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Original article

Cortical auditory responses according to hearing rehabilitation in unilateral hearing loss

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ABSTRACT

Objective: To assess the impact of rehabilitation systems (CROS: Contralateral Routing of Signal; BAHA: Bone-Anchored Hearing Aid; CI: cochlear implant) on cortical auditory evoked potentials (CAEP) and auditory performance in unilateral hearing loss.
Subjects and method: Twenty-one adults with unilateral hearing loss, using CROS ($n=6$), BAHA ($n=6$) or CI ($n=9$), were included. Seven normal-hearing subjects served as controls. CAEPs were recorded for a (/ba/) speech stimulus; for patients, tests were conducted with and without their auditory rehabilitation. Amplitude and latency of the various CAEP components of the global field power (GFP) were measured, and scalp potential fields were mapped. Behavioral assessment used sentence recognition in noise, with and without spatial cues.
Results: Only CI induced N1 peak amplitude change ($P<0.05$). CI and CROS increased polarity inversion amplitude in the contralateral ear, and frontocentral negativity on the scalp potential map. CI improved understanding when speech was presented to the implanted ear and noise to the healthy ear, and vice-versa.
Conclusion: Cochlear implantation had the greatest impact on CAEP morphology and auditory performance. A longitudinal study could analyze progression of cortical reorganization.

1. Introduction

Unilateral hearing loss impacts speech comprehension in noise and acoustic location, with consequences for overall quality of life [1–5]. Auditory rehabilitation can use the CROS (contralateral routing of the signal) system, which transfers input to the impaired ear to the healthy ear by Wi-Fi, or the BAHA (bone-anchored hearing aid) system, which transfers the information by bone conduction [6]. Neither method rehabilitates binaural hearing, as the brain receives and processes input from one side only. The only system that actually rehabilitates binaural hearing as such is cochlear implantation (CI). Currently in France, this is reserved for severe to profound bilateral hearing loss with <50% speech comprehension at 60 dB using hearing aids [7]. CI in unilateral hearing loss is allowed only for research purposes. CI obviates the outer and middle ear, directly stimulating the auditory nerve fibers. Studies demonstrated benefit in this indication for acoustic location, speech comprehension and quality of life [8,9]. No studies have analyzed the impact of a given type of rehabilitation of unilateral hearing loss on the auditory cortex. Cortical auditory responses in unilateral hearing loss patients with auditory rehabilitation can be studied by means of cortical auditory evoked potentials (CAEP). These are the sum of the electrical potentials of large fields of activated neurons; following auditory stimulation, adult CAEPs recorded at the vertex show 4 main components, differing in amplitude and in latency: 2 positive waves, P1 (50ms) and P2 (200ms), and 2 negative waves, N1 (100ms) and N2 (250ms) [10–12]. The most frequently studied wave in adults is N1, the generators of which lie in the supratemporal auditory cortex, as polarity inversion is observed at the Sylvian fissure [13–15].

Study of cortical auditory responses in these patients can highlight differences in cortical processing of auditory information according to the type of auditory rehabilitation, and can identify neurophysiological indices such as P1 wave latency, known to track auditory cortex maturation in children with CIs [16,17].

The aim of the present electrophysiological study was to assess cortical auditory responses and speech comprehension in adults

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with unilateral hearing loss according to their type of rehabilitation: CI, CROS or BAHA.

2. Subjects and method

2.1. Subjects

The inclusion criterion for the "healthy" ear was a mean pure-tone threshold Pure Tone Average (PTA) of ≤ 25 dB HL at 0.5, 1, 2 and 3 kHz, in line with the AMA (American Medical Association) guide to Evaluation of Permanent Impairment [18], and as in previous studies [19,20]. Twenty-one right-handed adults with unilateral profound sensorineural hearing loss (PTA = 95 ± 18 dB) and a normal-hearing contralateral ear (PTA ≤ 25 dB HL) were tested. Nine had a CI (5 female; 4 with right-side impairment; mean age, 60 ± 7 years), 6 had a BAHA (5 female; 4 with right-side impairment; mean age, 61 ± 17 years), and 6 had a CROS system (3 female; 3 with right-side impairment; mean age, 66 ± 11 years), all with at least 1 year's rehabilitation. The Cochlear® processors used the ACE strategy and adaptive directivity mode; the Digisonic® processors used the Crystalis XDP strategy and omnidirectional directivity. The University Hospital review board approved the study protocol (N°ID RCB N° 2015-A01249-40); all subjects provided written consent. Table 1 presents demographic data.

Seven normal-hearing adults (PTA ≤ 25 dB HL; 5 female; mean age, 53 ± 2.4 years) served as controls. Kruskal-Wallis analysis showed no significant differences between unilateral hearing-loss patients and controls for age ($P = 0.2$, K = 5.1) or gender ($P = 0.6$, K = 1.9), and no differences between the 3 patient groups for duration of hearing loss ($P = 0.2$, K = 3) or PTA in the normal ($P = 0.5$, K = 1.2) or the impaired ear ($P = 1$, K = 0.06).

2.2. Procedure

Patients were tested in random order with and without rehabilitation devices. Audiometry and CAEP were assessed in the same session.

A-weighted decibels (dBa) were used, to approximate the frequency sensitivity of the human ear [21], and because they are widely used in other studies [20,22–24].

2.2.1. Speech audiometry

Sentence recognition in noise was assessed on an adaptive procedure, using French sentences from the Marginal Benefit from Acoustic Amplification (MBAA) corpus, comprising 33 lists of 15 sentences each [25]. For each test condition, lists were selected randomly and delivered in free field. The noise was continuous white noise, calibrated in dBa. Speech stimuli were presented at 65 dBa, with noise adjusted by 5 dB increments according to response quality: the signal-to-noise ratio (SNR) was diminished by 5 dB when the subject repeated the whole sentence correctly, or else increased by 5 dB. The intelligibility threshold was calculated as the SNR associated with 50% comprehension. Three spatial conditions were tested: 1) speech to the normal-hearing ear (NE), noise to the impaired ear with CI/CROS/BAHA ($S_{NE}N_{CI/CROS/BAHA}$); 2) speech and

Table 1
Demographic data for unilateral hearing-impaired subjects equipped with CI (S1–S9), CROS (S10–S15) or BAHA (S16–S21) and controls (S22–S28).

Subject	Gender	Age (years)	Duration (years)	Etiology	PTA non-impaired ear (dB HL)	PTA impaired ear (dB HL)	Impaired side	Type of rehabilitation
S1	M	53	1.5	Unknown	20	120	G	CI (CI52 ^{C})
S2	F	60	2	Sudden	14	90	D	CI (CI51 ^{C})
S3	F	66	5.5	Sudden	5	78	G	CI (CI51 ^{C})
S4	F	65	30	Sudden	20	73	G	CI (CI51 ^{C})
S5	M	48	2	Sudden	18	120	G	CI (Digisonic SP ^{N})
S6	M	65	2	Sudden	20	116	D	CI (Digisonic SP ^{N})
S7	F	54	3	Sudden	20	68	D	CI (CI51 ^{C})
S8	F	65	20	Unknown	20	120	G	CI (Digisonic SP ^{N})
S9	M	65	10	Menière	22	79	D	CI (CI52 ^{C})
Moyenne, std		60.0 ± 7	9.0 ± 11		17.7 ± 5	96 ± 22.6		
Médiane		65	3		20	90		
S10	F	67	7	Sudden	11	76	D	BAHA (5 Cochlear)
S11	F	72	44	Cholesteatoma	20	72.5	G	BAHA (5 Cochlear)
S12	M	57	2	Postoperative	16	120	D	BAHA (5 Cochlear)
S13	F	52	40	Unknown	19	84.4	D	BAHA (PONTO Oticon)
S14	F	83	14	Otitis sequelae	25	98.8	D	BAHA (PONTO Oticon)
S15	F	34	10	Unknown	23	115	D	BAHA (BP110 Cochlear)
Mean ± SD		60.8±17.12	19.5±17.9		18.9±4.9	94.5±20		
Median		62	12		19	92		
S16	M	75	20	Sudden	25	83.8	D	CROS (Phonak)
S17	F	61	6	Sudden	15	97.5	D	CROS (Phonak)
S18	M	62	2	Menière	25	90.6	G	CROS (Phonak)
S19	M	76	20	Unknown	24	103.8	G	CROS (Phonak)
S20	F	47	7	Sudden	14	75	D	CROS (Phonak)
S21	F	72	25	Sudden	20	103.8	G	CROS (Phonak)
Mean ± SD		65.5 ± 11.1	13.3 ± 9.4		20.5 ± 4.9	92.4 ± 11.5		
Median		67	13.5		22	94		
S22	M	61			16	16		Control
S23	F	50			20	20		Control
S24	F	51			6	5		Control
S25	F	56			18	16		Control
S26	M	53			14	13		Control
S27	M	55			8	6		Control
S28	F	51			20	19		Control
Mean ± SD		53.9 ± 3.8			14.6 ± 5.4	13.6 ± 5.9		
Median		53			16	16		

PTA: pure tone average at 0.5, 1.0, 2.0 and 3.0 kHz ^{C}: Cochlear device; ^{N}:Neurelec device, CI: cochlear implant; BAHA: Bone-Anchored Hearing Aid; CROS: Contralateral Routing of Signal.

noise via a single loudspeaker (S0N0); and speech to the impaired ear with CI/CROS/BAHA and noise to NE ($S_{CI/CROS/BAHA}N_{NE}$).

2.2.2. Cortical auditory evoked potentials

2.2.2.1. Stimuli. The speech stimulus was a 125 ms/ba/delivered at 70 dBa via 2 loudspeakers situated 1.3 m from the subject at −45° and +45°. One thousand one hundred and fifty stimuli were presented with a 700 ms interval. Neurophysiological recording took about 20 minutes per subject.

2.2.2.2. Electroencephalography (EEG). Subjects were seated comfortably in an armchair in a soundproof room with low lighting, and watched a silent movie during EEG recording.

Recording used the Neuroscan system (Synamps RT amplifier, Curry 7 and Neuroscan Stim^2 software) with 64 electrodes referenced on line to the nose according to the international 10–20 system. Impedance was < 5 kΩ. Sampling frequency was 500 Hz with 0–200 Hz filtering.

EEG data were analyzed on Matlab software (Mathworks, Natick, MA) via the EEGLAB interface [26]. Filtering used a 0.3–70 Hz band-pass. Recordings made during movement by the subject were identified manually for rejection; mean artifact rejection rate was < 25% per subject per session. Supplementary artifacts due to eye movement or to the rehabilitation device were rejected by independent component analysis (ICA) on EEGLAB [27]. ICA components implicating the device were identified as being ipsilateral to the device (temporoparietal), synchronous with the auditory stimulation, and large amplitude; they were found in all CI users and 2 BAHA users. This methodology has been validated in many studies [28–31]. The EEG recording was then segmented by epochs from −100 to 500 ms relative to stimulus onset. The epochs were corrected according to baseline in a 100 ms window before stimulus onset, with a low band-pass filter of 30 Hz.

Cortical auditory responses were assessed in terms of global field power (GFP): the standard deviation over the electrodes over time, quantifying the simultaneous activity of all electrodes [32]. GFP contains 3 peaks (P1, N1 and P2), identified visually.

Responses in subjects with right-sided impairment were reversed with respect to the midline so as to average responses between right- and left-sided impairment, as in previous studies [33]. Thus, in the scalp potential field maps, left-side recordings are those contralateral to the rehabilitated ear and right-side recordings are ipsilateral.

2.3. Statistical analysis

As distributions were not always normal, non-parametric tests were used. The Wilcoxon test was used to analyze CI, CROS and BAHA efficacy, comparing results with and without the system.

Improvement in speech audiometry ("with" minus "without") was compared on non-parametric Kruskal–Wallis ANOVA (analysis of variance) with post-hoc Dunns test.

The non-parametric Mann–Whitney test was used to compare patients versus controls.

Results are presented as box-plots showing the 25th, 50th and 75th percentiles, with error bars indicating the 5th and 95th percentiles.

3. Results

3.1. Auditory performance

Speech intelligibility thresholds in the 3 acoustic configurations are shown in Fig. 1.

The Wilcoxon test showed significant improvement in threshold with rehabilitation only for CI ($n = 9$), in conditions $S_{NE}N_{IC}$ ($P = 0.02$, $W = 33$) and $S_{IC}N_{NE}$ ($P = 0.04$, $W = 31$ (Fig. 1; Table 2).

The Kruskal–Wallis ANOVA showed differential performance according to system (BAHA, $n = 6$; CROS, $n = 6$; CI, $n = 9$) in condition $S_{NE}N_{IC/CROS/BAHA}$ ($P = 0.031$, $K = 6.95$), but the post-hoc Dunn's test found no superiority for any one system ($P > 0.05$; Table 2).

In condition S0N0, the Mann–Whitney test found better performance in controls ($n = 7$) than in patients with BAHA ($n = 6$; "with": $p = 0.01$; "without": $P = 0.01$) or CROS ($n = 6$; "with": $P = 0.01$; "without": $P = 0.04$), but no difference with respect to the CI group ($n = 9$; $P > 0.05$).

In condition $S_{IC/CROS/BAHA}N_{NE}$, controls ($n = 7$) showed better results than patients overall, with or without their devices: CI ($n = 9$): "without": $P = 0.005$, "with": $P = 0.02$; CROS ($n = 6$): "without": $P = 0.02$, "with": $P = 0.04$; BAHA ($n = 6$): "without": $P = 0.007$, "with": $P = 0.04$.

In condition $S_{NE}N_{CI/CROS/BAHA}$, there were no significant differences between controls and patients, with or without devices ($P > 0.05$).

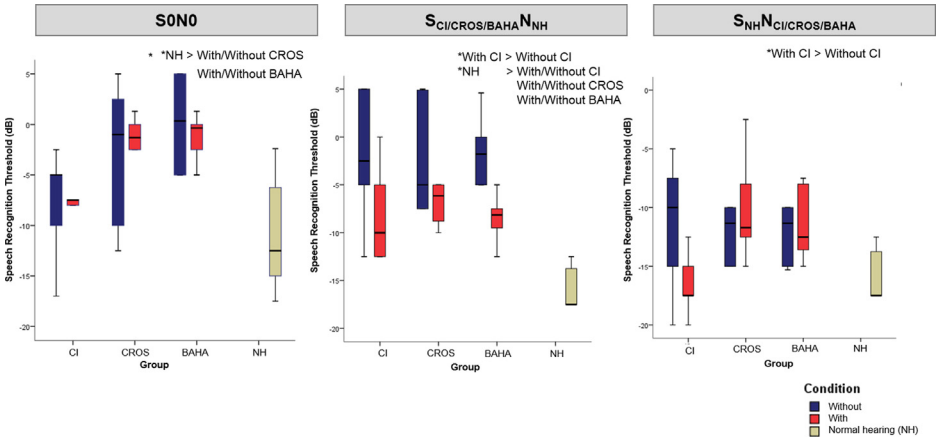

Fig. 1. Auditory performance assessment on speech intelligibility threshold in the 4 groups (CI, CROS, BAHA, controls) under 3 spatial conditions ($S_{NE}N_{IC/CROS/BAHA}$; S0N0; $S_{IC/CROS/BAHA}N_{NE}$). Red bars, with; blue bars, without rehabilitation device; black bars, controls. Box-plots showing 25th, 50th and 75th percentiles; error bars showing 5th and 95th percentiles. Asterisks indicate significant differences between rehabilitation devices (*$P < 0.05$, Wilcoxon test) or between controls and patients according to device ($P < 0.05$, Mann–Whitney test).

Table 2
Results under the 3 conditions ($S_{IC/CROS/BAHA}N_{NE}$, $S_{NE}N_{IC/CROS/BAHA}$ et S0N0) to asses system efficacy (Wilcoxon test), and compare patients versus controls (Mann–Whitney test).

	$S_{IC/CROS/BAHA}N_{NE}$		$S_{NE}N_{IC/CROS/BAHA}$		S0N0	
System efficacy (Wilcoxon test)	*P*	W	*P*	W	*P*	W
with CI vs. without CI (*n* = 9)	0.04*	31	0.02*	33	0.8	6
with CROS vs. without CROS (*n* = 6)	0.2	11	1	0	1	0
with BAHA vs. without BAHA (*n* = 6)	0.06	15	1	−1	0.4	7
Comparison of efficacy (Kruskal–Wallis test)	*P*	K	*P*	K	*P*	K
With-without: CROS (*n* = 6) vs. BAHA (*n* = 6) vs. CI (*n* = 9)	0.68	0.77	0.031*	6.95	0.97	0.068
Comparison versus control (Mann–Whitney test)	*P*	U	*P*	U	*P*	U
with CI (*n* = 9) vs. control (*n* = 7)	0.02*	9	0.6	26	0.5	25
without CI (*n* = 9) vs. control (*n* = 7)	0.005**	4.5	0.1	16.5	0.3	22
with CROS (*n* = 6) vs. control (*n* = 7)	0.04*	6	0.06	7.5	0.01*	4
without CROS (*n* = 6) vs. control (*n* = 7)	0.02*	4	0.08	8.5	0.04*	6.5
with BAHA (*n* = 6) vs. control (*n* = 7)	0.04*	6.5	0.1	9.5	0.01*	2.5
without BAHA (*n* = 6) vs. control (*n* = 7)	0.007**	2	0.1	10	0.01*	3

Asterisks and italics represent significant differences (*<0.05, **< 0.01). CI: cochlear implant; CROS: Contralateral Routing of Signal; BAHA: Bone-Anchored Hearing Aid.

3.2. Cortical auditory evoked potentials

Fig. 2 shows results (amplitude and latency) for waves P1, N1 and P2.

Comparing results "with" and "without" devices showed significantly increased amplitude only for N1 peak ($P = 0.03$, $W = -37$) in CI ("with": 0.9 ± 0.3 > "without": 0.7 ± 0.2 μV). The BAHA and CROS groups showed no significant differences in peak amplitude for P1, N1 or P2 ($P > 0.05$). There were no significant differences in P1, N1 or P2 peal latency between the 3 patient groups ($P > 0.05$).

Latency analysis found no significant differences between patients and controls, except for N1 latency, which was delayed in CROS ("with": 117 ± 9 ms; $P = 0.04$, $U = 5$; "without": 131 ± 19 ms; $P = 0.03$, $U = 6$; controls: 100 ± 21 ms), and P2 latency, which was delayed in CI ("with": 212 ± 19 ms; controls: 182 ± 16 ms; $P = 0.0081$, $U = 6$).

There were no differences in amplitude between controls and the 3 patient groups on any wave ($P > 0.05$).

3.3. Potential field mapping

For wave N1, without auditory rehabilitation all patients presented frontocentral negativity (Fig. 3). Negativity increased with use of CI or CROS, coming closer to control values. In controls, there was also an inversion of polarity (positivity) in the temporal and mastoid regions. Without auditory rehabilitation, all patients showed less or no temporomastoid inversion on the healthy side. Using the CI induced contralateral temporomastoid polarity inversion with more symmetrical activation, as in controls. With CROS, inversion increased on the contralateral side, but with persistent ipsilateral asymmetry. With BAHA, the positive amplitude observed without the system became negative when the system was used.

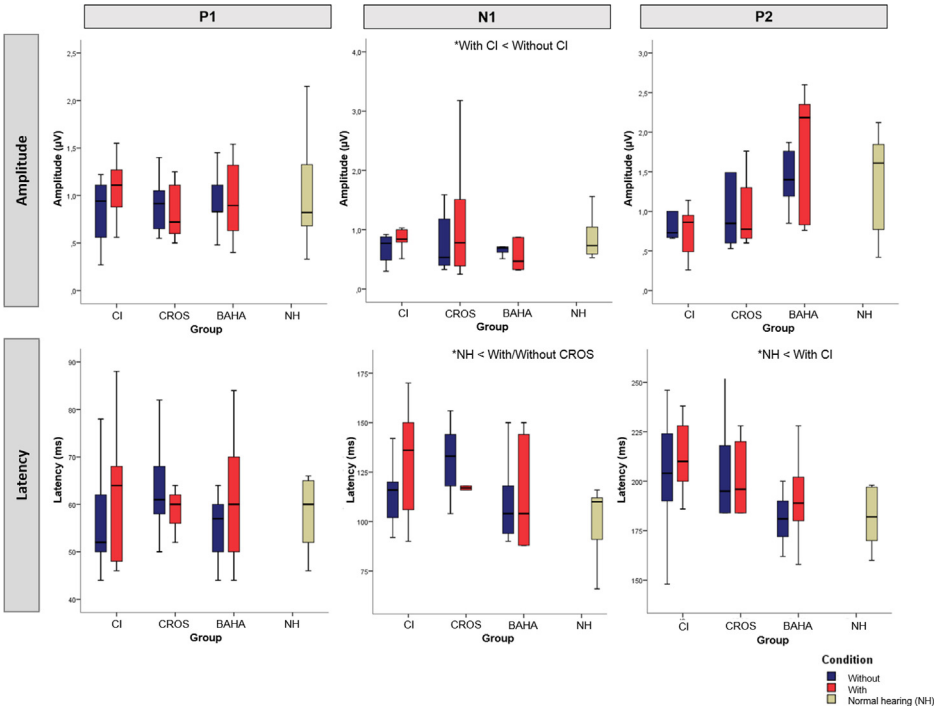

Fig. 2. Box-plots for amplitude (above) and latency (below) of cortical auditory response waves P1, N1 and P2 (left to right). Red bars, with; blue bars, without rehabilitation device; black bars, controls. Box-plots showing 25th, 50th and 75th percentiles; error bars showing 5th and 95th percentiles. Asterisks indicate significant differences between rehabilitation devices (*$P < 0.05$, Wilcoxon test) or between controls and patients according to device ($P < 0.05$, Mann–Whitney test).

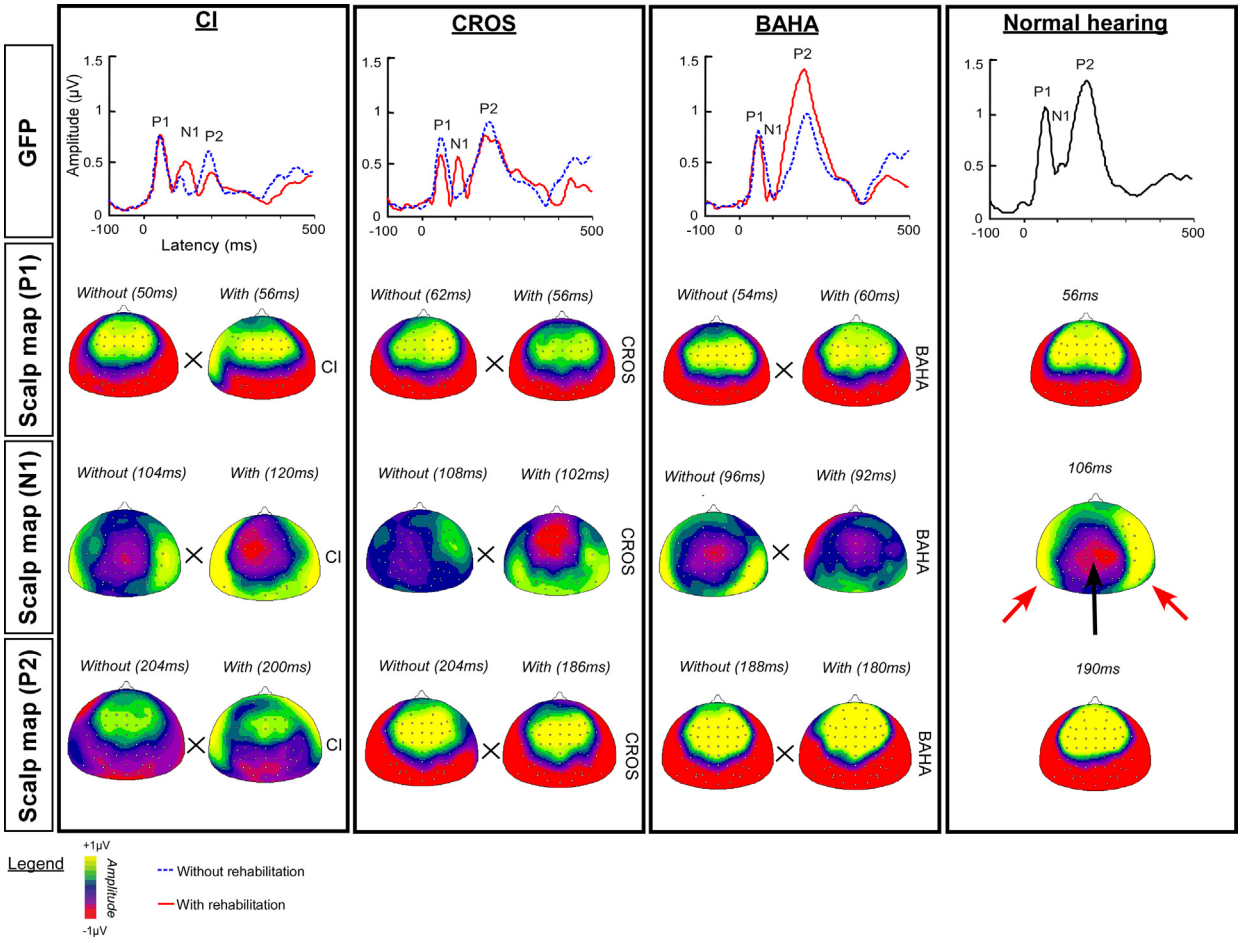

Fig. 3. Mean Global Field Power (GFP) in (left to right) CI, CROS and BAHA groups with and without device, and controls. From top to bottom: GFP curves, wave P1, N1 and P2 potential field maps. Arrows on control N1 potential field map indicate frontocentral negativity (black) and temporomastoid electrode polarity inversion (positive amplitude), (red).

For waves P1 and P2, controls and patients with their devices showed similar maps, with positive frontocentral amplitude and polarity inversion (negative amplitude) in the temporal and mastoid regions (Fig. 3).

4. Discussion

The present study demonstrated differences in cortical reorganization and auditory performance in the $S_{IC/CROS/BAHA}N_{NE}$ and $S_{NE}N_{IC/CROS/BAHA}$ conditions following restoration of binaural hearing by CI, in contrast to the CROS and BAHA rehabilitation systems.

The impact of loss of binaural hearing was greater when speech and noise input were separated ($S_{IC/CROS/BAHA}N_{NE}$ and $S_{NE}N_{IC/CROS/BAHA}$ conditions). The human auditory system then can no longer analyze differences in intensity and timing between speech and noise delivered to the two ears. Restoring binaural hearing by CI enables the auditory system to makes use of the advantages of the better ear by the head-shadow effect, and integrating binaurality unmasks the target signal. As other studies have also shown, the present results demonstrate that only CIs improved discrimination of speech in noise in the $S_{IC/CROS/BAHA}N_{NE}$ [8,19,34] and $S_{NE}N_{IC/CROS/BAHA}$ [34] conditions. The CROS system cannot stimulate the impaired ear, and BAHA could not do so sufficiently with the severe-to-profound hearing loss determined by the present inclusion criteria. Information transfer from the better side is artificial and insufficient to improve understanding (Fig. 1). Comparison of efficacy between the three systems revealed a difference for $S_{NE}N_{IC/CROS/BAHA}$, without selecting any one system. Meta-analyses of the HINT data [4,35] showed better performance with CROS, than BAHA. The present results suggest better performance with CI than CROS or BAHA. Comparison between the 3 groups versus controls showed that performance for $S_{IC/CROS/BAHA}N_{NE}$ remained poor, highlighting the fact that a CI is not equivalent to a normal-hearing ear.

In S0N0, speech and noise arrive simultaneously in the two ears with no interaural differences in intensity or timing. In this situation, none of the 3 systems improved intelligibility. The summation effect was thus not significant, as in other studies [19,20,23,34].

Wave N1 corresponds to the moment of signal detection, and is therefore of greater interest in adults [13]. On potential field maps, it shows frontocentral positivity and polarity inversion in the temporomastoid region in the hemisphere contralateral to the hearing loss when binaural hearing is restored by the CI (Fig. 3), due to increased activation in the corresponding superior temporal gyrus [36]. GFP wave N1 peak amplitude is significantly increased with CI, but not with CROS. This increase may be due to cortical reorganization and binaural summation with CI [37] and hearing-aid amplification with CROS [38]. BAHA seems not to induce any change in cortical auditory response; A recent longitudinal study demonstrated CI effects in unilateral hearing loss [20]; before CI rehabilitation, there was no activation in temporal and mastoid areas contralateral to the hearing loss as seen for the N1 wave

on potential field maps. In the present study, electrode activation in temporal and mastoid areas contralateral to the hearing loss was low-amplitude in CI patients not using their CI and in BAHA and CROS patients whether their device was activated or not (Fig. 3). This suggests that only CI activates these areas. The lack of significant amplitude difference between patients and controls may have been due to study design, with free-field stimulus delivery.

Statistical analysis of the GFP for amplitude found that P1 and P2 peaks did not differ between patients (3 groups) and controls. Potential field maps (Fig. 2) showed similar P1 peaks for patients and controls, and a lower P2 peak with CI than in the CROS, BAHA and control groups; this may have been due to greater inter-individual variation, as it did not emerge from the statistical analysis. In the literature, wave P1 corresponds to early perceptual processing of acoustic signal characteristics [39] and P2 to finer signal characteristics (e.g., whether it is a speech signal or not) [40]. The present study used a single/ba/speech stimulus, without comparative stimulation, which could not lead to any variation in waves P1 or P2.

There was no difference in GFP latency with or without auditory rehabilitation, as previously shown [41]. Comparison of the various peak latencies found no significant differences except for N1 in CROS, where latency was longer; the same applied to P2 peak latency in CI bearers, suggesting that potentials peak later in these patients for certain components [32].

The present study had certain limitations: group sizes were small, and side and duration of hearing loss were not taken into account. Other CAEP studies reported more rapid cortical reorganization in left-sided impairment and greater reorganization in hearing loss of more than 2 years' duration [42–44]. BAHA more frequently had right-sided ($n = 5/6$) and more long-standing (19 years) hearing loss than the other groups. This suggests more rapid cortical reorganization in these patients, with less difference in EEG results between the "with" and "without BAHA" conditions. On the other hand, hearing loss duration does not impact understanding of speech in noise [34,45], and the impact of side has never been assessed. As the CI and CROS groups had equivalent numbers of right- and left-side impairment and slightly shorter durations of hearing loss, it is more difficult to determine the impact of rehabilitation on cortical reorganization. The impact of microphonic directivity (adaptive directivity in CP910 processors versus omnidirectional directivity in Digisonic SP) and mean pure-tone threshold were not analyzed due to lack of power (small group), although the mean pure-tone threshold in the impaired ear may affect speech audiometry in noise, according to Vannson et al. (2017) [46].

It is also important to consider the wide inter-individual variations in results (large standard deviations): the present findings are to be interpreted with caution, and a larger cohort would be need for confirmation.

Another study limitation concerns artifacts generated by the CI. Manual independent component analysis is a long process, requiring double visual inspection by an experienced team [28,29]. Studies are underway to validate a technique of automatic artifact rejection by independent component analysis [30,31].

And lastly, the 3 types of rehabilitation were assessed independently in 3 groups, with consequent inter-individual variability. We wanted subjects to have been using their device for at least 1 year, so as to be able to analyze impact. We set an inclusion threshold for the normal-hearing ear at < 25 dB, for purposes of comparison with other reports [19,20,23,47], although other studies have used thresholds of 20 dB [48–51] or 30 dB [8,34,52]. To date, few patients have received auditory rehabilitation for unilateral severe to profound hearing loss, and many desire rehabilitation for thresholds > 30 dB.

5. Conclusion

CI improved GFP N1-wave peak amplitude and speech intelligibility under certain conditions, unlike the CROS or BAHA systems. These findings confirm the contribution of CI in unilateral severe to profound hearing loss, but careful patient selection remains mandatory. A longitudinal study could better assess and shed light on the progression of cortical reorganization following CI.

Disclosure of Interest

The authors declare that they have no competing interest.

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References

[1] McLeod B, Upfold L, Taylor A. Self reported hearing difficulties following excision of vestibular schwannoma. Int J Audiol 2008;47:420–30.
[2] Borton SA, Mauze E, Lieu JEC. Quality of life in children with unilateral hearing loss: a pilot study. Am J Audiol 2010;19:61–72.
[3] Giolas TG, Wark DJ. Communication problems associated with unilateral hearing loss. J Speech Hear Disord 1967;32:336–43.
[4] Wazen JJ, Spitzer JB, Ghossaini SN, Fayad JN, Niparko JK, Cox K, et al. Transcranial contralateral cochlear stimulation in unilateral deafness. Otolaryngol Head Neck Surg 2003;129:248–54.
[5] Vannson N, James C, Fraysse B, Strelnikov K, Barone P, Deguine O, et al. Quality of life and auditory performance in adults with asymmetric hearing loss. Audiol Neurootol 2015;20(Suppl 1):38–43.
[6] Valente M, Peterein J, Goebel J, Neely JG. Four cases of acoustic neuromas with normal hearing. J Am Acad Audiol 1995;6:203–10.
[7] Haute Autorité de Santé Traitement de la surdité par pose d'implants cochléaires ou d'implants du tronc cérébral n.d. https://www.has-sante.fr/portail/jcms/r_1498772/fr/traitement-de-la-surdite-par-pose-d-implants-cochleaires-ou-d-implants-du-tronc-cerebral.(accessed June 3, 2018).
[8] Arndt S, Laszig R, Aschendorff A, Hassepass F, Beck R, Wesarg T. Cochlear implant treatment of patients with single-sided deafness or asymmetric hearing loss. HNO 2017.
[9] Kitterick PT, Lucas L, Smith SN. Improving health-related quality of life in single-sided deafness: a systematic review and meta-analysis. Audiol Neurootol 2015;20(Suppl 1):79–86.
[10] Ponton CW, Don M, Eggermont JJ, Waring MD, Masuda A. Maturation of human cortical auditory function: differences between normal-hearing children and children with cochlear implants. Ear Hear 1996;17:430.
[11] Sharma M, Purdy SC, Newall P, Wheldall K, Beaman R, Dillon H. Effects of identification technique extraction method, and stimulus type on mismatch negativity in adults and children. J Am Acad Audiol 2004;15:616–32.
[12] Lippé S, Martinez-Montes E, Arcand C, Lassonde M. Electrophysiological study of auditory development. Neuroscience 2009;164:1108–18.
[13] Näätänen R, Picton T. The N1 wave of the human electric and magnetic response to sound: a review and an analysis of the component structure. Psychophysiology 1987;24:375–425.
[14] Vaughan HG, Ritter W. The sources of auditory evoked responses recorded from the human scalp. Electroencephalogr Clin Neurophysiol 1970;28:360–7.
[15] Peronnet F, Michel F, Echallier JF, Girod J. Coronal topography of human auditory evoked responses. Electroencephalogr Clin Neurophysiol 1974;37:225–30.
[16] Sharma A, Dorman MF, Spahr AJ. A sensitive period for the development of the central auditory system in children with cochlear implants: implications for age of implantation. Ear Hear 2002;23:532–9.
[17] Sharma A, Dorman M, Spahr A, Todd NW. Early cochlear implantation in children allows normal development of central auditory pathways. Ann Otol Rhinol Laryngol Suppl 2002;189:38–41.
[18] A.M.A. In: Rondinelli RD, editor. Guides to the Evaluation of Permanent Impairment. 6th ed Chicago: American Medical Association; 2008.
[19] Dorbeau C, Galvin J, Fu Q-J, Legris E, Marx M, Bakhos D. Binaural perception in single-sided deaf cochlear implant users with unrestricted or restricted acoustic hearing in the non-implanted ear. Audiol Neurootol 2018;23:187–97.
[20] Legris E, Galvin J, Roux S, Gomot M, Aoustin J-M, Marx M, et al. Cortical reorganization after cochlear implantation for adults with single-sided deafness. PloS One 2018;13:e0204402.
[21] Byers JF, Waugh WR, Lowman LB. Sound level exposure of high-risk infants in different environmental conditions. Neonatal Netw NN 2006;25:25–32.
[22] Peterson GE, Lehiste I, Revised CNC. lists for auditory tests. J Speech Hear Disord 1962;27:62–70.

[23] Galvin JJ, Fu Q-J, Wilkinson EP, Mills D, Hagan SC, Lupo JE, et al. 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 Hear 2018.
[24] Fuller CD, Galvin JJ, Maat B, Başkent D, Free RH. Comparison of two music training approaches on music and speech perception in cochlear implant users. Trends Hear 2018;22 [2331216518765379].
[25] Fraysse B, Dillier N, Klenzner T, Laszig R, Manrique M, Morera Perez C, et al. Cochlear implants for adults obtaining marginal benefit from acoustic amplification: a European study. Am J Otol 1998;19:591–7.
[26] Delorme A, Makeig S. EEGLAB: an open source toolbox for analysis of single-trial EEG dynamics including independent component analysis. J Neurosci Methods 2004;134:9–21.
[27] Debener S, Hine J, Bleeck S, Eyles J. Source localization of auditory evoked potentials after cochlear implantation. Psychophysiology 2008;45:20–4.
[28] Bakhos D, Roux S, Robier A, Bonnet-Brilhault F, Lescanne E, Bruneau N. Minimization of cochlear implant artifact in cortical auditory evoked potentials in children. Int J Pediatr Otorhinolaryngol 2012;76:1627–32.
[29] Viola FC, Thorne JD, Bleeck S, Eyles J, Debener S. Uncovering auditory evoked potentials from cochlear implant users with independent component analysis. Psychophysiology n.d.;48:1470-80.
[30] Viola FC, Thorne J, Edmonds B, Schneider T, Eichele T, Debener S. Semi-automatic identification of independent components representing EEG artifact. Clin Neurophysiol 2009;120:868–77.
[31] Viola FC, De Vos M, Hine J, Sandmann P, Bleeck S, Eyles J, et al. Semi-automatic attenuation of cochlear implant artifacts for the evaluation of late auditory evoked potentials. Hear Res 2012;284:6–15.
[32] Skrandies W. Global field power and topographic similarity. Brain Topogr 1990;3:137–41.
[33] Guiraud J, Besle J, Arnold L, Boyle P, Giard M-H, Bertrand O, et al. Evidence of a tonotopic organization of the auditory cortex in cochlear implant users. J Neurosci Off J Soc Neurosci 2007;27:7838–46.
[34] Távora-Vieira D, De Ceulaer G, Govaerts PJ, Rajan GP. Cochlear implantation improves localization ability in patients with unilateral deafness. Ear Hear 2015;36:e93–8.
[35] Niparko JK, Cox KM, Lustig LR. Comparison of the bone anchored hearing aid implantable hearing device with contralateral routing of offside signal amplification in the rehabilitation of unilateral deafness. Otol Neurotol 2003;24:73–8.
[36] Knight RT, Scabini D, Woods DL, Clayworth C. The effects of lesions of superior temporal gyrus and inferior parietal lobe on temporal and vertex components of the human AEP. Electroencephalogr Clin Neurophysiol 1988;70:499–509.
[37] Firszt JB, Holden LK, Reeder RM, Cowdrey L, King S. Cochlear implantation in adults with asymmetric hearing loss. Ear Hear 2012;33:521–33.
[38] Billings CJ, Tremblay KL, Souza PE, Binns MA. Effects of hearing aid amplification and stimulus intensity on cortical auditory evoked potentials. Audiol Neurotol 2007;12:234–46.
[39] Čeponienė R, Alku P, Westerfield M, Torki M, Townsend J. ERPs differentiate syllable and nonphonetic sound processing in children and adults. Psychophysiology 2005;42:391–406.
[40] Crowley KE, Colrain IM. A review of the evidence for P2 being an independent component process: age, sleep and modality. Clin Neurophysiol 2004;115:732–44.
[41] Tringali S, Marzin A, Dubreuil C, Ferber-Viart C. Bone-anchored hearing aid in unilateral inner ear deafness: electrophysiological results in patients following vestibular schwannoma removal. Acta Otolaryngol (Stockh) 2008;128:1203–10.
[42] Hanss J, Veuillet E, Adjout K, Besle J, Collet L, Thai-Van H. The effect of long-term unilateral deafness on the activation pattern in the auditory cortices of French-native speakers: influence of deafness side. BMC Neurosci 2009; 10:23.
[43] Khosla D, Ponton CW, Eggermont JJ, Kwong B, Don M, Vasama J-P. Differential ear effects of profound unilateral deafness on the adult human central auditory system. J Assoc Res Otolaryngol 2003;4:235–49.
[44] Ponton CW, Vasama JP, Tremblay K, Khosla D, Kwong B, Don M. Plasticity in the adult human central auditory system: evidence from late-onset profound unilateral deafness. Hear Res 2001;154:32–44.
[45] Welsh LW, Welsh JJ, Rosen LF, Dragonette JE. Functional impairments due to unilateral deafness. Ann Otol Rhinol Laryngol 2004;113:987–93.
[46] Vannson N, James CJ, Fraysse B, Lescure B, Strelnikov K, Deguine O, et al. Speech-in-noise perception in unilateral hearing loss: relation to pure-tone thresholds and brainstem plasticity. Neuropsychologia 2017;102:135–43.
[47] Prejban DA, Hamzavi J-S, Arnoldner C, Liepins R, Honeder C, Kaider A, et al. Single sided deaf cochlear implant users in the difficult listening situation: speech perception and subjective benefit. Otol Neurotol 2018;39:e803–9.
[48] Vermeire K, Van de Heyning P. Binaural hearing after cochlear implantation in subjects with unilateral sensorineural deafness and tinnitus. Audiol Neurootol 2009;14:163–71.
[49] Gartrell BC, Jones HG, Kan A, Buhr-Lawler M, Gubbels SP, Litovsky RY. Investigating long-term effects of cochlear implantation in single-sided deafness: a best practice model for longitudinal assessment of spatial hearing abilities and tinnitus handicap. Otol Neurotol 2014;35:1525–32.
[50] Zeitler DM, Dorman MF, Natale SJ, Loiselle L, Yost WA, Gifford RH. Sound source localization and speech understanding in complex listening environments by single-sided deaf listeners after cochlear implantation. Otol Neurotol 2015;36:1467–71.
[51] Litovsky RY, Moua K, Godar S, Kan A, Misurelli SM, Lee DJ. Restoration of spatial hearing in adult cochlear implant users with single-sided deafness. Hear Res 2018.
[52] Mertens G, Desmet J, De Bodt M, Van de Heyning P. Prospective case-controlled sound localisation study after cochlear implantation in adults with single-sided deafness and ipsilateral tinnitus. Clin Otolaryngol 2016;41:511–8.