Loudness in the Normal-Hearing Ear of Single-Sided Deaf Patients

N. Di Maio

Student number: 6103642

Major research internship
May 2023 – August 2023

Supervisors

dr. H. Versnel
dr. J.A.A. van Heteren

Department of Otorhinolaryngology

University Medical Center Utrecht

List of abbreviations

ACALOS	Adaptive Categorical Loudness Scaling
ANOVA	Analysis of Variance
BCD	Bone Conduction Device
CI	Cochlear Implant, Cochlear Implantation
CLS	Categorical Loudness Scaling
CROS	Contralateral Routing of Sound hearing aid
CU	Categorical Units
HSE	Head Shadow Effect
NH	Normal-Hearing
PTA	Pure Tone Average
SNR	Speech to Noise Ratio
SPL	Sound Pressure Level
SSD	Single Sided Deafness
QoL	Quality of Life

Table of contents

Abstract

Background: Single-sided deafness (SSD) has been shown to lead to a reduction in inhibition and an increase in excitatory responses in inferior colliculus and auditory cortex in the ipsilateral hemisphere when sounds are presented to the normal ear. These increased excitatory responses may lead to an increase in loudness. Therefore, we examined SSD patients hypothesizing an increase in loudness compared to normal-hearing (NH) controls. Additionally, we examined the effect of cochlear implantation (CI) in the deaf ear on loudness in the normal ear of the SSD patients.

Methods: We applied the Oldenburg Adaptive Categorical Loudness Scaling (ACALOS) procedure monaurally on 16 adult NH subjects (age range 22-55 years) and 18 SSD subjects (age range 19-73 years). The NH subjects were tested at each ear and the SSD subjects were tested twice: 1 to 4 months before and 4 to 8 months after CI. Accordingly, data were fitted yielding 3 outcome measures: slope of the lower segment of the loudness function (m_{low}), slope of the higher segment (m_{high}), and the level at the intersection of the two slopes (L_{cut}). Additionally, the effect of age and PTA thresholds on loudness was measured.

Results: Firstly, the level at the intersection of the two slopes, L_{cut}, was lower for SSD subjects than for NH subjects by 2 to 8 dB ($p = 0.060$), most prominently at 0.5 and 1 kHz, which reflects an increase in loudness in the SSD group. Secondly, L_{cut} increased with older age and PTA thresholds. Thus, the older age and hearing loss resulted in a decrease of loudness, while SSD seemed to result in an increase of loudness. Lastly, CI of the deaf ear had no significant effect on loudness in the normal ear of the SSD patients.

Conclusion: Our data confirmed the hypothesis of an increase in loudness, although age and hearing loss counteracted this effect slightly. Nevertheless, we assume that the increase in loudness is caused by an increase in excitatory neural responses in the ipsilateral hemisphere, which in its turn is caused by a reduction in inhibition from the contralateral ear.

1. Introduction

Single-sided deafness

Single-sided deafness (SSD), also known as unilateral hearing loss, refers to a condition where an individual experiences significant hearing loss in one ear, while the other ear maintains normal to near-normal hearing abilities (Peters et al., 2021). It is defined as a hearing loss ≥70 dB HL in the affected ear, and a pure tone average (PTA) threshold of at least 25-30 dB HL in the good (Galvin et al., 2019; Kurz et al., 2019; Peters et al., 2021). It has been estimated that SSD affects between 12 and 27 per 100.000 adults among the general population (Zeitler & Dorman, 2019). Causes of SSD are vestibular schwannoma, Ménière's disease, and inflammatory diseases, such as otitis media and labyrinthitis (Baguley et al., 2006; Kurz et al., 2019; Usami et al., 2017). However, the etiology of SSD often remains unknown, which makes idiopathic sudden deafness the most common cause for SSD.

Most people with SSD experience problems in sound localization and speech perception in noise (Galvin et al., 2019; Peters et al., 2021). These issues occur because of the absence of three functional benefits seen only with binaural hearing: the head shadow effect (HSE), the binaural squelch effect, and binaural summation (see Fig. 1) (Zeitler & Dorman, 2019). Firstly, the HSE hinders the propagation of sound waves due to the head acting as an acoustic barrier and the necessity for sound waves to travel a greater distance to reach the opposite ear (Avan et al., 2015; Van Wanrooij & Van Opstal, 2004). Secondly, the binaural squelch effect occurs when speech and background noise are separated and both ears receive different input (Zeitler & Dorman, 2019). The brain will then suppress noise on the side with the best signal to noise ratio (SNR). Lastly, sound can be perceived up to 3 dB louder if it reaches both ears instead of one normal-hearing (NH) ear. This "doubling" of perceptual loudness is referred to as binaural loudness summation (Avan et al., 2015; Zeitler & Dorman, 2019). Most advantages of binaural hearing are attributed to the HSE (Bernstein et al., 2017; Brown et al., 2007).

Besides the difficulties that patients with SSD encounter in benefiting from the effects of binaural hearing, they also frequently suffer from tinnitus in the affected ear (Peters et al., 2021). All these hearing-related issues may result in a reduced quality of life (QoL). Therefore, appropriate intervention is of great importance for these patients.

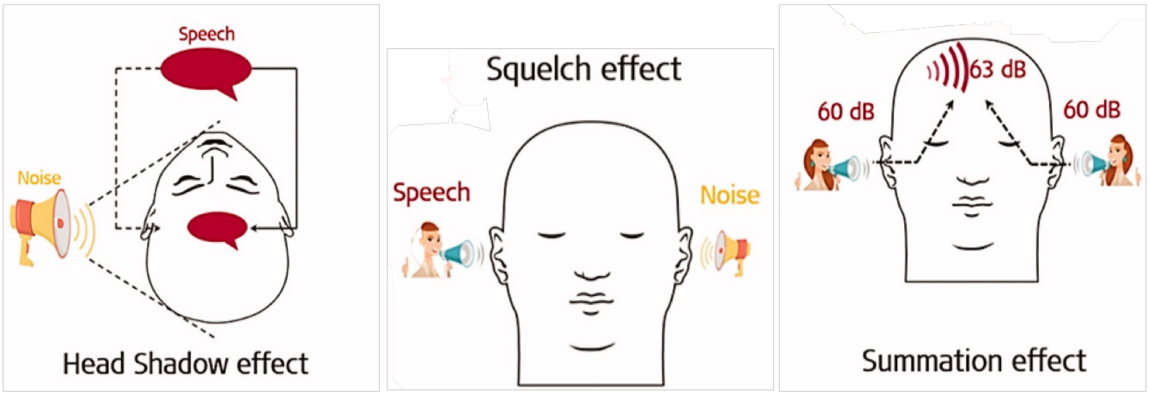


Fig. 1: Three functional benefits of binaural hearing: the head shadow effect (HSE), the squelch effect and the summation effect.

Treatment options

Traditional treatment options for patients with SSD are Contralateral Routing of Sound hearing aids (CROS) and Bone Conduction Devices (BCDs). CROS hearing aids are worn on the impaired ear and transfer signals to the better ear through a microphone and a transmitter. A BCD transmits sound from the poor ear to the cochlea of the better ear by vibration of the skull bone. However, the main limitation of CROS and BCDs is that binaural hearing is not restored, because both treatments do not provide auditory input to the deaf ear.

Cochlear implantation (CI), on the other hand, does provide input to the auditory nerve and helps to restore binaural hearing (Galvin et al., 2019; Macherey & Carlyon, 2014). Therefore, there are several SSD cochlear implant (CI) users worldwide, but most have been implanted "off label" and CIs are currently not approved for SSD patients (Galvin et al., 2019). Furthermore, CI improves sound localization and speech perception in noise (Galvin et al., 2019; Peters et al., 2021). Additionally, several studies have shown that CI can reduce tinnitus severity (Arts et al., 2012; Van de Heyning et al., 2008). In general, all these improvements lead to an enhanced QoL (Peters et al., 2021).

Loudness

Loudness refers to the subjective perception of sound intensity or volume (Dreschler & Lamoré, 2007a). This sound intensity is typically measured in decibels (dB), which is a logarithmic scale that quantifies sound pressure levels (SPL). The perceived loudness of a sound varies, next to stimulus level, with several other physical factors such as the frequency spectrum, the duration, and individual differences in hearing sensitivity. Categorical loudness scaling (CLS) is a commonly used tool in daily practice to assess individual loudness perception (Brand & Hohmann, 2002; Oetting et al., 2014).

Instructions for this test are simple, patients must categorize loudness in terms of “soft” and “loud”. CLS procedures determine loudness functions not only at their thresholds but also at moderate levels, which are most relevant for daily life situations (Brand & Hohmann, 2002). Furthermore, the measurement can be conducted quickly, and the results can be compared between people with normal hearing (NH) and those with SSD (Dreschler & Lamoré, 2007b).

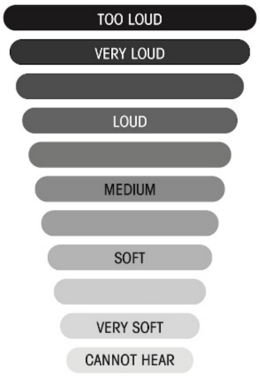


Fig. 2: Category scale with 11 response alternatives used by the participants to subjectively rate the loudness during Categorical Loudness Scaling (CLS). Every response option indicates a categorical unit (CU) from 0 to 50, which were used for data storage and analysis.

Aim of this study

The present study aimed to investigate loudness in the good ear of SSD patients, since literature mostly focusses on the deaf ear. In a study by Moore et al. (1997), the surgical removal of one cochlea led to new connections between the cochlear nucleus and brainstem in the intact ear. Additionally, even when acquired in adulthood, SSD has been shown to lead to a reduction in inhibition and an increase in excitatory responses in inferior colliculus and auditory cortex in the ipsilateral hemisphere when sounds are presented to the normal ear (Kral, Hubka, et al., 2013; Mossop et al., 2000; Tillein et al., 2016). These increased excitatory responses may lead to an increase in loudness. Therefore, we examined adult subjects with acquired SSD hypothesizing an increase in loudness compared to NH controls. Secondly, we examined the effect of CI in the deaf ear on loudness in the good ear of the SSD patients.

2. Methods

Study population

We evaluated NH subjects and adults with acquired SSD who received a CI in the CINGLE-study (Peters et al., 2021). All patients were admitted to the University Medical Center Utrecht, the Netherlands, between 13/08/2015 and 19/08/2019. SSD patients were eligible for inclusion if they had a PTA threshold at 0.5, 1, 2, and 4 kHz of maximum 30 dB HL in the good ear and a minimum of 70 dB HL in the poor ear, while for NH adults a maximum of 15 dB HL in both ears was required. For further details about inclusion and exclusion criteria of SSD patients, see Peters et al. (2021). The PTA threshold of the NH subjects in Table 1 displays the average PTA threshold of both ears combined. In the present study, we exclusively focused on the good ear of SSD patients, we did not consider the deaf ear. We divided the participants into 2 groups: 1) NH adults, 2) the NH-ear of SSD patients before CI (SSD-NH pre-CI) and after CI (SSD-NH post-CI).

Loudness Scaling Procedure

Narrow-band noises with center frequencies at 0.5, 1, 2 and 4 kHz were presented monaurally to 16 adult NH subjects and 18 SSD patients implanted with a CI. The first 4 SSD patients received a Nucleus1 CI422 with a Slim Straight electrode array, the other 14 SSD patients were implanted with a Nucleus1 CI512 with a Contour Advance electrode array. All tests were conducted in a soundproof booth and the listeners were seated 1 m from the speaker. NH subjects were tested at each ear with the other ear plugged and covered by an earmuff. The SSD subjects were tested twice: 1 to 4 months before and 4 to 8 months after cochlear implantation (Peters et al., 2021). The average duration of CI use before the second test was 4.4 months. We applied the Oldenburg Adaptive Categorical Loudness Scaling (ACALOS) procedure. Subjects performed multiple procedures subsequently, lasting about 30 minutes in total. Firstly, a test was conducted at 1 kHz to make the subjects familiar with the procedure. Accordingly, stimulus levels were varied between -10 and 105 dB SPL, with a resolution of 1 dB. It was the participants task to rate the loudness of each stimulus using 11 different categorical units (CU) as shown in Fig. 2. The scale starts at 0 CU for “inaudible” and increases in steps of 5 CU until reaching 50 CU for “too loud”. The “categorical loudness level” is the level assigned to a given loudness, for example L_{35}, which corresponds to “loud”. L_{35} is expressed as the average dB SPL at which participants pressed the 35 CU. To see a detailed description of the test, we refer to Appendix 1.

Outcomes

Figure 3 displays data of the ACALOS procedure from one SSD patient. The offered stimulus levels (dB SPL) are displayed on the x-axis and the CU are displayed on the y-axis. The marked stars indicate responses of the subject according to the stimulus level. Afterwards, data were fitted according to Brand & Hohmann (2002) yielding 3 outcome measures: slope of the lower segment of the loudness function (m_{low}), slope of the higher segment (m_{high}), and the level at the intersection of the two slopes (L_{cut}) (see Fig. 4A). Furthermore, L_{40} was calculated by smoothening the transition region between the levels L_{30} and L_{50} (see Fig. 4B). The slope of the smoothed line was classified as $slope_{40}$. Lastly, the percentage of high loudness scores (N) was calculated by dividing the responses between L_{30} and L_{50} by the overall number of responses.

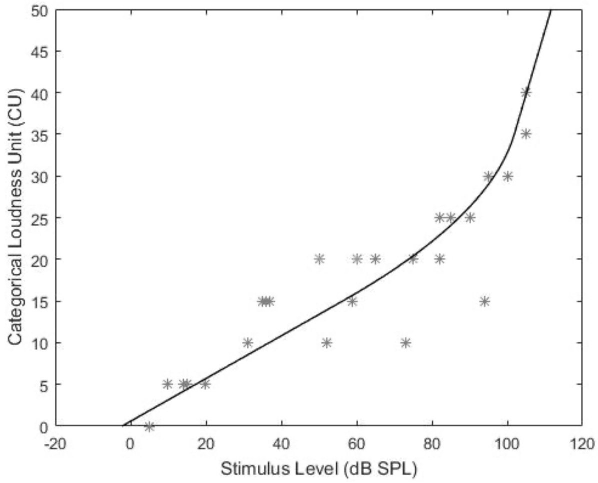


Fig. 3: Typical data of the Adaptive Categorical Loudness Scaling (ACALOS) procedure from a single-sided deaf (SSD) patient. The marked stars indicate responses of the subject according to the stimulus level (dB SPL). The black line shows the estimated loudness function.

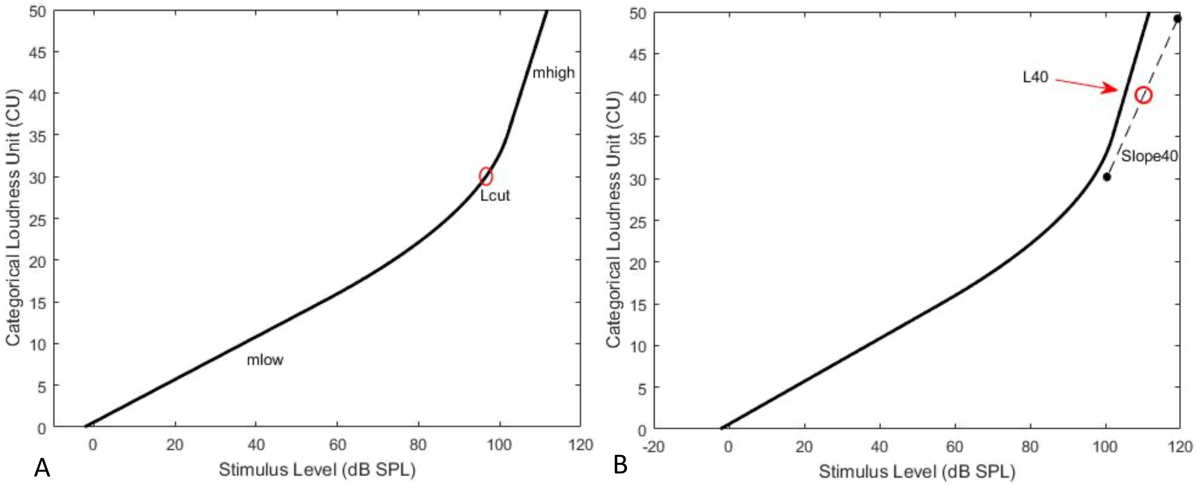


Fig. 4: Example of the outcome variables m_{low}, L_{cut}, m_{high}, L_{40}, $slope_{40}$, and the percentage of high loudness scores (N). The loudness function is based on the responses of a single-sided deaf (SSD) patient.

Statistical Analysis

Baseline characteristics were compared by Fischer’s Exact Test for categorical variables. Continuous variables were reported as means with standard deviations (SD) and medians with ranges and were subsequently compared using analysis of variance (ANOVA).

To compare between the right and left ear of NH adults we assessed the Wilcoxon signed-rank test. Additionally, NH adults were compared with SSD patients before CI by repeated measures ANOVA. The same test was used to examine whether there were any differences between the SSD subjects before and after CI. We applied the linear regression to see whether our data was correlated with PTA thresholds at 0.5-4 kHz and age at inclusion, as well as to examine the relationship between these two variables.

Data acquisition and graph plotting were conducted utilizing Matlab (MathWorks Inc., Natick, USA). All statistical analyses were performed using IBM SPSS Statistics 26 Academic. A p-value ≤ 0.05 was considered statistically significant.

3. Results

Baseline characteristics

The patient characteristics are displayed in Table 1. The mean age at inclusion of NH participants was 29.1 ± 9.8 (SD) years, and the mean age of SSD patients was 50.5 ± 14.5 (SD) years (p <0.001). Furthermore, for NH adults the median PTA of both ears combined was 4.4 dB (range -3.75 - 10.0), and the median PTA of the good ear of SSD patients was 12.5 dB (range 5.0 - 22.5; p <0.001). For an overview of the PTA of the NH individuals, we refer to Fig. 5. The median duration of deafness was 1.3 years (range 3 months – 8 years), and the most frequent cause of SSD was sudden deafness (61.1%).

Table 1: Patient characteristics.

Characteristics	SSD n = 18	NH n = 16	P value
Age at inclusion, years (mean ± SD)	50.5 ± 14.5	29.1 ± 9.8	<0.001
Gender, Male:Female	6:12	11:5	0.084
PTA 0.5-4 kHz (dB)			
Mean ± SD	13.0 ± 5.6	3.9 ± 3.9	<0.001
Median (range)	12.5 (5.0-22.5)	4.4 (-3.75-10.0)	
Deaf ear, AD:AS	8:10		
Duration of deafness, years (median ± range)	1.3 (0.3-8.0)		
Etiology, n (%)			
Unknown	2 (11.1)		
Sudden deafness	11 (61.1)		
Labyrinthitis	3 (16.7)		
Ménière’s disease	2 (11.1)		

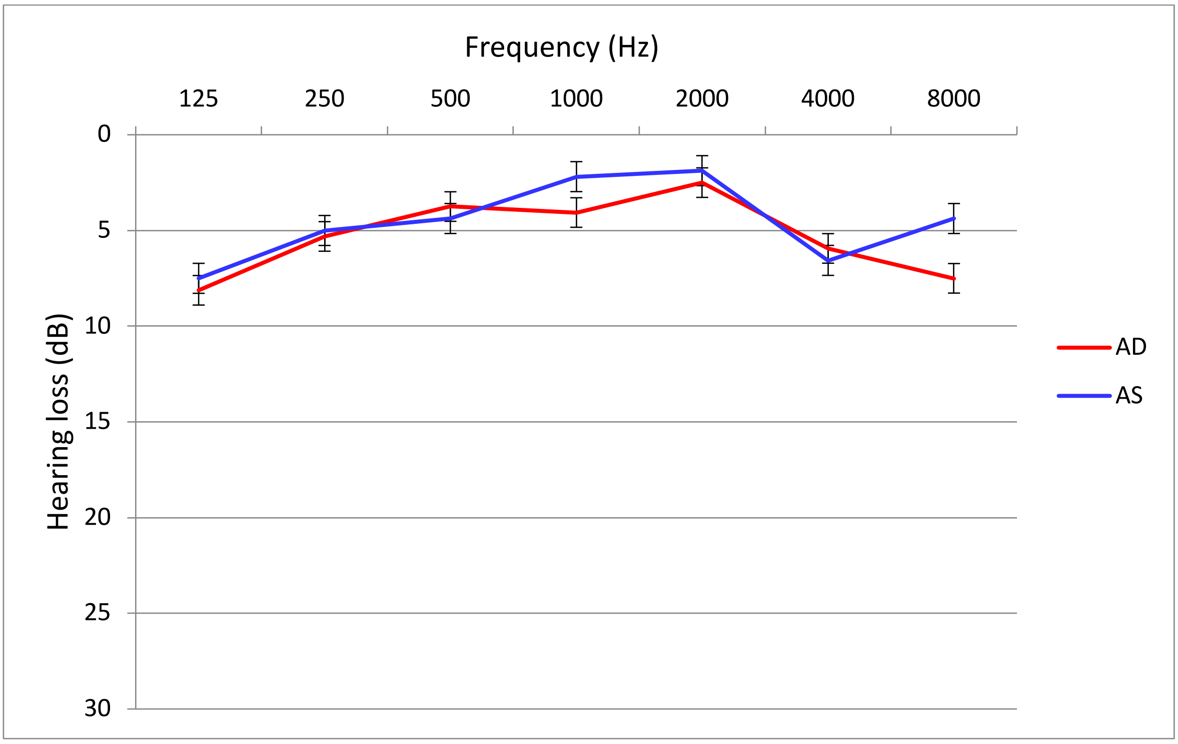


Fig. 5: Average hearing loss (dB) for the right and left ear of the normal-hearing (NH) subjects. Error bars represent standard deviations (SD).

Comparing both ears in NH individuals

Comparison of the right and left ear of NH subjects did not show any statistically significant differences at 1, 2 and 4 kHz. At 0.5 kHz, m_{low} showed a value of 0.259 for the right ear, and 0.242 for the left ear ($p = 0.015$). L_{40} was 98.6 dB SPL for the right ear, and 104.5 dB SPL for the left ear ($p = 0.008$). Lastly, $slope_{40}$ was 0.670 for the right ear, and 0.479 for the left ear ($p = 0.039$). Since the majority of the outcomes did not show any statistically significant differences, and significant differences were small, we decided to merge the results of both ears.

SSD patients before CI versus NH controls

Figure 6 shows loudness function at 0.5, 1, 2 and 4 kHz based on the average values of m_{low}, m_{high}, and L_{cut}. These loudness curves indicate that NH subjects perceived noise at low stimulus levels louder, while, in contrast, SSD patients perceived noise at higher stimulus levels louder. Furthermore, we observed that slope m_{low} was significantly steeper at all frequencies for SSD patients than for the NH subjects ($p < 0.001$) (see Fig. 7 and Table 2). Secondly, there were no significant differences for m_{high} between the two groups (see Fig. 8 and Table 2). Thirdly, L_{cut} was lower for SSD subjects than for NH controls by 2 to 8 dB ($p = 0.060$), most prominently at 0.5 and 1 kHz (see Fig. 9 and Table 2). Furthermore, L_{40} was 1 to 10 dB lower for SSD subjects compared to NH controls, most prominently at

1 kHz (p = 0.146) (see Fig. 10 and Table 2). Lastly, as illustrated in Figs. 11 and 12, we observed no significant differences for $slope_{40}$ and the percentage of high loudness scores (N) between SSD patients and NH controls (see Table 2).

At some frequencies we found that L_{cut} increased with higher PTA thresholds, as shown in Table 3. At 0.5 kHz, a 10 dB increase in PTA threshold corresponded to an approximately 10 dB increase in L_{cut} for both SSD patients (p = 0.052) and NH controls (p = 0.050). We observed the same trend at 2 kHz for the two groups (both presenting with a p-value of 0.012). At 4 kHz, a 10 dB rise in PTA threshold resulted in an elevation of even more than 10 dB in L_{cut}, but only for SSD patients (p = 0.001). Furthermore, a 10-year increase in age was associated with an approximately 5 dB increase in L_{cut} for SSD patients at 0.5 kHz (p = 0.001), 1 kHz (p = 0.018), and 4 kHz (p= 0.009). For L_{40}, only at 4 kHz a 10 dB rise in PTA threshold or a 10-year increase in age resulted in a 5-10 dB elevation of L_{40} for SSD patients (p = 0.003 and p = 0.001, respectively). Lastly, after examining a correlation between age and PTA thresholds, a 10-year increase in age resulted in an elevation of PTA thresholds by 2-3 dB for the SSD patients (p <0.001).

Effect of CI - comparison between SSD subjects

When investigating loudness in the good ear of SSD subjects, CI in the deaf ear did not seem to have a significant effect on our outcome measures, as shown in Table 2.

After CI, we observed increases of L_{cut} with higher PTA thresholds as well (see Table 3). A 10 dB increase in PTA threshold corresponded to an approximately 10 dB increase in L_{cut} at 0.5 kHz (p = 0.021) and 2 kHz (p = 0.001). Before the operation, there was a correlation between L_{cut} and the PTA threshold at 4 kHz, which disappeared post-operatively (p = 0.100). As mentioned earlier, L_{cut} increased with aging for SSD patients pre-operatively at 0.5, 1 and 4 kHz. However, we observed that all correlations vanished after CI. Furthermore, a 10 dB rise in PTA threshold still resulted in an elevation of more than 10 dB in L_{40} at 4 kHz (p = 0.010). Lastly, also at 4 kHz, L_{40} increased by approximately 5 dB with a 10-year increase in age (p = 0.026).

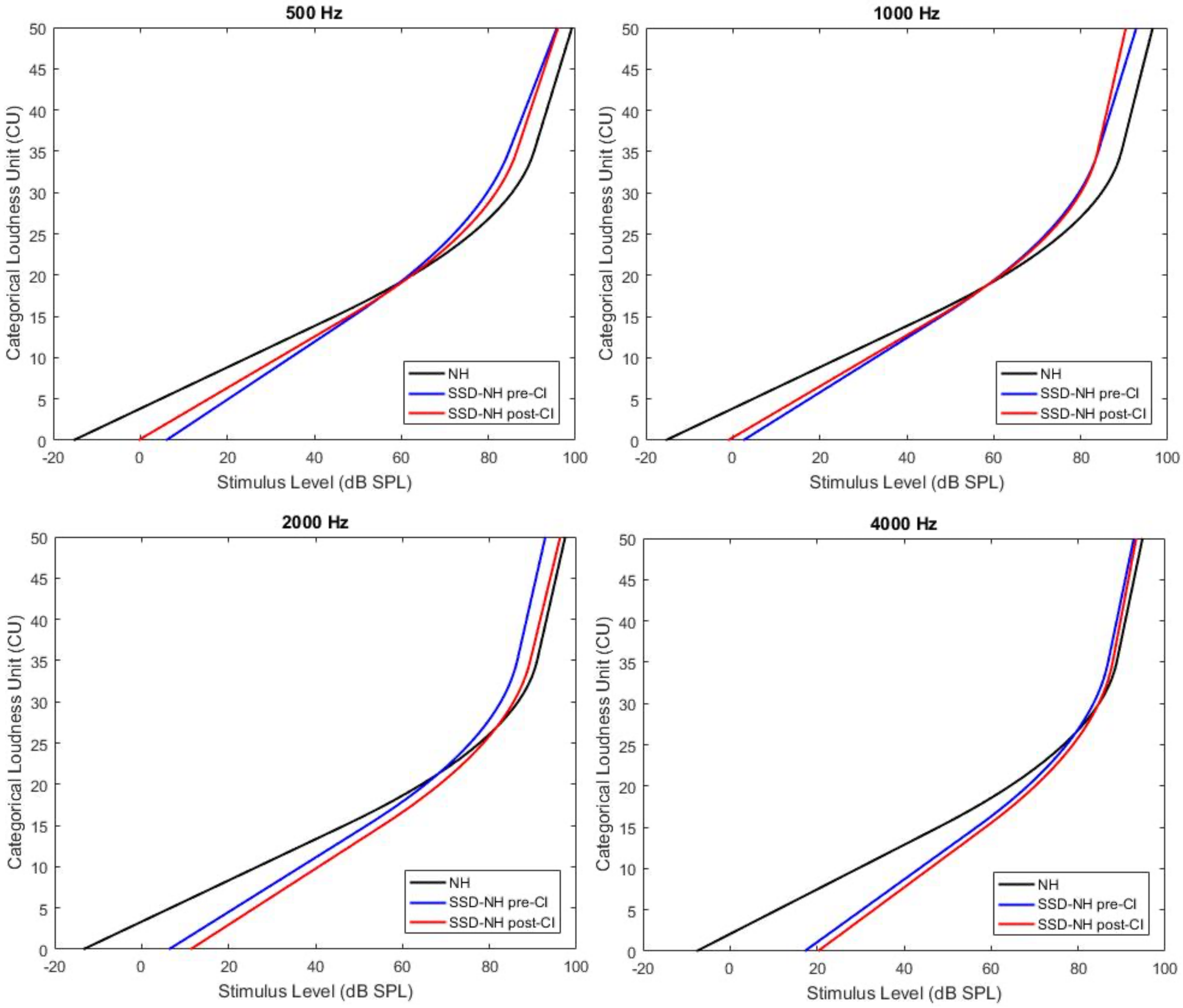


Fig. 6: Averaged loudness curves at 0.5, 1, 2 and 4 kHz based on the average values of m_{low}, m_{high}, and L_{cut} for normal-hearing (NH) subjects and single-sided deaf (SSD) patients, before and after CI.

Table 2: The effects (F with p-values) of single-sided deafness (SSD) compared to NH adults, and the effects of cochlear implantation (CI) within SSD patients on the outcome measures m_{low}, m_{high}, and L_{cut}, L_{40}, $slope_{40}$, and N.

	SSD versus NH		Effect of CI in SSD patients	
Outcomes	F	P value	F	P value
M_{low}	30.128	<0.001	1.050	0.320
M_{high}	0.230	0.635	1.963	0.179
L_{cut}	3.811	0.060	2.277	0.150
L_{40}	2.215	0.146	1.518	0.236
$Slope_{40}$	0.486	0.491	1.038	0.323
N	0.034	0.855	<0.001	0.999

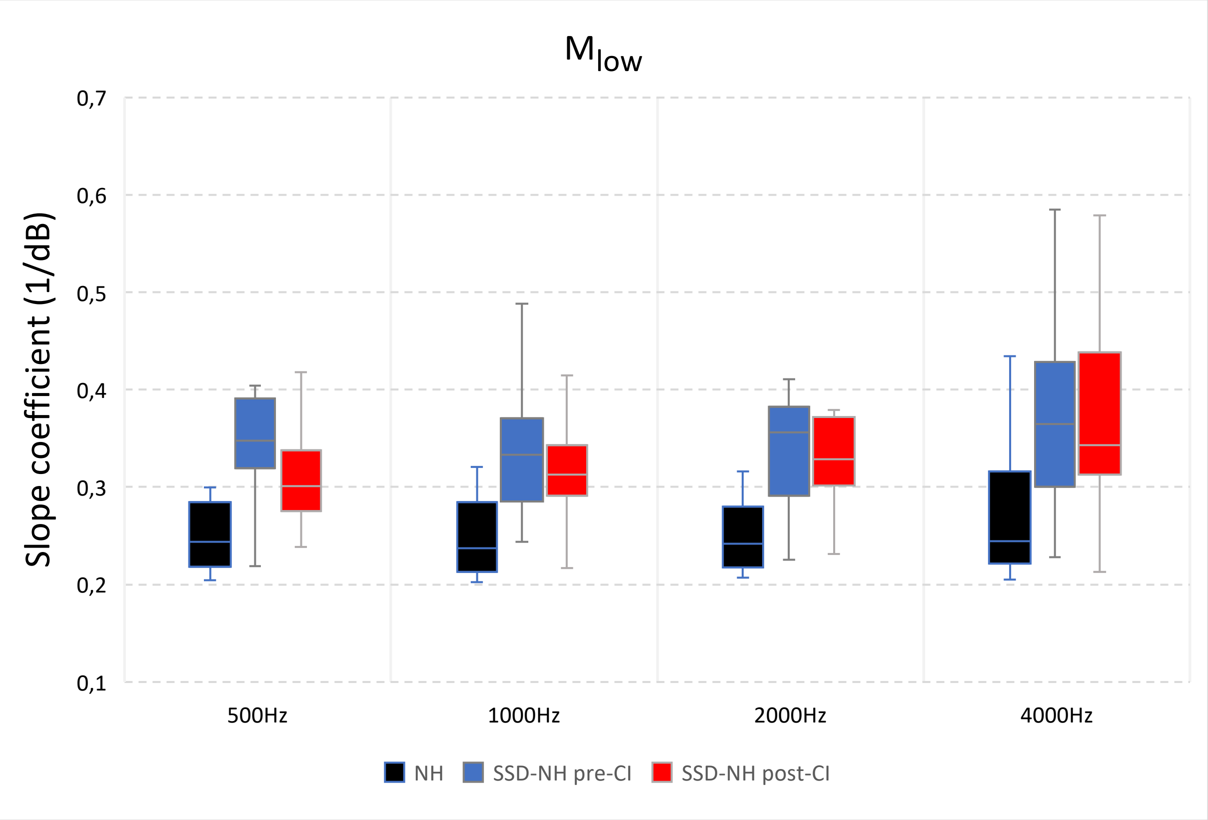


Fig. 7: Averaged m_{low} values at 0.5, 1, 2 and 4 kHz for normal-hearing (NH) subjects and single-sided deaf (SSD) patients, before and after CI.

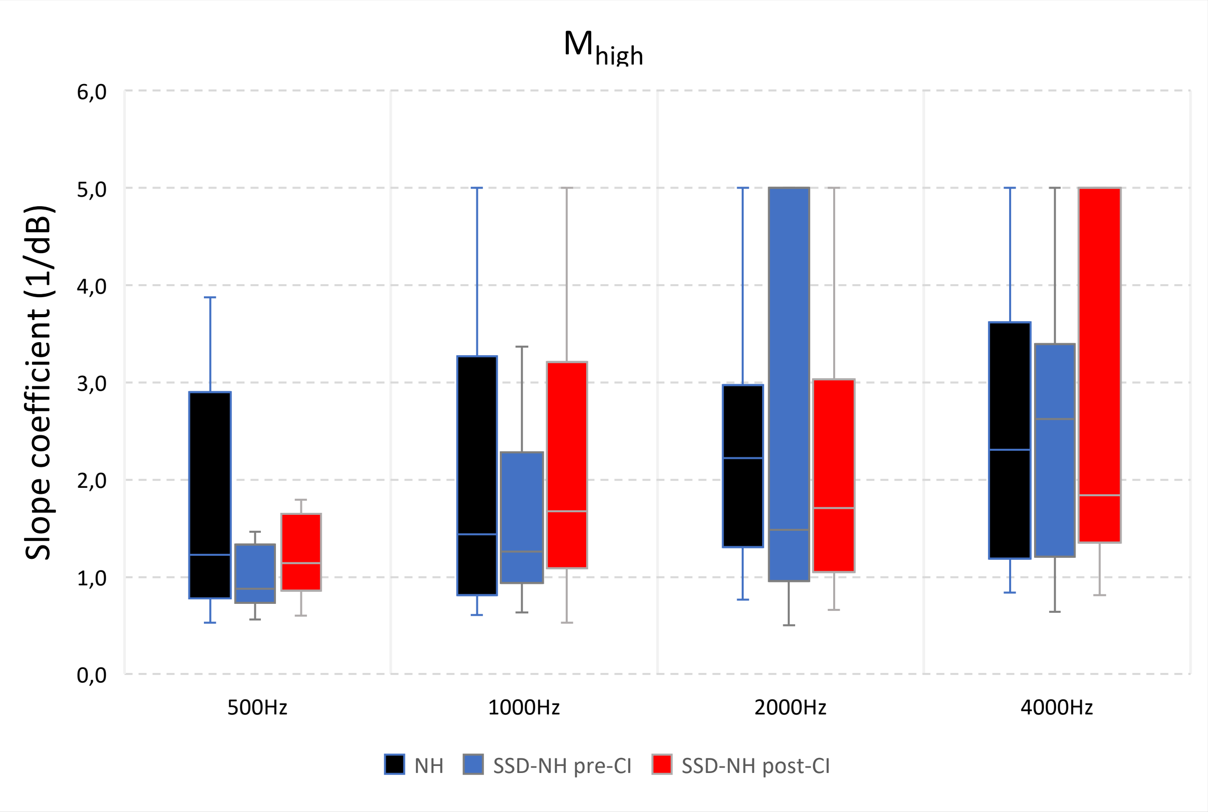


Fig. 8: Averaged m_{high} values at 0.5, 1, 2 and 4 kHz for normal-hearing (NH) subjects and single-sided deaf (SSD) patients, before and after CI.

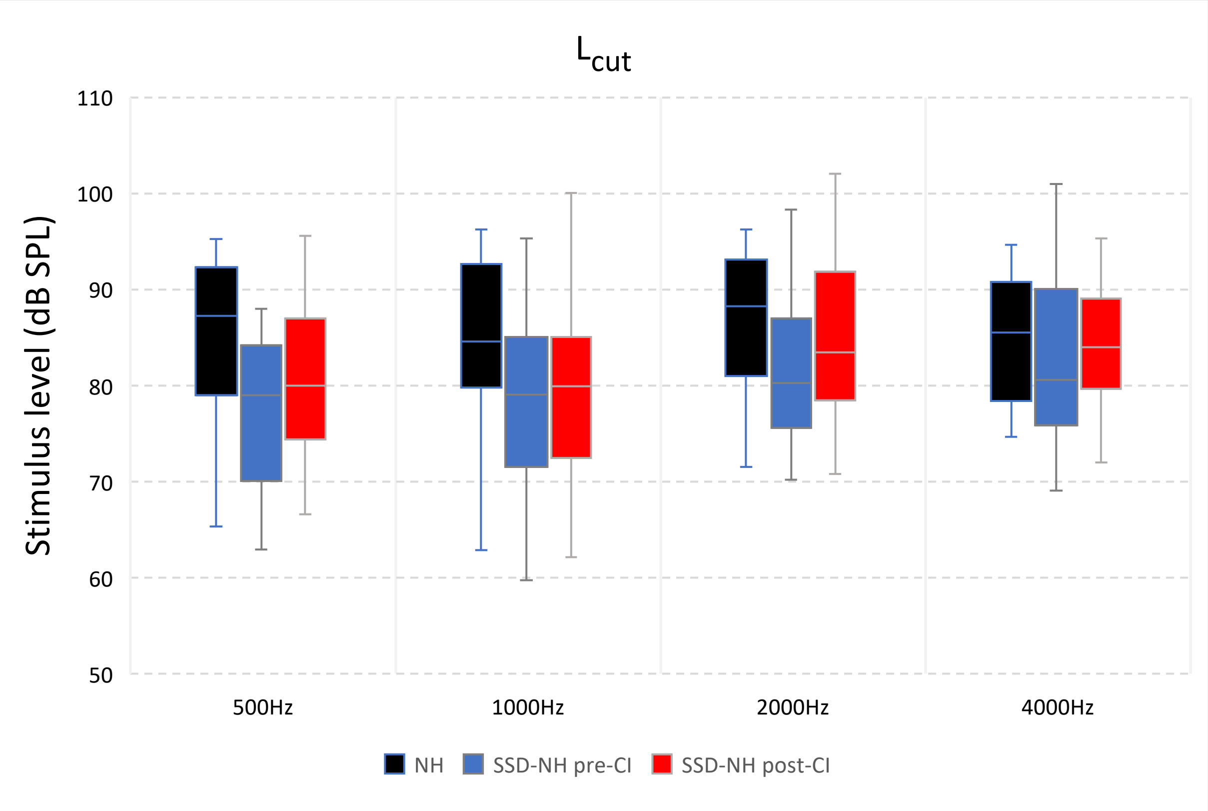


Fig. 9: Averaged L_{cut} values at 0.5, 1, 2 and 4 kHz for normal-hearing (NH) subjects and single-sided deaf (SSD) patients, before and after CI.

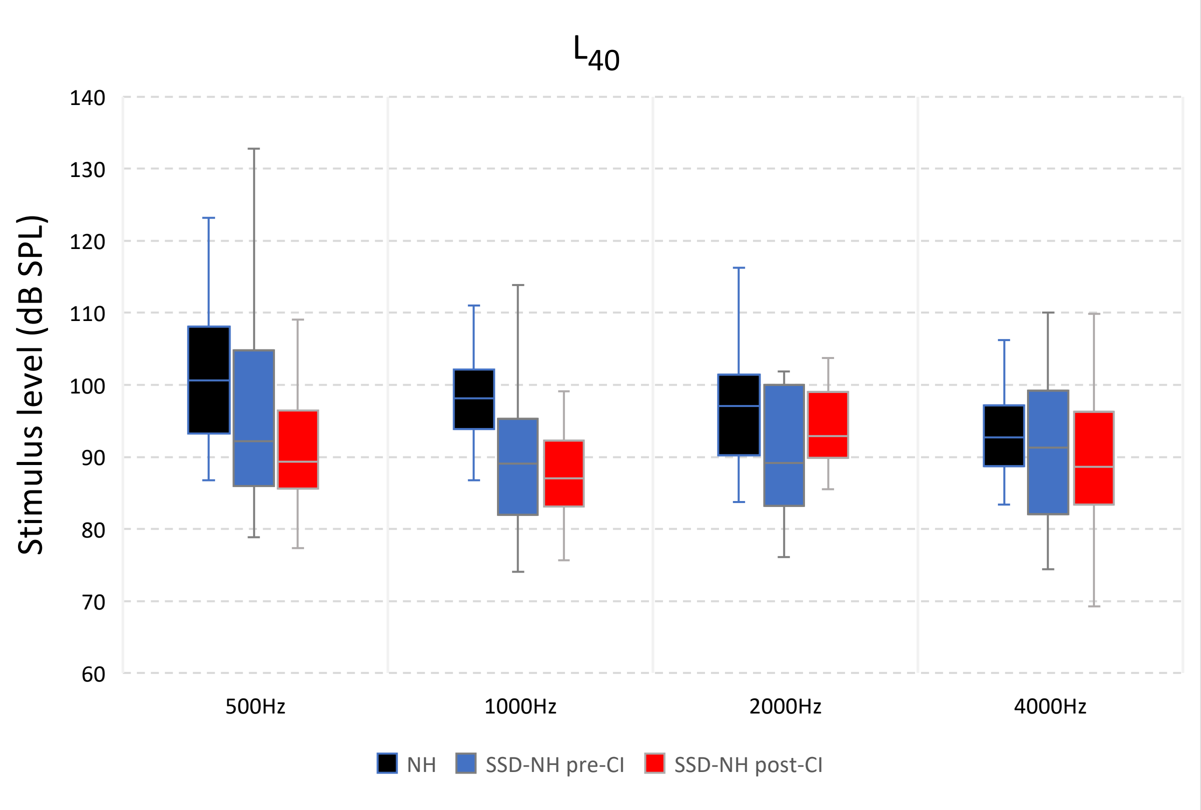


Fig. 10: Averaged L_{40} values at 0.5, 1, 2 and 4 kHz for normal-hearing (NH) subjects and single-sided deaf (SSD) patients, before and after CI.

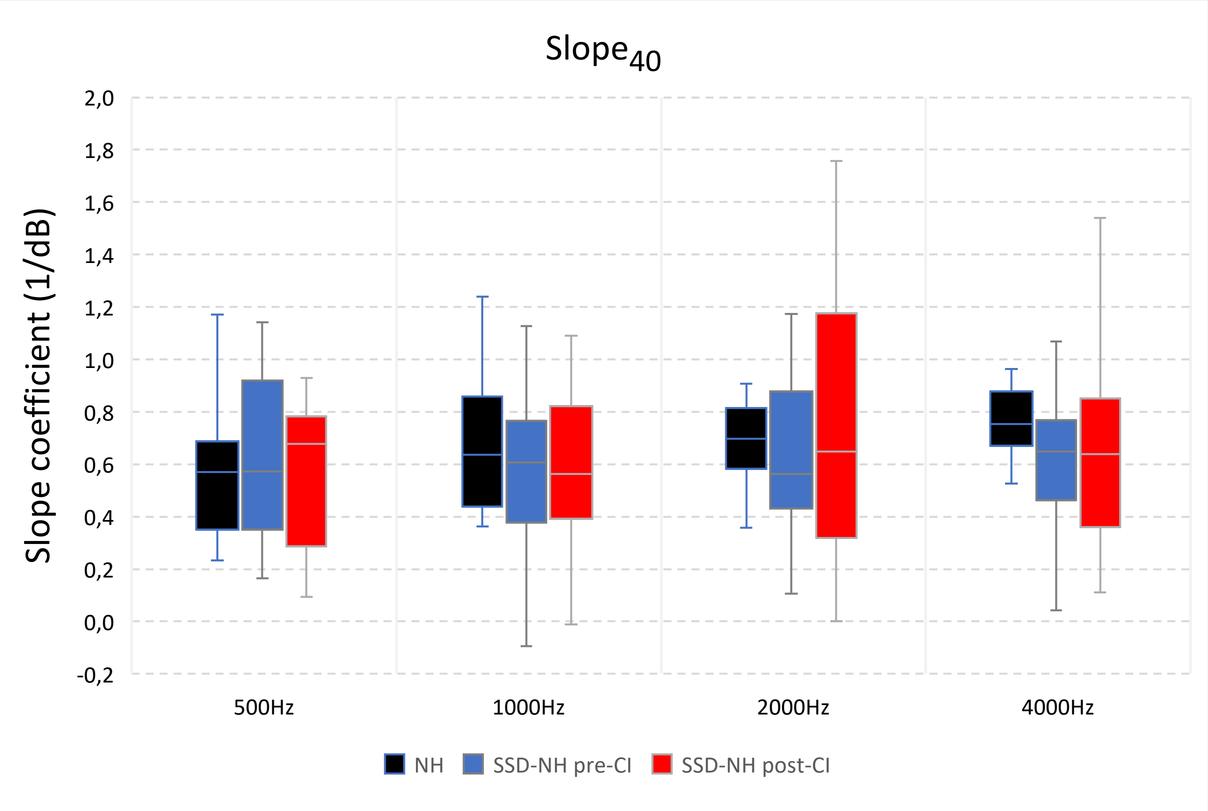


Fig. 11: Averaged slope$_{40}$ values at 0.5, 1, 2 and 4 kHz for normal-hearing (NH) subjects and single-sided deaf (SSD) patients, before and after CI.

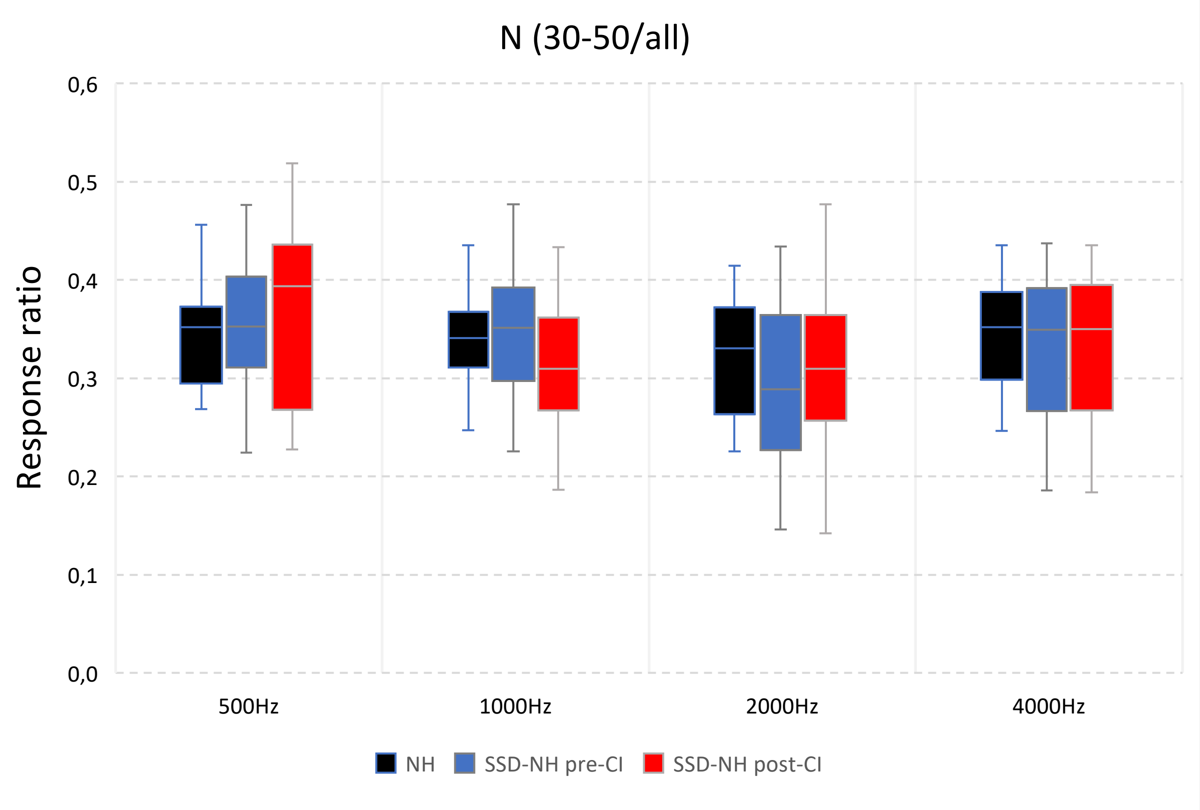


Fig. 12: Percentage of high loudness scores (N) at 0.5, 1, 2 and 4 kHz for normal-hearing (NH) subjects and single-sided deaf (SSD) patients, before and after CI.

Table 3: Correlations (R^2 with p-values) of L_{cut} and L_{40} with Age at inclusion and PTA thresholds at 0.5, 1, 2 and 4 kHz for normal-hearing (NH) subjects and single-sided deaf (SSD) patients, before and after CI.

		NH		SSD-NH pre-CI		SSD-NH post-CI	
Correlation with Age		R^2	P value	R^2	P value	R^2	P value
L_{cut}	0.5 kHz	0.042	0.447	0.516	0.001	0.052	0.363
	1 kHz	0.116	0.196	0.302	0.018	0.001	0.887
	2 kHz	< 0.001	0.943	0.172	0.087	0.091	0.223
	4 kHz	0.161	0.123	0.353	0.009	0.008	0.722
L_{40}	0.5 kHz	< 0.001	0.968	0.119	0.161	0.007	0.736
	1 kHz	< 0.001	0.981	0.006	0.753	0.039	0.430
	2 kHz	0.023	0.577	0.121	0.157	0.011	0.690
	4 kHz	0.195	0.087	0.498	0.001	0.273	0.026
Correlation with PTA		R^2	P value	R^2	P value	R^2	P value
L_{cut}	0.5 kHz	0.248	0.050	0.216	0.052	0.290	0.021
	1 kHz	0.187	0.094	0.081	0.253	0.064	0.313
	2 kHz	0.373	0.012	0.333	0.012	0.487	0.001
	4 kHz	0.175	0.107	0.536	0.001	0.160	0.100
L_{40}	0.5 kHz	0.037	0.477	0.105	0.189	< 0.001	0.935
	1 kHz	< 0.001	0.994	0.096	0.210	0.149	0.113
	2 kHz	0.008	0.737	0.008	0.727	0.146	0.130
	4 kHz	0.006	0.775	0.428	0.003	0.350	0.010

4. Discussion

In the present study, we examined 18 adult subjects with acquired SSD hypothesizing an increase in loudness compared to 16 NH controls. As mentioned earlier, this rise in loudness may be caused by increased excitatory responses in inferior colliculus and auditory cortex in the ipsilateral hemisphere when sounds are presented to the normal ear (Kral, Hubka, et al., 2013; Mossop et al., 2000; Tillein et al., 2016). Additionally, we examined the effect of CI in the deaf ear on loudness in the normal ear of the SSD patients.

The first major finding of this study was that the moderate level of loudness halfway the loudness curve, L_{cut}, appeared to be lower for SSD patients than for NH controls. So, this suggests an increase in loudness in the SSD group, which is in accordance with our hypothesis (see Fig. 8 and Table 2). Secondly, the stimulus level at which participants pressed the 40 CU, expressed as L_{40}, did not show large significant differences in loudness between these two groups (see Fig. 10 and Table 2). However, aging and hearing loss seemed to have impacted our results (see Table 3). To clarify, our SSD group was significantly older than the NH controls, since we did not apply age matching (see Table 1). In addition, they had significantly more hearing loss in their good ear, which can probably be attributed to their age, since the prevalence of hearing loss increases with age (Kim et al., 2019). Therefore, the older age and higher PTA thresholds resulted in a decrease of loudness, while SSD on the other hand resulted in an increase of loudness (see Figs. 9 and 10, and Table 2). So, these opposite effects of aging and hearing loss, which were not controlled for in this study, could have made our results less notable. Lastly, regarding loudness in the good ear of SSD patients, we did not observe a significant change in loudness after CI of the deaf ear. This lack of effect may be related to the relatively short duration of CI use. Interestingly however, the increase of L_{cut} and L_{40} with aging and hearing loss appeared to diminish after CI. We cannot provide an explanation for this, as the literature on loudness and SSD is generally scarce.

As displayed in Fig. 6, we generally observed that loudness function slopes were well in line with other studies, since most studies showed a shallow slope for low stimulus levels and an increasing slope for high stimulus levels (Ewert & Oetting, 2018; Oetting et al., 2016). This may be explained by the fact that differences in sound intensity are reflected in the extent of stereocilia movement and subsequent depolarization of hair cells. Higher intensities cause stronger depolarizations and a higher probability of action potential formation in multiple neurons, leading to stronger activation of the cochlea (Kapteyn & Lamoré, 2009). Furthermore, the median duration of deafness was 1.3 years, which is considerably short (see Table 1). However, even in this short period of time there may be a possible effect on loudness in the intact ear, caused by short-term reorganization of excitation in the inferior colliculus ipsilateral to the intact ear (Moore et al., 1997).

Methodological considerations and future research

One of the limitations of CLS is the highly subjective nature of the test, because participants might be subject of the “sequence effect”. If, for example, a series of relatively soft sounds are presented, participants may scale relatively high due to the tendency to use the entire scale per measurement. After a few soft sounds, a loud stimulus will be scaled relatively loud (and vice versa). However, these are well-known disadvantages of the procedure. To enhance the test’s reproducibility participants underwent a practice test to become familiar with the procedure, as mentioned earlier. Additionally, we implemented a standardize protocol with consistent patient instructions (see Appendix 1).

Further research is necessary to investigate loudness in the intact ear of SSD patients, preferably involving a larger study cohort. Additionally, an older NH control group is acquired to understand the effect of age and PTA thresholds on loudness. Lastly, it would be of high interest to understand the underlying mechanisms behind the increase of loudness and to explore the potential of CI in modulating loudness.

Conclusion

Our data confirmed the hypothesis of an increase in loudness, although age and hearing loss counteracted this effect slightly. Nevertheless, we assume that the increase in loudness is caused by an increase in excitatory neural responses in the ipsilateral hemisphere, which in its turn is caused by a reduction in inhibition from the contralateral ear.

References

Arts, R. A. G. J., George, E. L. J., Stokroos, R. J., & Vermeire, K. (2012). Review: cochlear implants as a treatment of tinnitus in single-sided deafness. *Current Opinion in Otolaryngology & Head and Neck Surgery*, *20*(5), 398–403. https://doi.org/10.1097/MOO.0b013e3283577b66

Avan, P., Giraudet, F., & Büki, B. (2015). Importance of Binaural Hearing. *Audiology and Neurotology*, *20*(Suppl. 1), 3–6. https://doi.org/10.1159/000380741

Baguley, D. M., Bird, J., Humphriss, R. L., & Prevost, A. T. (2006). The evidence base for the application of contralateral bone anchored hearing aids in acquired unilateral sensorineural hearing loss in adults. In *Clinical Otolaryngology* (Vol. 31, Issue 1, pp. 6–14). https://doi.org/10.1111/j.1749-4486.2006.01137.x

Bernstein, J. G. W., Schuchman, G. I., & Rivera, A. L. (2017). Head shadow and binaural squelch for unilaterally deaf cochlear implantees. *Otology and Neurotology*, *38*(7), e195–e202. https://doi.org/10.1097/MAO.0000000000001469

Brand, T., & Hohmann, V. (2002). An adaptive procedure for categorical loudness scaling. *The Journal of the Acoustical Society of America*, *112*(4), 1597–1604. https://doi.org/10.1121/1.1502902

Brown, K. D., Balkany, T. J., & Williams, L. (2007). Benefits of bilateral cochlear implantation: a review. In *Curr Opin Otolaryngol Head Neck Surg* (Vol. 15).

Dreschler & Lamoré. (2007a). 2.3.1.1(2). Inleiding. In *Audiologieboek*.

Dreschler & Lamoré. (2007b). 2.3.1.8(2). Methoden om luidheid te meten. In *Audiologieboek*.

Ewert, S. D., & Oetting, D. (2018). Loudness summation of equal loud narrowband signals in normal-hearing and hearing-impaired listeners. *International Journal of Audiology*, *57*, S71–S80. https://doi.org/10.1080/14992027.2017.1380848

Galvin, J. J., Fu, Q.-J., Wilkinson, E. P., Mills, D., Hagan, S. C., Lupo, J. E., Padilla, M., & Shannon, R. V. (2019). Benefits of Cochlear Implantation for Single-Sided Deafness: Data From the House Clinic-University of Southern California-University of California, Los Angeles Clinical Trial. *Ear and Hearing*, *40*(4), 766–781. https://doi.org/10.1097/AUD.0000000000000671

Kapteyn & Lamoré. (2009). 6.2.2.2(2). Niveau 0 – De haarcellen en de gehoorzenuw. In *Audiologieboek*.

Kim, H., Ha, J., & Park, H. Y. (2019). Pure Tone Audiometry Threshold Changes for 10 Years in the Same Individuals of General Population: A Retrospective Cohort Study. *Annals of Otology, Rhinology and Laryngology*, *128*(5), 433–440. https://doi.org/10.1177/0003489419826142

Kurz, A., Grubenbecher, M., Rak, K., Hagen, R., & Kühn, H. (2019). The impact of etiology and duration of deafness on speech perception outcomes in SSD patients. *European Archives of Oto-Rhino-Laryngology*, *276*(12), 3317–3325. https://doi.org/10.1007/s00405-019-05644-w

Moore, D. R., France, S. 1, Mcalpine, D., Mossop, J. E., & Versnel, H. (1997). *Plasticity of Inferior Colliculus and Auditory Cortex Following Unilateral Deafening in Adult Ferrets*.

Oetting, D., Brand, T., & Ewert, S. D. (2014). Optimized loudness-function estimation for categorical loudness scaling data. *Hearing Research*, *316*, 16–27. https://doi.org/10.1016/j.heares.2014.07.003

Oetting, D., Hohmann, V., Appell, J. E., Kollmeier, B., & Ewert, S. D. (2016). Spectral and binaural loudness summation for hearing-impaired listeners. *Hearing Research*, *335*, 179–192. https://doi.org/10.1016/j.heares.2016.03.010

Peters, J. P. M., van Heteren, J. A. A., Wendrich, A. W., van Zanten, G. A., Grolman, W., Stokroos, R. J., & Smit, A. L. (2021). Short-term outcomes of cochlear implantation for single-sided deafness compared to bone conduction devices and contralateral routing of sound hearing aids-Results of a Randomised controlled trial (CINGLE-trial). *PloS One*, *16*(10), e0257447. https://doi.org/10.1371/journal.pone.0257447

Usami, S. I., Kitoh, R., Moteki, H., Nishio, S. Y., Kitano, T., Kobayashi, M., Shinagawa, J., Yokota, Y., Sugiyama, K., & Watanabe, K. (2017). Etiology of single-sided deafness and asymmetrical hearing loss. *Acta Oto-Laryngologica*, *137*, S2–S7. https://doi.org/10.1080/00016489.2017.1300321

Van de Heyning, P., Vermeire, K., Diebl, M., Nopp, P., Anderson, I., & De Ridder, D. (2008). Incapacitating Unilateral Tinnitus in Single-Sided Deafness Treated by Cochlear Implantation. In *Rhinology & Laryngology* (Vol. 117, Issue 9).

Van Wanrooij, M. M., & Van Opstal, A. J. (2004). Contribution of head shadow and pinna cues to chronic monaural sound localization. *The Journal of Neuroscience : The Official Journal of the Society for Neuroscience*, *24*(17), 4163–4171. https://doi.org/10.1523/JNEUROSCI.0048-04.2004

Zeitler, D. M., & Dorman, M. F. (2019). Cochlear Implantation for Single-Sided Deafness: A New Treatment Paradigm. *Journal of Neurological Surgery. Part B, Skull Base*, *80*(2), 178–186. https://doi.org/10.1055/s-0038-1677482

Appendices

Appendix 1

1. Retrieve the laptop (MSI MS-175 A) from the room adjacent to 'hearing research room 2', inside the cabinet (key is on the shelf next to the cabinet).
2. Power on the computer using the username (KNOResearch3) and password (Onderzoek3).
3. Connect the computer to the charger and the audio interface (USB should be inserted into the port furthest away from the HDMI input). Then, connect the audio interface to the audio cable (located next to the green wire in 'decos audiology').
4. Take the monitor, place it on the speaker, and plug in the power cord. Next, connect the laptop to the monitor using the blue DVI cable. Lastly, turn on the speaker.
5. Position the chair at 1m distance, connect a mouse to the laptop, and provide something for participants to move their mouse on.
6. Set the volume to 100%.
   Configure the speaker properties in 'advanced' settings to 24-bits 44100 Hz.
   Use the U24XL sound card.
7. Open Matlab R2016b and open the file 'exampleACALOS_cfg.m'.
8. On lines 52 and 53, make the appropriate choice. One of the two options should be marked in black, and the other in green. Any line with a '%' symbol will not be executed.
9. For the pre-test, ensure that line 52 is marked black and line 53 is marked green. In this case, measurement will be done at 1000 Hz. Press SAVE.
10. If everything is set correctly, type in or under the 'Command Window':
    afc('main','exampleACALOS','LS_Name_Date_Test','nbb') and then press ENTER.
11. The program will take some time to start, afterwards orange text will appear in the Command Window.
12. Once the program is launched, maximize the screen and press a button to begin the experiment. After completion, you can close the window, and the 'Workspace' will contain a variable named 'work' if everything went well.
13. Now we will start the ‘real’ experiment, make line 53 black and line 52 green by removing the '%' symbol from one line and adding it to the other. This will measure four different frequencies: def.exppar1 = [500 1000 2000 4000]'.
14. Press Save (top-left corner)!!!!

For NH participants:

1. Have the participant insert an earplug into the left ear and wear an earmuff on the same ear.
2. Type in or under the 'Command Window':
   afc('main','exampleACALOS','LS_Name_Date_AD','nbb') and then press ENTER.

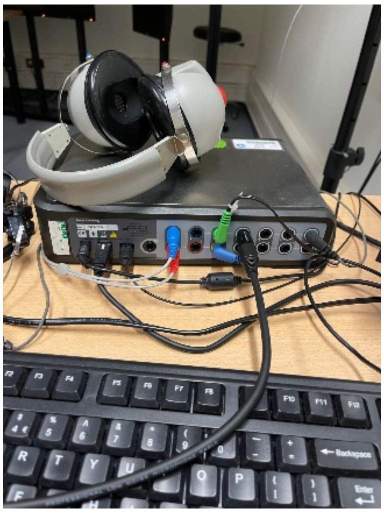
3. Perform the measurement and check if there is a variable 'work' in the workspace.
4. Have the participant do the same as step 1, but this time on the right ear.
5. Type in or under the 'Command Window': afc('main','exampleACALOS','LS_Name_Date_AS','nbb') and then press ENTER.
6. Perform the measurement and check if there is a variable 'work' in the workspace.
7. Have the participant remove the earplug and take off the earmuff.
8. To check the data, type in the Command Window: plotACALOS(work,1). Note that the numbers 1, 2, 3, or 4 represent 500, 1000, 2000, and 4000 Hz, respectively. After plotting a frequency, type 'close' to proceed to the next one, generating separate plots for each frequency.
9. Verify if the data is saved in: /experiments/sessions.

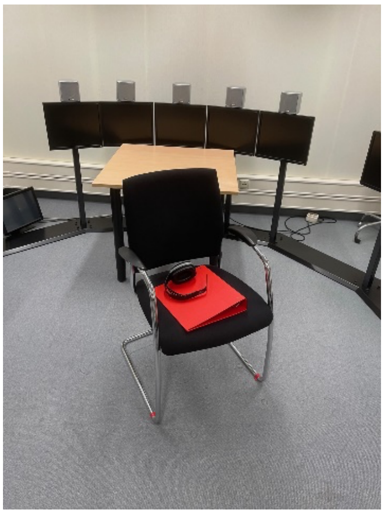
For SSD-NH pre-CI patients:

1. Have the participant insert an earplug into the deaf ear and wear an earmuff on the same ear.
2. Type in or under the 'Command Window': afc('main','exampleACALOS','LS_Sub_pre_NH','nbb') and then press ENTER.
3. Perform the measurement and check if there is a variable 'work' in the workspace.
4. Have the participant remove the earplug and take off the earmuff.
5. To check the data, type in the Command Window: plotACALOS(work,1). Note that the numbers 1, 2, 3, or 4 represent 500, 1000, 2000, and 4000 Hz, respectively. After plotting a frequency, type 'close' to proceed to the next one, generating separate plots for each frequency.
6. Verify if the data is saved in: /experiments/sessions.

For SSD-NH post-CI patients:

7. Have the patient remove the CI. Provide a plug and an earmuff on the side of the CI ear (even if it seems 'useless').
8. Type in or under the 'Command Window': afc('main','exampleACALOS','LS_Sub_post_NH','nbb') and then press ENTER.
9. Perform the measurement and check if there is a variable 'work' in the workspace.
10. Have the participant remove the earplug and take off the earmuff.
11. To check the data, type in the Command Window: plotACALOS(work,1). Note that the numbers 1, 2, 3, or 4 represent 500, 1000, 2000, and 4000 Hz, respectively. After plotting a frequency, type 'close' to proceed to the next one, generating separate plots for each frequency.
12. Verify if the data is saved in: /experiments/sessions.






OUTPUT
INPUT