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Original Article

Role of Cochlear Nerve Diameter as a Prognostic Indicator for Hearing Recovery in Older Adults with Idiopathic Sudden Sensorineural Hearing Loss

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BACKGROUND: Idiopathic sudden sensorineural hearing loss is a disabling condition that lowers the quality of life specifically in older adults living alone. It is crucial to determine the outcome of the disease and to offer early treatment to prevent isolation caused by hearing impairment in this population. The objective of our study was to investigate whether the initial cochlear nerve thickness may predict the outcome of hearing recovery in older adults with idiopathic sudden sensorineural hearing loss.

METHODS: The study population was composed of older adults that were referred with idiopathic sudden sensorineural hearing loss in 1 ear. Long-term audiological data of the cohort were analyzed according to Siegel's criteria on hearing recovery and were grouped according to complete recovery or treatment failure. Cochlear nerve diameters of the diseased and safe ears of each group, measured on reformatted images on magnetic resonance imaging, at the fundus, in the mid-internal acoustic canal, and at the entry point into the Pons were compared in each group and between groups.

RESULTS: Mean cochlear nerve diameter was significantly larger in the recovered older adults (1.11 ± 0.27 mm) than in the non-recovered adults (0.94 ± 0.21 mm) at the mid-internal acoustic canal (Student's *t*-test, $P < .05$). Cochlear nerve thickness at mid-internal acoustic canal (≤0.8 mm) sensitivity for recovery failure was 89% and displayed an odds ratio 5.333, 95% CI (1.000-28.435).

CONCLUSION: Cochlear nerve thickness in mid-internal acoustic canal in non-recovered older adults with idiopathic sudden sensorineural hearing loss is significantly thinner than the completely recovered group. Older adults with mid-internal acoustic canal cochlear nerve greatest diameter cutoff level of ≤0.8 mm are 5.33 times more exposed to recovery failure.

KEYWORDS: Cochlear nerve, ear surgery, hearing loss, sudden, magnetic resonance imaging

INTRODUCTION

Idiopathic sudden sensorineural hearing loss (ISSHL) is an otologic emergency, and while being researched intensively, its origin still remains not fully explained. The undefined pathogenesis of the disease gives rise to unpredictable outcomes that may sometimes be unsatisfactory in gaining normal hearing thresholds. Obviously, this condition lowers the quality of patients' lives of every age group, but specifically disabling older adults, who live alone. Several studies based on laboratory and clinical findings, through the use of imaging modalities such as a computed tomography (CT) scan and magnetic resonance imaging (MRI), have identified genetic abnormalities, infections, and trauma of the inner ear, as well as vascular or neoplastic diseases of the pontocerebellar angle that may be some causes of hearing loss. Furthermore, the strong correlation between the cochlear nerve (CN) diameter and total spiral ganglion cells, elucidated in recent imaging studies, helped clinicians to evaluate the CN for the success of cochlear implants for potential candidates with acquired or congenital hearing loss among children and younger age groups.[1,2]

Since the late 1990s, based on MRI findings denoting inflammatory changes in the labyrinth and neural structures in ISSHL, treatment has been focused on the urgent reversal of this inflammation using systemic steroids as a first-line therapy, along with the addition of hyperbaric oxygen (HBO) and intratympanic steroid in patients needing salvage treatment.[3]

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A great majority of publications concerning either the outcome or clinical and laboratory findings of ISSHL reported data obtained from younger cohorts. However, there is a paucity of information about the etiological factors and the outcome of the disease among older age groups. Differing from the previous research analyzing younger populations, we aimed to investigate the differences in CN sizes of older adults who either completely recovered or did not recover at all, in order to define the role of the initial CN size in predicting the outcome of hearing recovery in older people after an attack of ISSHL.

MATERIAL AND METHODS

This entailed a long-term follow-up of audiometric evaluation and retrospective imaging reviews of older adults that were diagnosed with ISSHL at an otorhinolaryngology clinic of a tertiary referral center.[4] An Ethical Committee approval was obtained from SBU Haydarpaşa Numune E.R. Hospital ethics committee, in accordance with the Helsinki Declaration (registration ID: KAEK 2012/20). Patients aged ≥60 years of age who were treated for ISSHL between 2012 and 2018 were invited for long-term audiologic evaluation in 2020. Informed consent was obtained from all participants.

Eligibility criteria for inclusion were as follows: being ≥60 years of age, having no history of previous hearing loss, ISSHL in 1 ear, no known systemic disease, being patients who had undergone simultaneous CT and MRI of the temporal bone with no evidence of any congenital abnormality or trauma in either part of the inner, middle, and external ear structures, and having had treatment that started within 5 days of the symptom onset. Exclusion criteria were as follows: SSHL with a known cause regarding patients' laboratory and imaging findings and past medical history (systemic disease, autoimmune disorders, hypertension, diabetes mellitus, coagulation disorders), patients who had not undergone either CT and MRI, referral to the clinic later than 5 days after symptom onset, and being patients of <60 years of age.

Hearing recovery was classified according to Siegel's hearing recovery rate.[5] This is a system based on the average gain in dB at 500, 1000, 2000, and 4000 Hz. Siegel's classification details are as follows:

(i) Complete recovery: Patients whose final hearing level was better than 25 dB regardless of the size of the gain.
(ii) Partial recovery: Patients who showed more than 15 dB of gain and whose final hearing level was between 25 and 45 dB.
(iii) Slight recovery: Patients who showed more than 15 dB of gain and whose final hearing level was poorer than 45 dB.
(iv) No recovery: Patients who showed <15 dB of gain or whose final hearing level was poorer than 75 dB.

Audiologic data were collected and grouped with regard to their long-term recovery rate. Of these, data of the cohort whose hearing had either completely recovered or not recovered at all were included in the study. Cochlear nerve diameters of the diseased and safe ears measured at 3 points have been detailed in the next section and were compared in each group and between groups.

A General Electric (GE) Signa 1.5 T MRI system (GE Healthcare, Milwaukee, WI, USA) with an 8-channel head coil was used for imaging, while the MRI was performed with T2-weighted fast spin-echo images of the whole brain (TR, 4500 ms; TE, 104 ms; NEX, 1.5; section thickness, 5.5 mm; intersection spacing, 1.5 mm; matrix size, 352/352). The standard temporal bone protocol included axial and coronal 3-dimensional (3D) T1-weighted images (TR, 12.3 ms; TE, 5.4 ms; NEX, 2; section thickness, 0.8 mm; intersection spacing, 0.4 mm; matrix size, 256/256, field of view, 200/200 mm), axial and sagittal 3D fast imaging employing steady-state acquisition (FIESTA) sequence images (TR, 5.9 ms; TE, 2.3 ms; NEX, 4; flip angle, 65°; section thickness, 0.8 mm; intersection spacing, 0.6 mm; matrix size, 416/416, field of view, 200/200 mm).

Radiological data were analyzed retrospectively via the hospital workstation software (picture archiving and communications system, Marotech, Seoul, Korea). All images were evaluated by 2 radiologists blinded to the affected ear with 17 (neuroradiology board-certified) and 20 years of experience. Final measurements were made by consensus. An open-source Digital Imaging and Communications in Medicine imaging was used to view the soft tissue window at 100% magnification. Oblique sagittal reformatted 3D-FIESTA images perpendicular to the long axis of the internal acoustic canal (IAC) from the entry point of the vestibulocochlear nerve to the brainstem to the exit point of the CN from the cochlea (fundus) were obtained from the real axial images of the nerve. Measurements of the greatest diameters of the CN trunk were recorded in a total of 6 points on both affected and control sides: (i) At the point nearest the fundus where the CN could be identified separately from the facial and vestibular nerves (Figure 1A and B), (ii) At the mid-internal acoustic canal (mid-IAC) 1.8 mm away from

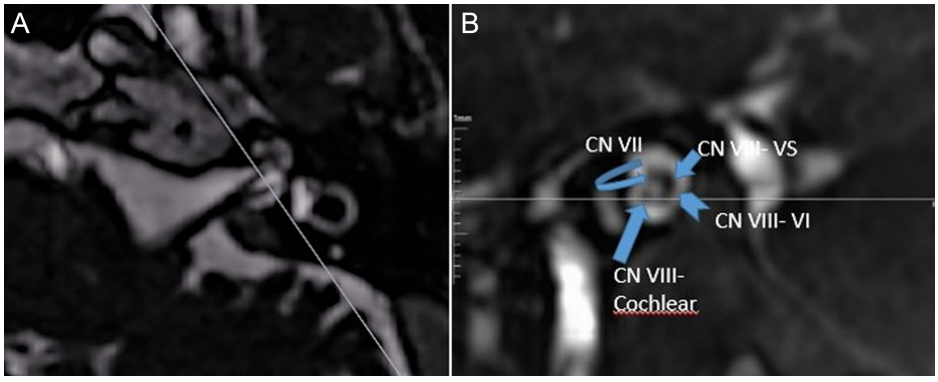

Figure 1. Cranial nerve (CN) VII, CN VIII cochlear and vestibular components at the fundus level of the cochlea: (A) axial section; (B) sagittal reformatted image. Rounded blue arrow shows the facial nerve, large arrow shows the cochlear nerve, upper arrowhead points out the superior vestibular nerve, and lower arrowhead points out the inferior vestibular nerve.

the first measurement point where the cochlear, vestibular, and facial nerves were also clearly identifiable (Figure 2A and B), (iii) At the entry point to the brainstem (Root entry zone) where the CN was determined before entering the brainstem (Figure 3A and B). Data obtained were compared between the safe and affected ears of the overall cohort and between completely recovered and non-recovered groups.

Number Cruncher Statistical System (NCSS) 2007 (NCSS, Kaysville, Utah, USA) was used for statistical analysis of the results. Descriptive (mean, SD, frequency, median) and quantitative statistical methods were used in the evaluation of the study data. The congruity of the quantitative data with normal distribution was questioned using graphical methods, Kolmogorov–Smirnov, and Shapiro–Wilk tests. The Student's *t*-test was used when comparing 2 groups of quantitative data with normal distribution, the Spearman's correlation analysis was used for evaluating the relationship between variables, and the paired sample *t*-test was used for comparing the measurements between diseased and safe ears. Qualitative data were compared using the Pearson's chi-square test. A receiver operating characteristic (ROC) curve analysis with a 95% CI and diagnostic tests (sensitivity, specificity, positive predictive value, and negative predictive value) were performed to determine the cutoff values of the parameters with an accepted *P* level of significance $P < .05$.

RESULTS

Of the 184 patients treated for ISSHL between 2012 and 2018, a total of 43 older adults (55.8% female and 44.2% male) met the inclusion criteria. The mean age of the patients was 64.04 ± 2.87 years of age with an age range varying from 60 to 69 years. Supplemental Digital Content 1 (SDC.1).

The mean age of the patients in the complete recovery group was 64.16 ± 2.69 (ranging between 60 and 68 years of age, with the median being 64) years of age; whilst the mean age of the non-recovery group was 63.8 ± 3.04 (ranging between 60 and 69 years of age with the median being 63) years of age. There was no statistically significant difference with regard to the mean age of the older patients who had either recovered fully or not recovered at all (Student's *t*-test, $P = .69$).

The period between the start of ISSHL and the long-term follow-up visit ranged from 2 to 7 years with a mean time interval of about 3.82 ± 1.32 years (median 4 years) (SDC.1).

Except for 3 (7%) patients who stopped corticosteroid medication due to side effects, 37 (86%) patients received combined systemic and intratympanic steroids and HBO therapy. Two (4.7%) patients were given only intratympanic steroids and 1 (2.3%) patient was given only systemic steroids with no additional HBO therapy.

The total recovery group consisted of 18 (41.9%) patients and the non-recovery group consisted of 25 (58.1%) patients, who were older adults that did not improve after treatment (SDC.1).

The mean air conduction/bone conduction thresholds of the recovery and non-recovery groups were 19 ± 7.63 dB /17.77 ± 6.95 dB, 75.64 ± 21.18 dB/62.08 ± 10.71 dB, respectively (SDC.2).

The overall mean CN diameters of the cohort's diseased and safe ears were at the fundus 0.73 ± 0.16 mm; 0.69 ± 0.15 mm, respectively. At the mid-IAC overall mean CN diameters of the affected and safe ears were 1.01 ± 0.25 mm and 1.05 ± 0.28 mm. Whilst at the root entry

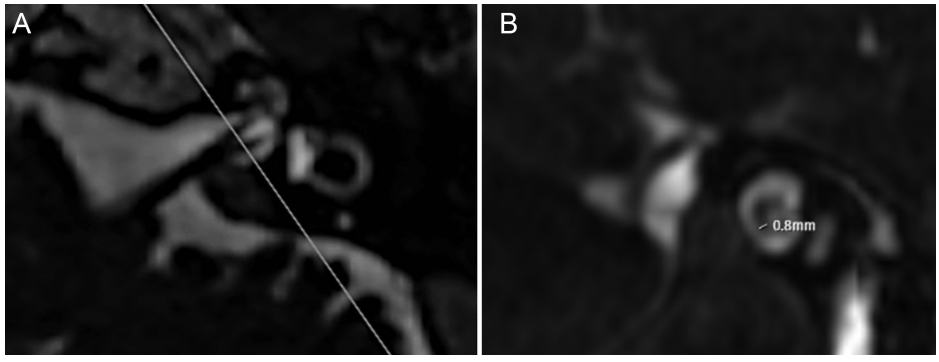

Figure 2. Cochlear nerve in the mid-internal acoustic canal level: (A) axial section; (B) sagittal reformatted image.

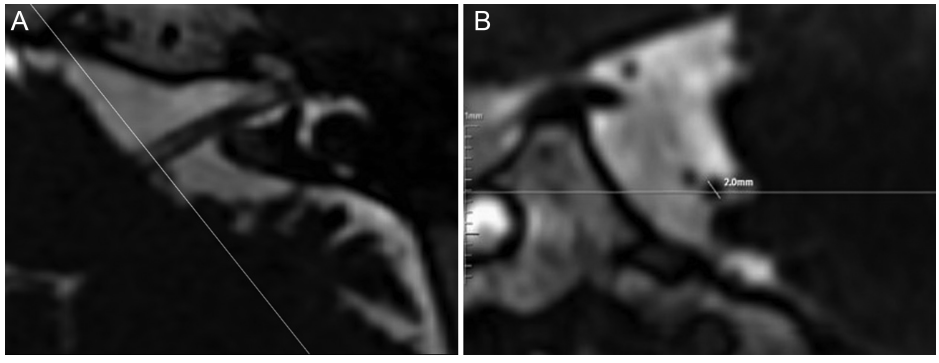

Figure 3. Cochlear nerve N VIII cochlear component near the entry zone into the brain stem: (A) axial section; (B) sagittal reformatted image.

Table 1. Comparison of Cochlear Nerve Diameters of the Diseased and Safe Ears in the Overall Cohort

			Diseased Ear	Safe Ear	*P*
Overall group (n = 43)	At the entry into the brainstem CN (mm)	Minimum–maximum (median)	0.7-1.6 (1.2)	0.7-1.8 (1.2)	**.604 NS**
		Mean ± SD	1.17 ± 0.22	1.15 ± 0.28	
	Mid-IAC CN (mm)	Minimum–maximum (median)	0.6-1.6 (0.9)	0.6-1.6 (1.0)	**.310 NS**
		Mean ± SD	1.01 ± 0.25	1.05 ± 0.28	
	Fundus CN (mm)	Minimum–maximum (median)	0.5-1.3 (0.7)	0.3-1.0 (0.7)	**.136 NS**
		Mean ± SD	0.73 ± 0.16	0.69 ± 0.15	
Non-recovery group (n = 25)	At the entry to brainstem CN (mm)	Minimum–maximum (median)	0.7-1.6 (1.2)	0.7-1.8 (1.1)	**.522 NS**
		Mean ± SD	1.15 ± 0.22	1.12 ± 0.29	
	Mid-IAC CN (mm)	Minimum–maximum (median)	0.6-1.4 (0.9)	0.6-1.6 (0.9)	**.203 NS**
		Mean ± SD	0.94 ± 0.21	1.00 ± 0.29	
	Fundus CN (mm)	Minimum–maximum (median)	0.5-1.0 (0.7)	0.3-0.9 (0.6)	**.540 NS**
		Mean ± SD	0.70 ± 0.15	0.68 ± 0.16	
Recovery group (n = 18)	At the entry to brainstem CN (mm)	Minimum–maximum (median)	0.9-1.6 (1.2)	0.7-1.7 (1.2)	**1.000 NS**
		Mean ± SD	1.20 ± 0.22	1.20 ± 0.26	
	Mid-IAC CN (mm)	Minimum–maximum (median)	0.7-1.6 (1.0)	0.6-1.6 (1.1)	**1.000 NS**
		Mean ± SD	1.11 ± 0.27	1.11 ± 0.26	
	Fundus CN (mm)	Minimum–maximum (median)	0.6-1.3 (0.7)	0.4-1.0 (0.7)	**.127 NS**
		Mean ± SD	0.77 ± 0.17	0.71 ± 0.15	

Paired sample *t*-test; $P < .05$.
CN, cochlear nerve; IAC, internal acoustic canal. NS: Non Significant p value.

zone, the CN's greatest diameters were 1.17 ± 0.22 mm and 1.15 ± 0.28 mm in the diseased and safe ears, respectively.

No statistically significant difference was observed in the overall cohorts with regard to CN diameters of the diseased and safe ears measured at the fundus, in the mid-IAC, and at the entry point to the brainstem (paired sample *t*-test, $P > .05$).

Likewise, there was no statistically significant difference between the diseased and safe ears of the either non-recovered or completely recovered groups regarding mean CN sizes at the fundus, in the mid-IAC, and at the entry point to the brainstem (paired sample *t*-test, $P > .05$) (Table 1).

Similar results were seen when comparing the diameters of the diseased CN at the fundus (0.77 ± 0.17 mm; 0.70 ± 0.15 mm) and the entry point to the brainstem (1.20 ± 0.22 mm; 1.15 ± 0.22 mm) in the completely recovered and non-recovered groups respectively. However, there was a significant difference between the recovered and non-recovered groups with regard to mean diseased CN diameters in the mid-IAC. The mean CN diameter was significantly larger in the recovered group (1.11 ± 0.27 mm) than in the non-recovered group (0.94 ± 0.21 mm) at the mid-IAC (Student's *t*-test, $P = 0.034$; $P < .05$) (Table 2). Older adults with a thicker CN diameter in the mid-IAC regained their original level of hearing after treatment.

Table 2. Comparison of Diseased Cochlear Nerve Diameters Between the Completely Recovered and Non-Recovered Groups

Total (n = 43)		Recovery		*P*
		Negative (n = 25)	Positive (n = 18)	
At the entry to brainstem CN (mm)	Minimum–maximum (median)	0.7-1.6 (1.2)	0.9-1.6 (1.2)	**.453 NS**
	Mean ± SD	1.15 ± 0.22	1.20 ± 0.22	
Mid-IAC CN (mm)	Minimum–maximum (median)	0.6-1.4 (0.9)	0.7-1.6 (1.0)	**.034* ss**
	Mean ± SD	0.94 ± 0.21	1.11 ± 0.27	
Fundus CN (mm)	Minimum–maximum (median)	0.5-1.0 (0.7)	0.6-1.3 (0.7)	**.149 NS**
	Mean ± SD	0.70 ± 0.15	0.77 ± 0.17	

*$P < .05$; Student's *t*-test.
CN, cochlear nerve; IAC, internal acoustic canal; NS: Non Significant; SS: Statistically Significant.

Table 3. Results of Diagnostic Scan Tests and Receiver Operating Characteristic (ROC) Curve Analysis of the Cochlear Nerve Diameters in the Internal Acoustic Canal with Regard to the Improvement of Idiopathic Sudden Sensorineural Hearing Loss in Older Adults

	Diagnostic Scan Tests					ROC Curve		*P*
	Cutoff	Sensitivity	Specificity	Positive Predictive Value	Negative Predictive Value	Area	95% CI	
Mid-IAC CN diameter (mm)	**≤0.8**	40.00	88.89	83.33	51.61	**0.672**	0.506-0.838	**.041*** SS

CN, cochlear nerve; IAC, internal acoustic canal; SS: Statistically Significant.
*$P < .05$.

Based on the significant difference between the mean affected CN diameters in the mid-IAC of the recovered and non-recovered groups, we aimed to investigate the cutoff value of the nerve diameter that may predict the outcome of ISSHL. The cutoff value was found to be 0.8 mm when using ROC curve analysis and diagnostic scan tests. The sensitivity, specificity, positive predictive value, and negative predictive value for 0.8 mm as a cutoff value were, respectively, 40.00%; 88.89%; 83.33%, and 51.61%. The area under the curve was 67.2% with a SD of 8.0%; a 95% CI (0.506-0.838, lower and upper limits) was observed for the ROC curve analysis of the data (Table 3).

There was a statistically significant relationship between the cutoff value (≤0.8 mm) of the CN diameter in the IAC and the worsening of ISSHL ($P = .037$; $P < .05$). The odds ratio for the CN diameter less than 0.8 mm in the IAC was found to be 5.333 (95% CI: 1.000-28.435). Older adults with a CN diameter thinner than 0.8 mm in the mid-IAC were 5.3 times more susceptible to persistent hearing loss after ISSHL (SDC.3).

DISCUSSION

Idiopathic sudden sensorineural hearing loss is a serious otologic emergency that needs early medical intervention for securing the oxygenation of the CN fibers. Predicting the outcome of ISSHL still poses challenges due to the multifactorial etiology of the disease. The mainstay of the treatment is to rule out cardiovascular and central nervous system pathologies as well as temporal bone fractures and traumas and to start systemic steroids for protecting the nerve integrity from degeneration caused by hypoxia.

Informing the patient with ISSHL about whether she/he will have a complete recovery or not is a distressing situation for the physician, because the percentage of totally recovered patients is far from being high, displaying only a complete recovery rate ranging between 28.3% and 39% according to past literature.[6-8]

Studies investigating the factors that may have a negative impact on complete recovery are still ongoing in the literature. Profound hearing loss at the onset of the disease, being in the older age group, the time interval exceeding 10 days between the onset of the disease and the start of treatment, systemic comorbidities like diabetes or hypertension, and associated vestibular system disorders are among the most reported negative factors in scientific studies.[9,10]

In the early 1990s with the advent of cochlear implants (CI), studies were oriented toward measuring the CN caliber to analyze the effect of the CN caliber on CI performance in congenital SHL.[11]

The cadaveric study conducted by Nadol and Xu[11] displayed the mean greatest dimensions of the CN as being 1.04 ± 0.11 mm (range: 0.90-1.20 mm) in healthy and 0.81 ± 0.15 mm (range: 0.50-1.20 mm) in deaf ears. Meanwhile, maximum diameters of the vestibulocochlear nerve were found to be 2.02 ± 0.08 mm (range: 1.90-2.10 mm) and 1.57 ± 0.26 mm (range: 1.00-2.10 mm) in normal and deaf ears, respectively. The authors observed the strong correlation between the CN and the total spiral ganglion cell count and assumed that MRI with a high precision degree could be a prognosticator of CI success.

Later on, the development of the high-resolution T2-weighted fast spin-echo MRI provided very precise imaging of the cochlear, vestibular, and facial nerves both in the IAC and in the cistern.[12]

The MRI evaluations are also approved by the American Academy of Otolaryngology-Head and Neck Surgery as a recommendation for ruling out retrocochlear pathologies in SSHL.[13]

A great majority of the papers on MRI visualization of the CN are centered on the measurement of the CN caliber in congenital HL or deaf adults. However, there is a shortage of studies done to investigate the outcome of ISSHL related to CN size among older adults. In case of the failure of hearing gain, early fitting should be recommended in this group who may be more prone to isolation if living alone. With regard to the drawbacks of ISSHL in the aging population, we conducted the present study to question the relation of CN size with the outcome of ISSHL in older adults. The status of the CN was examined along its entire length, from the fundus to the point of entry into the brain stem. Patients with bony CN canal stenosis or any other temporal bone abnormality depicted on CT were withdrawn from the study considering the risk of bias. The greatest CN diameters were compared between patients with and those without complete recovery in order to investigate the cause of healing failures in older adults in terms of CN size after ISSHL.

Since the maximum diameter of the CN is known to better correlate with the number of spiral ganglion cells, the smaller CN diameters and cross-sectional areas were not included when comparing the groups.[11] Instead, maximum diameters at the fundus and at the most cephalic part of the CN were added to the mid-IAC measurements.

Cochlear nerve diameters of the overall cohorts of the completely recovered and non-recovered groups were found to gradually increase from the fundus to the IAC and to the entry point of the brainstem, both in the diseased and safe ears. Moreover, CN diameters were observed to be not significantly different either in the diseased or safe ears of the overall patients. Likewise, no significant differences were observed between diseased and safe ears of each group with regard to CN diameters measured at the fundus, in the mid-IAC, and at the entry point to the brainstem (Table 3). This

statement was contrary to those of others who observed smaller CN size on the sides affected by acquired SSHL.[12] However, the cadaveric study held by Nadol and Xu[11] in 1992 demonstrated that the maximum diameter of the CN was independent of the side of the temporal bone or the duration of hearing loss.

Our statements bear no significant differences between ear sides with regard to CN diameters in older adults with ISSHL and are in line with the aforementioned author's statements. This sounds more realistic in temporal bones where there is no apparent canal stenosis on CT as is in our cohorts.

However, when analyzing the maximum diameters of the CN between groups that had completely recovered or not recovered, we observed a significant difference with regard to the maximum CN diameter in the mid-IAC ($P = .034$). Patients with the greatest CN diameters measuring 1.11 ± 0.27 mm in mid-IAC were actually able to regain their initial hearing levels after ISSHL, while those with 0.94 ± 0.21 mm were not. Furthermore, although not statistically significant, there was a tendency towards the CN diameters being smaller in the non-recovered patients at the fundus (0.70 ± 0.15 mm vs. 0.77 ± 0.17 mm in the non-recovery and complete recovery groups, respectively) and at the entry to Pons (1.15 ± 0.22 mm vs. 1.20 ± 0.22 mm in the non-recovery and complete recovery groups, respectively).[14]

Out of the total of the present cohorts, the CN diameters of the aged patients who recovered completely from ISSHL were greater than the cadaveric CN sizes of patients with normal hearing in Nadol and Xu's[11] study. However, they were smaller than the patients' CNs with normal hearing, measured by Jaryszak et al[15] on MRI.[11]

Normative data for the greatest CN diameters suggested by Jaryszak et al[15] were reported as 1.4 ± 0.21 mm. However, older adults who were either transiently or persistently affected by SSHL in the current study displayed greater CN diameters of 1.11 ± 0.27 mm and 0.94 ± 0.21 mm, respectively.

The reason why aged patients with CN diameters less than 1 mm (0.94 ± 0.21 mm) did not recover from ISSHL may be attributable to the strong correlation of the number of CN axons with spiral ganglion cells. Smaller numbers of spiral ganglion cells have been substantiated to be exposed to total degeneration and to failure compared to spontaneous regeneration. Moreover, smaller CN sizes have also been demonstrated to indicate fewer spiral ganglion cells and a reduced number of intact dendrites, as well as a reduced regeneration potential after SSHL.[16,17] However, even older people who had completely recovered from SSHL in the present study had smaller CN diameters than the younger patients of Jaryszak et al[15] with normal hearing. Apparently, smaller CN sizes in the present study underline the evidence of a thinning CN structure in older adults.[16,17]

Recently, reports of greater CN diameters of younger age groups with normal hearing (varying from 1.10 ± 0.21 mm to 1.34 ± 0.17 mm), have been published in literature.[18] However, there is not yet a consensus over the normative size of the CN in IAC and it is hard to draw an objective conclusion that patients with CN diameters less than the published normative data are candidates for an attack of SSHL or vice versa. In our opinion, measuring the CN size depends on the radiologist's interpretation and the brand name of the MR machine being used in the imaging. It would be preferable for an otologist to store and analyze imaging data interpreted by the same radiologist and captured by the same device.

Keeping this point in mind, we investigated the accuracy of our results by analyzing the cutoff value of CN sizes that may predict older adults who will not recover after SSHL. With a 95% CI (0.506-0.838), ≤0.8 mm was determined to be the CN size cutoff level with a sensitivity of 40% for patients whose hearing level will not improve. However, the specificity and positive predictive value of the ≤0.8 mm cutoff level (88.89% and 83.33%, respectively) were much higher than the sensitivity. Moreover, CN sizes of ≤0.8 mm were associated with a 5.33 times higher risk of recovery failure (95% CI: 1.000-28.435).

All older adults received the same treatment regimen in the present study, nonetheless, 88.89% of them with a CN size of ≤0.8 mm did not recover from the disease in the long term. In such circumstances, the questions that arose in our minds were whether it is necessary to use treatments such as steroids that have side effects or whether would it be better to use higher doses of this medication (if there is no contraindication for its use) in aged patients whose CN is ≤0.8 mm in its greater diameter.

Although the present study was constructed in a uniform group of patients that was followed up on for a long period of time, it still has some limitations. Smaller diameters and cross-sectional areas of the CN were ignored based on the correlation of spiral ganglion cell count with the greatest CN diameters, reported in an earlier publication.[11] Since there was no additional cadaver study comprising those who recovered from ISSHL, we were only able to include this data in our study. Another limitation was the small sample size of the study. The cohorts were constituted of aged patients with no comorbid conditions and those who had recovered completely or had not recovered at all from ISSHL. These were the reasons that restricted our sample size and sensitivity. However, the 5.33-fold association between the greatest CN diameters and recovery failure is adequate to inform patients of their rate of improvement failure.

To the best of our knowledge, this is the first study investigating the greatest diameters of the CN from the fundus to the entry point into the Pons on sagittal reformatted images on the MRI FIESTA sequence among older adults. Normative data reported by recent publications vary among countries, radiologists, and the MRI devices used. For future assessments, it would be preferable for an otologist to store measurement data for CN images interpreted by the same radiologist and the same MRI machine.

Cochlear nerve diameters do not differ between diseased and safe ears in older patients with ISSHL. The greatest diameter of the CN in the mid-IAC of aged patients who completely failed to improve is significantly smaller than those who improved completely after ISSHL. Older adults with the greatest CN diameter cutoff level of ≤0.8 mm at the mid-IAC are 5.33 times more exposed to total recovery failure. Although not statistically significant, the CN diameter measurements at the fundus and the entry point into the Pons of the aged, non-recovered ISSHL patients show a tendency of being smaller in its entire course than those who recovered completely.

Ethics Committee Approval: Ethical committee approval was received from the Ethics Committee of SBU University Haydarpaşa Numune E.R. Hospital (Approval No: HNEAH-KAEK 2012/20).

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

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