIGINAL ARTICLE
AUDIOLOGY

DOI: 10.5152/B-ENT.2022.21629

Hearing Aid Use on Auditory Temporal Processing in Conductive Hearing Loss

Merve Torun Topçu[1,2], Ayça Çiprut[1], Esra Akdeniz[3]

[1]Department of Audiology, Marmara University School of Medicine, İstanbul, Turkey
[2]Department of Audiology, İstanbul Medeniyet University, School of Health Sciences, İstanbul, Turkey
[3]Department of Medical Education, Marmara University School of Medicine, İstanbul, Turkey

Cite this article as: Torun Topçu M, Çiprut A, Akdeniz E. Hearing aid use on auditory temporal processing in conductive hearing loss. B-ENT 24 May 2022 10.5152/B-ENT.2022.21816 [Epub Ahead of Print]

ABSTRACT

Objective: The chronicity of the problem of conductive pathologies, the lack of improvement with treatment, and the progression of hearing loss are seen as important criteria for using hearing aids. The aim of this study was to investigate the effect of conductive pathologies and the use of hearing aids on temporal processing.

Methods: In this study, 68 adults (aged 19-58 years [mean 38.26 years]) with mild to moderate bilateral conductive hearing loss were included in the study: 23 bilateral hearing aid users, 22 unilateral hearing aids users, and 23 patients with no use of amplification, and 31 healthy adults (aged 22-60 years [mean 34.13 years]) were included as a control group. After the complete audiological test battery, gaps in noise threshold, total percentage score, duration pattern, and frequency pattern tests were performed for temporal processing.

Results: The results of frequency pattern test, duration pattern test, and total percentage score were obtained from high to low, respectively, as normal hearing, bilateral hearing aid users, aided ear of unilateral hearing aid group, unaided ear of unilateral hearing aid group, and no hearing aid group. Ranked from highest to lowest, gaps in noise threshold threshold results were the reverse of total percentage score results, as expected. Finally, the results of patients with long-term conductive hearing loss who had never used hearing aids were significantly lower when compared with the hearing aid users group and the control group.

Conclusion: As a result of this study, although the cochlea and auditory nerve are intact in long-term conductive pathologies, the existing hearing loss may lead to auditory deprivation and affect temporal processing. Early intervention with appropriate amplification in conductive pathologies on the other hand may contribute to temporal processing.

Keywords: Gaps in noise, temporal auditory processing, conductive pathologies, hearing aid use

Introduction

Chronic conductive hearing loss (CHL) is characterized by a long-lasting and persistent decrease in hearing due to outer and/or middle ear disorders. Many researchers have noted that this prolonged sensory deprivation can produce irreversible changes in the anatomical and functional integrity of central auditory structures, such as changes in the relative size of neuron dendrites in subcortical nuclei or synaptic and spike adaptation disruptions in the auditory cortex.[1-6] Moore et al[7] (2003) stated that CHL may affect the correct processing of the acoustic signal's time structure (e.g. delaying low frequency sounds entering the inner ear for up to 150 μs).[7] Auditory temporal processing (ATP), which is shown as one of the supraliminal auditory processing mechanisms, refers to the ability of the hearing system to process the temporal properties of a sound stimulus in a certain period of time.[1,5,8,9] Studies have indicated that ATP may be an essential component of many auditory processing abilities, such as being a prerequisite for language and speech acquisition in the processing of speech transitions and sound information.[7,10] It is extremely important to recognize the location, duration of sound, and the difference between other stimuli and to collect and combine all this information in understanding speech. Speech understanding depends on the listener's ability to notice temporal changes in the stimulus.[11] Evaluating temporal processing requires a combination of temporal resolution and temporal sequence tests. There are studies in the literature that examine the relationship between gap detection performance and aging, maturation, sensorineural and CHL, and cochlear implant use,[12-15] but

Corresponding author: Merve Torun Topçu, e-mail: tugba.merve.torun@hotmail.com
Received: July 30, 2021 **Accepted:** March 3, 2022 **Available online**: May 24, 2022
Available online at www.b-ent.be

Table 1. Descriptive Statistics for Hearing Thresholds of the CHL Group

	(500-4000 Hz mean ± SD)					
Groups	Air ConductionThresholds		Bone Conduction Thresholds		WDS% (Mean ± SD)	
Ear	Right	Left	Right	Left	Right	Left
Bilateral HA (n=46)	55.97 (9.26)	58.20 (8.34)	21.75 (6.42)	18.80 (7.55)	94.09 (6.25)	94.78 (5.31)
Unilateral HA (n=44)	53.40 (11.22)	54.60 (9.42)	18.18 (8.84)	16.28 (10.61)	93.27 (7.13)	94.18 (6.01)
No HA (n=46)	56.30 (10.42)	59.25 (11.32)	17.28 (8.68)	19.72 (9.22)	83.83 (7.86)	84.52 (7.93)

CHL, conductive hearing loss group; SD, standard deviation; HA, hearing aid.

there is no comprehensive study showing the effect of hearing use on temporal processing, especially in long-term conductive pathologies. This study was created considering that long-term medical and surgical treatment processes and patients' loss of hearing during this period have a negative effect on temporal processing. Therefore, the purposes of the present study were to (1) investigate the effects of CHL on ATP ability, (2) evaluate the effect of hearing aid (HA) use on ATP in conductive pathologies, and (3) determine whether unilateral or bilateral HA use differs in ATP results in conductive pathologies. For this purpose, gaps in noise (GIN) test, duration pattern test (DPT), and frequency pattern test (FPT) were used to evaluate temporal processing ability in CHL patients.

Methods

The study was approved by Marmara University Medical School Clinical Studies Ethics Committee (March 2, 2018-09.2018.239), and informed consent was obtained from all participants

Participants

The study population included 2 different groups of conductive pathologies, namely 43 patients with chronic otitis media, 25 patients diagnosed with otosclerosis, and 31 normal-hearing healthy adults as a control. All subjects showed normal scores on the mini-mental state examination. In the selection of the patient group, attention was paid to ensure that the 4 frequency averages of the bone conduction thresholds should not be worse than 35 dB so that the cochlear function does not come into play. The inclusion criteria for the hearing loss group were as follows: (1) bilateral mixed (10 patients) or conductive (58 patients) hearing loss, (2) stable hearing level for the previous year, (3) individuals who did not use HA must have had hearing loss for a minimum of 5 years, (4) at least 2 years of regular use of HA in the group using HAs, (5) no active middle ear effusion, and (6) good speech discrimination score; better than 70%.

Main Points

- Hearing loss should not be ignored in conductive pathologies.
- The importance of evaluation with temporal processing tests in addition to standard pure tone audiometry and speech audiometry in revealing the central effect of conductive hearing loss.
- Determination and implementation of rehabilitation options in conductive pathologies without wasting time.

Conductive and Mixed Hearing Loss (CHL-MHL) group (n = 68; 136 ears): This group had bilateral symmetrical (difference between the pure tone averages [500, 1000, 2000, and 4000 Hz] of 2 ears is less than 15 dB) mild to moderate conductive and mixed type hearing loss. This group consisted of 33 males and 35 females (aged 19-58 years [mean 38.26 years]). Pure tone air conductive averages (500, 1000, 2000, and 4000 Hz) ranged from 26 dB HL to 65 dB HL and bone conductive averages (500, 1000, 2000, and 4000 Hz) ranged from 9 dB HL to 32 dB HL. The distribution of the audiometric results of the CHL group is shown in descriptive statistics for the hearing thresholds of the group in Table 1. This group was divided into 3 groups: bilateral HAs users (n = 23, 46 ears), unilateral HA users (n = 22, 44 ears), and no HA users (n = 23, 46 ears). Participants using unilateral HAs were divided into 2 groups because a statistically significant difference was found between the scores of the ear with and without a HA ($P < .05$).

Control Group (n=31; 62 ears): This group was composed of healthy subjects (17 males, 14 females aged 22-60 years [mean 34.13 years]) without a history of otitis media and showed a threshold of 20 dB HL or less for octave frequencies between 250 Hz and 8000 Hz, bilaterally. None of the subjects had any major medical, neurological, or psychiatric histories. Transient evoked otoacoustic emissions were performed and must show 6 dB SPL SNR for at least 3 frequencies to be considered normal. Normal middle-ear function was assessed by acoustic immittance testing.

Procedures

Hearing tests were carried out in a sound-isolated booth using GSI 61 clinical audiometers (Grason-Stadler, Eden Prairie, Minn, USA) and TDH-39 supra-aural headphones (Telephonics, Farmingdale, NY, USA). Air conduction thresholds were measured from 250 Hz to 8 kHz (in half-octave steps).

Bone-conduction thresholds were determined from 500 Hz to 4000 Hz using a bone vibrator (Radioear B-71). When the bone conduction threshold differed by more than 10 dB at each frequency from the air conduction threshold in the same ear, masking was performed by introducing a narrowband noise to the untested ear. Speech reception threshold in quiet and word recognition tests were performed on the same audiometry equipment. Acoustic immitancemetry was performed with the GSI Tympstar tympanometer (Grason-Stadler) using a 226 Hz probe tone. Gaps in noise, duration pattern, and frequency pattern tests were used to evaluate auditory temporal processing. The entire test battery lasted for a total of approximately 120 minutes, with rest breaks when the patient needed it.

Gaps in Noise (GIN) Test: The test is a behavioral test used when it is necessary to detect gaps in a continuous auditory stimulus to assess auditory temporal processing. In the test, there are 4 different test lists containing 60 gaps with 2, 3, 4, 5, 6, 8, 10, 12, 15, and 20 ms durations, and each test is randomly sorted 6 times. The number of these gaps embedded in the 6 seconds white noise segments ranges from 0 to 3 and the interstimulus interval (segment) is 5 seconds. Test stimuli are applied to the patient at 30 dB SL level with a calibrated audiometer through a supra-aural headset through the compact disc.[16] A random list of tests was selected for each subject, and practice samples were given to all subjects to ensure understanding of the task before administering the test. The moment they heard a small gap in the noise, they were asked to press the answer button on their hands. Pressing the answer button during the gap was considered as positive response, not pressing the button when a gap occurred as incorrect response, randomly pressing the button when there was no gap was counted as a false-positive response. The result of the test yielded 2 scores: GIN threshold and total percentage score (TPS). Gaps in noise threshold are the shortest gap time with at least 4 out of 6 correct definitions, and the TPS was calculated as total number of gaps identified/total number of gaps in the list) $\times$ 100.

Duration Pattern Test (DPT): The test included 2 tones of different duration, short (S 250 ms) or long (L 500 ms), and stimuli were randomly presented to each patient at 30 dB SL level with supra-aural headphones.[17] The patients were asked to verbally state the stimulus sequences in three phrases such as "short, long, short." The frequency of the tones was 1 kHz with a 300-ms interval between successive tones in the sequence.

Frequency Pattern Test (FPT): The test is used to assess the temporal ordering and sequencing that are responsible for most auditory processing skills and speech perception.[18] For standard assessment, stimuli were presented to each patient at 30 dB SL. Subjects were asked to verbalize the perceived frequencies in the order of the given 3-tone series, such as "high, low, high." Ten exercises were given to all subjects to ensure that the task was understood. The test result was determined by calculating the percentage of correct answers. In the triple tone series in the test, 2 frequency tones, low (L 880 Hz) and high (H 1122 Hz), were used and 30 trials were conducted. The duration of each tone was 300 ms (10 ms rise–fall time), with a 300-ms interval between successive tones in the sequence.

All tests were performed in unilateral and bilateral HA users who participated in the study, and the right and left ears of these patients were tested separately with supra-aural headphones. During the test, the HAs were taken-off and the tests were performed at a hearing intensity that they could hear comfortably through the supra-aural headphones.

Statistical Analysis

Categorical variables were summarized using frequencies and percentages. Distributions of numerical variables (TPS, FPT, DPT, and GIN thresholds) were assessed using Kolmogorov–Smirnov test QQ and PP plots, boxplots, and skewness and kurtosis values. Homogeneity of variances was tested using Levene's test. For normally distributed variables, the Welch analysis of variance (ANOVA) test was employed when the homogeneity of variances assumption was not met, whereas, for non-normally distributed variables, Kruskal–Wallis test was employed to test differences between groups. For all-pairs comparisons in a one-factorial layout with normally distributed residuals but unequal groups variances, Tamhane's T2 test was performed. The Sidak-adjusted *P* values were given in the multiple comparisons tables. A *P*-value less than .05 is considered statistically significant. Jamovi, R version 4.1.2 (2021-11-01) and Statistical Package for the Social Sciences were used for statistical analysis.

An exact Wilcoxon-signed rank test was performed to distinguish whether there was a significant difference between the scores of the ear with and without a HA in the unilateral HA group.

Analysis of variance test was used to analyze whether there was a significant difference between the age values between the groups, and the Tamhane T2 post hoc test was performed, no significant difference was found between the groups ($P > .05$).

In order that the hearing loss of the subjects would not make a difference in the research design, the right-left ear air and bone conduction hearing thresholds of the bilateral, unilateral HA group, and the group that no HAs were analyzed with one-way ANOVA analysis and Tamhane test as post hoc test. As a result, no significant difference was found in the hearing thresholds of these groups ($P > .05$).

Results

Frequency Pattern Results

The mean FPT scores of the no HA group, bilateral HA group, normal hearing, aided and unaided ear of unilateral HA group were 69.65% (11.93), 86.87% (6.28), 88.61% (4.84), 82.73% (7.74), and 81.27% (8.01), respectively (Table 2). Ranked from highest to lowest, the results of FPT analysis were as follows: normal hearing, bilateral HA users, unilateral HA group aided ear, unilateral HA group unaided ear, and no HA group. The FPT results of the no HA group were significantly lower than the results of all other groups ($P < .001$; Table 3). No significant differences were identified between the FPT scores of the bilateral HA group and other groups except for no HA group ($P > .05$; Table 3). Further, no significant difference was found between aided and unaided ears of the unilateral HA group in terms of FPT scores ($P > .05$; Table 3).

Duration Pattern Test Results

The mean DPT scores of the no HA group , bilateral HA group, normal hearing, aided and unaided ear of unilateral HA group were 70.59% (11.07), 86.67% (5.27), 87.58% (4.14), 84.36% (6.61), and 82.14% (7.30), respectively (Table 2). Ranked from highest to lowest, the results of DPT analysis were as follows: normal hearing, bilateral HA users, unilateral HA group aided ear, unilateral HA group unaided ear, and no HA group. The DPT results of the no HA group were significantly lower than the results of all other groups ($P < .001$; Table 4). The DPT scores of the bilateral HA group showed no statistically significant

Table 2. Descriptive Statistics for Each Group

Groups	Results	Mean (SD)	Minimum–Maximum		n (ear)
No hearing aid	TPS, %	77.76 (6.68)	65.00	90.00	46
	GIN threshold	4.74 (1.18)	3.00	6.00	
	DPT score, %	70.59 (11.07)	46.00	86.00	
	FPT score, %	69.65 (11.93)	45.00	88.00	
	Age	40.00 (8.67)	25.00	55.00	
Bilateral hearing aid	TPS, %	89.13 (4.65)	76.00	96.00	46
	GINthreshold	2.61 (.71)	2.00	5.00	
	DPT score, %	86.67 (5.27)	76.00	96.00	
	FPT score, %	86.87 (6.28)	75.00	98.00	
	Age	40.13 (10.65)	19.00	58.00	
Normal hearing	TPS, %	91.52 (4.18)	84.00	100.00	62
	GINthreshold	2.65 (.66)	2.00	4.00	
	DPT score, %	87.58 (4.14)	75.00	95.00	
	FPT score, %	88.61 (4.84)	78.00	97.00	
	Age	34.13 (10.24)	22.00	60.00	
With HA ear of unilateral HA group	TPS, %	86.50 (5.38)	71.00	93.00	22
	GINthreshold	2.95 (1.00)	2.00	5.00	
	DPT score, %	84.36 (6.61)	64.00	94.00	
	FPT score, %	82.73 (7.74)	62.00	94.00	
	Age	38.23 (11.09)	20.00	55.00	
Without HA ear of unilateral HA group	TPS, %	84.27 (5.64)	68.00	92.00	22
	GINthreshold	3.05 (1.09)	2.00	5.00	
	DPT score, %	82.14 (7.30)	60.00	93.00	
	FPT score, %	81.27 (8.01)	62.00	95.00	
	Age	38.23 (11.09)	20.00	55.00	

SD, standard deviation; TPS, total percentage score; DPT, duration pattern test; GIN, gaps in noise; FPT, frequency pattern test.

differences from those of the normal hearing group ($P > .05$) and aided ear of the unilateral HA group. No statistically significant differences were found between aided and unaided ears of unilateral HA group in terms of DPT scores ($P > .05$; Table 4).

Gaps in Noise Test Results

The mean TPS of the no HA group, bilateral HA group, normal hearing group, aided and unaided ear of unilateral HA group were 77.76% (6.68), 89.13% (4.65), 91.52% (4.18), 86.50% (5.38), and 84.27% (5.64), respectively (Table 2). The TPS of the no HA group was significantly lower than that of all other groups ($P < .001$; Table 5). There were no significant differences in TPS between the aided and unaided ears of the unilateral HA group ($P > .05$; Table 5). However, significant differences were revealed among the bilateral HA group, no HA group, and unaided ear of the unilateral HA group ($P < .05$; Table 5).

Table 3. Pairwise comparisons of FPT between groups

Groups	No HA	Bilateral HA	Normal Hearing	Unilateral Aided	Unilateral Unaided
No HA	-	<0.001*	<0.001*	<0.001*	<0.001*
Bilateral HA	<0.001 *	-	0.724	0.303	0.066
Normal hearing	<0.001 *	0.724	-	0.024*	0.004*
Unilateral aided	<0.001 *	0.303	0.024*	-	1.000
Unilateral unaided	<0.001 *	0.066	0.004*	1.000	-

The cells in the table represent Tamhane multiple comparisons *P* values, *The mean difference is significant at the .05 level. (2-tailed). No HA, no hearing aid group; Bilateral HA, bilateral hearing aid group; Unilateral aided, with hearing aid ear of unilateral HA group; Unilateral unaided, without hearing aid ear of unilateral HA group. HA, hearing aid; FPT, frequency pattern test.

Table 4. Pairwise Comparisons of DPT between groups

Groups	No HA	Bilateral HA	Normal Hearing	Unilateral Aided	Unilateral Unaided
No HA	-	<0.001*	<0.001*	<0.001*	<0.001*
Bilateral HA	<0.001 *	-	0.984	0.826	0.129
Normal hearing	<0.001 *	0.984	-	0.346	0.027*
Unilateral aided	<0.001 *	0.826	0.346	-	0.970
Unilateral unaided	<0.001 *	0.129	0.027*	0.970	-

The cells in the table represent Tamhane multiple comparisons *P* values, *The mean difference is significant at the .05 level. (2-tailed). No HA, no hearing aid group; Bilateral HA, bilateral hearing aid group; Unilateral aided, with hearing aid ear of unilateral HA group; Unilateral unaided, without hearing aid ear of unilateral HA group. DPT, duration pattern test; HA, hearing aid.

Table 5. Pairwise Comparisons of TPS between groups

Groups	No HA	Bilateral HA	Normal Hearing	Unilateral Aided	Unilateral Unaided
No HA	-	<0.001*	<0.001*	<0.001*	<0.001*
Bilateral HA	<0.001*	-	0.069	0.441	0.012*
Normal hearing	<0.001*	0.069	-	0.004*	<0.001*
Unilateral aided	<0.001*	0.441	0.004*	-	0.874
Unilateral unaided	<0.001*	0.012*	<0.001*	0.874	-

The cells in the table represent Tamhane multiple comparisons *P* values, *The mean difference is significant at the .05 level. (2-tailed). No HA, no hearing aid group; Bilateral HA, bilateral hearing aid group; Unilateral aided, with hearing aid ear of unilateral HA group; Unilateral unaided, without hearing aid ear of unilateral HA group. TPS, total percentage score; HA, hearing aid.

The mean GIN threshold of the no HA group, bilateral HA group, normal hearing group, aided and unaided ear of unilateral HA group was 4.74 ms (1.18), 2.64 ms (0.71), 2.65 ms (0.66), 2.95 ms (1.00), and 3.05 ms (1.09), respectively (Table 2). Ranked from highest to lowest, GIN threshold results were the reverse of TPS results, as expected (Table 2). The GIN threshold of the no HA group was significantly higher than that of all other groups ($P < .001$; Table 6). No significant differences in GIN threshold were discovered between the bilateral HA group and other groups except for no HA group ($P > .05$; Table 6). Moreover, there were no significant differences between the aided and unaided ears of the unilateral HA group ($P > .05$; Table 6).

Discussion

Amplification is one of the main intervention methods for hearing rehabilitation. Understanding the functional consequences of hearing loss requires understanding the effects on central auditory processing (CAS), as well as processing at the cochlea and auditory nerve level. Decreases in the sound-evoked activity of the auditory nerve due to hearing loss also reduce the input signal transmitted to the brain and the neural activity in the CAS.

In some studies with adult animals, it was reported that increases in spontaneous and stimulus-induced nerve firings were observed in the auditory system following cochlear lesion.[19-21] It has been suggested that it may be due to homeostatic plasticity to regulate neural excitability.[22]

In other studies, the effects of conductive hearing impairment on sound-evoked neural activity have been evaluated to understand whether this effect is due to sensorineural deprivation or sensorineural pathology, and it has been shown that conductive hearing impairment causes increased neural firing in subcortical pathways.[4,23-27] It is thought to be important to investigate the effects of CHL in order to evaluate changes in supraliminal auditory processing. In our study, we examined the effect of auditory deprivation in conductive pathologies on temporal processing with behavioral tests. As a result, the GIN, DPT, and FPT results of patients with conductive pathology

Table 6. Pairwise Comparisons of Gaps in Noise Threshold Between Groups

Groups	No HA	Bilateral HA	Normal Hearing	Unilateral Aided	Unilateral Unaided
No HA	-	<0.001*	<0.001*	<0.001*	<0.001*
Bilateral HA	<0.001*	-	0.1000	0.815	0.641
Normal hearing	<0.001*	0.1000	-	0.874	0.027*
Unilateral aided	<0.001*	0.815	0.874	-	1.000
Unilateral unaided	<0.001*	0.641	0.027*	1.000	-

The cells in the table represent Tamhane multiple comparisons *P* values, *The mean difference is significant at the .05 level. (2-tailed). No HA, no hearing aid group; Bilateral HA, bilateral hearing aid group; Unilateral aided, with hearing aid ear of unilateral HA group; Unilateral unaided, without hearing aid ear of unilateral HA group. HA, hearing aid.

were worse when compared to those with normal hearing. In addition, the difference in the use of bilateral and unilateral HAs in the test scores indicates that binaural stimulation is important in conductive pathologies. As we know, conductive pathologies typically result in reduced sound energy reaching the cochlea, while the cochlea and CAS remain largely intact.[28] A number of studies have evaluated CHL to investigate neural gain changes. In an adult study to investigate central gain changes, they occluded 1 ear for 30 hours and then evaluated the ipsilateral acoustic reflex threshold (ART). As a result, a non-significant reduction was obtained compared to the initial ART test. A significant reduction in ART of the occluded ear was noted after 1 week of unilateral earplugging with prolonged duration.[29] The results of these studies are indirect evidence of an increase in neuronal response acquisition in the subcortical auditory pathways and a change in neuronal processing in the CAS after prolonged sensory deprivation.[27,29-31] Gürses et al[32] who conducted research on a group of 30 adults with unilateral deafness (SNHL) obtained the temporal processing result measured using the duration pattern and random gap detection tests with lower performance. They noted that this result may be due to some degree of deficiencies in temporal processing of difficulties in speech recognition in noise and that asymmetric inputs due to unilateral hearing loss will affect sound processing at the inferior colliculus level, which plays a critical role in signal integration (e.g., encoding amplitude modulation and spatial localization cues, etc.). In our study, the effects of auditory temporal processing in conductive pathologies using unilateral HAs showed results compatible with those found in the literature. Although the difference was not statistically significant in our study, the performance of the hearing-aid side of the unilateral HA group was better than the no-hearing-aid side.

Additionally, Tucci et al[4] showed that the relative sizes of neuron dendrites in the medial superior olive innervated by axons derived from each ear in animals change after a unilateral conductive loss, and they associated this with the fact that long-term CHL can change the anatomy and physiology of the central auditory system. Moore et al[7] stated that bilateral CHL had a negative effect on temporal resolution and auditory backward masking in ferrets. In the same study, they stated that after the hearing returned to normal, the BM thresholds of all ferrets returned to normal, but this process took about 2 years for some. In our study, we determined that patient with long-term conductive pathologies using HA performed better in temporal processing than those who did not use HA. This, in line with our study results, emphasizes the importance of rehabilitating conductive pathologies as quickly as possible.

In alignment with our study, Bayat et al[12] examined the effects of conductive pathology alone on GIN results without HA effects and compared with normal hearing. In line with our study, they obtained higher GIN thresholds and significantly lower total percentage scores from those with conductive pathology than from those with normal hearing.

In addition to conductive pathology, there are studies indicating that many factors such as sensorineural hearing loss, use of cochlear implants, and aging also affect auditory processing. Musiek and Chermak[33] stated in their study that the DPT was insensitive to pathologies at lower levels than the auditory cortex, but evidence is still lacking on whether long-term peripheral hearing loss affects the perception of duration pattern sequences.

Okada et al[34] in their retrospective study found that patients with chronic CHL had lower speech recognition scores on the side of pathology when compared with the healthy side. Animal studies showed prolonged adult-onset CHL causes cochlear synaptopathy.[35,36] These results show that long-term, adult-onset CHL causes cochlear synopathy and that at least moderate unilateral conduction pathologies have worse word recognition scores on the affected side, even if the bone conduction thresholds are bilaterally symmetrical and within normal limits as predicted in a dated animal study. This study also supports the idea that patients with unilateral chronic conduction pathologies where hearing cannot be medically improved, should benefit from either conventional amplification, or an osseointegrated device. In our study, although the word discrimination scores of patients with conductive pathology were good in silence, it was observed that their auditory temporal processing was affected due to insufficient amplification. This makes us think that in addition to the speech recognition test in silence, speech recognition tests in noise in conduction pathologies will contribute to this field of research.

As a result, in accordance with the literature, our study indicates that individuals without HAs need a longer duration to detect gaps in the GIN test than individuals with normal hearing and HAs in bilateral CHL. Similar results were observed in DPT and FPT test performances. These results show that conductive pathologies affect temporal processing when they are not rehabilitated. As a result, in patients with CHL, the auditory deprivation that occurs as the insufficient auditory stimulation continues affects temporal processing, even at the level of intensity they feel comfortabe. Although there are studies in the literature examining the effects of CHL on auditory processing, the HA effect has not been examined. In this respect, our study will contribute to the literature.

A number of methodological limitations specific to patient selection must be accepted in studies of conductive pathologies. In our study, the duration of hearing loss and the duration of HA use were not analyzed because the patients were not certain when their CHL started. We used information obtained from patient medical records to estimate the duration of hearing loss, and the inclusion criteria were as follows: minimum of 5 years of chronic CHL.

Conclusion

Conductive pathologies can have a long-term effect on temporal processing, causing mild to moderate hearing loss without treatment or rehabilitation, but this negative impact can be reversed with early intervention. Auditory verbal training, as well as effective amplification methods, can improve listening performance and speed recovery. The results show the necessity of evaluating temporal resolution in long-term conductive pathologies and that GIN, DPT, and FPT tests are a practical tests that can be used specifically for these patients.

Ethics Committee Approval: This study was approved by Ethics committee of Marmara University, (Approval No: 09.2018.239).

Informed Consent: Written informed consent was obtained from all participants who agreed to take part in the study.

Peer-review: Externally peer-reviewed.

Author Contributions: Concept – M.T.T., A.Ç.; Design – M.T.T., A.Ç.; Supervision – A.Ç.; Materials – M.T.T., A.Ç.; Data Collection and/or Processing – M.T.T.; Analysis and/or Interpretation – M.T.T., A.Ç., E.A.; Literature Review – M.T.T.; Writing – M.T.T.; Critical Review – A.Ç., E.A.

Declaration of Interests: The authors have no conflict of interest to declare.

Funding: The authors declared that this study has received no financial support.

References

1. Tollin DJ. The development of sound localization mechanisms. In: Popescu M V., Polley DB, eds. *Monaural Deprivation Disrupts Development of Binaural Selectivity in Auditory Midbrain and Cortex*. *Neuron*. 2010;65(5):718-731. Available at: https://linkinghub.elsevier.com/retrieve/pii/S0896627310001364.
2. Blumberg MS, Freeman JH, Robinson SR, eds. Oxford handbook of developmental behavioral neuroscience [internet]. Oxford: Oxford University Press; 2009. Available at: http://www.oxfordhandbooks.com/view/10.1093/oxfordhb/9780195314731.001.0001/oxfordhb-9780195314731.
3. Doyle WJ, Webster DB. Neonatal conductive hearing loss does not compromise brainstem auditory function and structure in rhesus monkeys. *Hear Res*. 1991;54(1):145-151. [CrossRef]
4. Tucci DL, Cant NB, Durham D. Effects of conductive hearing loss on gerbil central auditory system activity in silence. *Hear Res*. 2001;155(1-2):124-132. [CrossRef]
5. Sumner CJ, Tucci DL, Shore SE. Responses of ventral cochlear nucleus neurons to contralateral sound after conductive hearing loss. *J Neurophysiol*. 2005;94(6):4234-4243. [CrossRef]
6. Xu H, Kotak VC, Sanes DH. Conductive hearing loss disrupts synaptic and spike adaptation in developing auditory cortex. *J Neurosci*. 2007;27(35):9417-9426. [CrossRef]
7. Moore DR, Hartley DEH, Hogan SCM. Effects of otitis media with effusion (OME) on central auditory function. *Int J Pediatr Otorhinolaryngol*. 2003;67(suppl 1):S63-S67. [CrossRef]
8. Do Amaral MIR, Colella-Santos MF. Temporal resolution: performance of school-aged children in the GIN - Gaps-in-noise test. *Braz J Otorhinolaryngol*. 2010;76(6):745-752. [CrossRef]
9. Koravand A, Jutras B, Roumy N. Peripheral hearing loss and auditory temporal ordering ability in children. *Int J Pediatr Otorhinolaryngol*. 2010;74(1):50-55. [CrossRef]
10. Campbell K, Macdonald M. The effects of attention and conscious state on the detection of gaps in long duration auditory stimuli. *Clin Neurophysiol*. 2011;122(4):738-747. [CrossRef]
11. Shannon RV, Zeng FG, Kamath V, Wygonski J, Ekelid M. Speech recognition with primarily temporal cues. *Science*. 1995;270(5234):303-304. [CrossRef]
12. Bayat A, Farhadi M, Emamdjomeh H, Saki N, Mirmomeni G, Rahim F. Efeito da Perda Auditiva Condutiva Na Função Auditiva Central. *Braz J Otorhinolaryngol*. 2017;83(2):137-141. [CrossRef]
13. Cesur S, Derinsu U. Temporal processing performance in cochlear implant users. *J Am Acad Audiol*. 2018;8(2):242-243. Available at: http://libproxy.unl.edu/login?url=http://search.ebscohost.com/login.aspx?direct=true&db=aph&AN=131274927&site=ehost-live.
14. Trehub SE, Schneider BA, Henderson JL. Gap detection in infants, children, and adults. *J Acoust Soc Am*. 1995;98(5 Pt 1):2532-2541. [CrossRef]
15. Cesur S, Derinsu U. Effect of aging on temporal resolution. *Turk J Geriatr*. 2017;20(3):178-186. Available at: http://libproxy.unl.edu/login?url=http://search.ebscohost.com/login.aspx?direct=true&db=aph&AN=131274927&site=ehost-live.
16. Musiek FE, Shinn JB, Jirsa R, Bamiou DE, Baran JA, Zaida E. GIN (gaps-in-noise) test performance in subjects with confirmed central auditory nervous system involvement. *Ear Hear*. 2005;26(6):608-618. [CrossRef]
17. Musiek FE. Frequency (pitch) and duration pattern tests. *J Am Acad Audiol*. 1994;5(4):265-268.
18. Füllgrabe C, Moore BCJ, Stone MA. Age-group differences in speech identification despite matched audiometrically normal hearing: contributions from auditory temporal processing and cognition. *Front Aging Neurosci*. 2015;7:1-25.
19. Roberts LE, Eggermont JJ, Caspary DM, Shore SE, Melcher JR, Kaltenbach JA. Ringing ears: the neuroscience of tinnitus. *J Neurosci*. 2010;30(45):14972-14979. [CrossRef]
20. Schaette R. Tinnitus in men, mice (as well as other rodents), and machines. *Hear Res*. 2014;311:63-71. [CrossRef]
21. Brotherton H, Plack CJ, Maslin M, Schaette R, Munro KJ. Pump up the volume: could excessive neural gain explain tinnitus and hyperacusis? *Audiol Neurootol*. 2015;20(4):273-282. [CrossRef]
22. Turrigiano GG. Homeostatic plasticity in neuronal networks: the more things change, the more they stay the same. *Trends Neurosci*. 1999;22(5):221-227. [CrossRef]
23. Cai S, Ma WLD, Young ED. Encoding intensity in ventral cochlear nucleus following acoustic trauma: implications for loudness recruitment. *J Assoc Res Otolaryngol*. 2009;10(1):5-22. [CrossRef]
24. Dehmel S, Pradhan S, Koehler S, Bledsoe S, Shore S. Noise overexposure alters long-term somatosensory-auditory processing in the dorsal cochlear nucleus-possible basis for tinnitus-related hyperactivity? *J Neurosci*. 2012;32(5):1660-1671. [CrossRef]
25. Hickox AE, Liberman MC. Is noise-induced cochlear neuropathy key to the generation of hyperacusis or tinnitus? *J Neurophysiol*. 2014;111(3):552-564. [CrossRef]
26. Chambers AR, Resnik J, Yuan Y, et al. Central Gain Restores Auditory processing following Near-Complete Cochlear Denervation. *Neuron*. 2016;89(4):867-879. [CrossRef]
27. Maslin MRD, Munro KJ, Lim VK, Purdy SC, Hall DA. Investigation of cortical and subcortical plasticity following short-term unilateral auditory deprivation in normal hearing adults. *NeuroReport*. 2013;24(6):287-291. [CrossRef]
28. Moore DR, Hutchings ME, King AJ, Kowalchuk NE. Auditory brain stem of the ferret: some effects of rearing with a unilateral ear plug on the cochlea, cochlear nucleus, and projections to the inferior colliculus. *J Neurosci*. 1989;9(4):1213-1222. [CrossRef]
29. Decker TN, Howe SW. Short-term auditory deprivation: effect on brainstem electrical response. *Hear Res*. 1981;4(3-4):251-263. [CrossRef]
30. Munro KJ, Blount J. Adaptive plasticity in brainstem of adult listeners following earplug-induced deprivation. *J Acoust Soc Am*. 2009;126(2):568-571. [CrossRef]
31. Munro KJ, Turtle C, Schaette R. Plasticity and modified loudness following short-term unilateral deprivation: evidence of multiple gain mechanisms within the auditory system. *J Acoust Soc Am*. 2014;135(1):315-322. [CrossRef]
32. Gürses E, Türkyılmaz MD, Kalaycıoğlu C, et al. Evaluation of temporal and suprasegmental auditory processing in patients with unilateral hearing loss. *Auris Nasus Larynx*. 2020;47(5):785-792. [CrossRef]
33. Musiek FE, Chermak GD. Psychophysical and behavioral peripheral and central auditory tests [internet]. *Handbook of Clinical Neurology*. 2015;129:313-332. [CrossRef]
34. Okada M, Welling DB, Liberman MC, Maison SF. Chronic conductive hearing loss is associated with speech intelligibility deficits in

patients with normal bone conduction thresholds. *Ear Hear*. 2020;41(3):500-507. [CrossRef]

35. Liberman MC, Liberman LD, Maison SF. Chronic conductive hearing loss leads to cochlear degeneration. *PLoS ONE*. 2015;10(11): e0142341. [CrossRef]

36. Lobarinas E, Salvi R, Ding D. Insensitivity of the audiogram to carboplatin induced inner hair cell loss in chinchillas. *Hear Res*. 2013;302:113-120. [CrossRef]