Published in final edited form as:

Audiol Neurotol. 2016 ; 21(3): 141–149. doi:10.1159/000444739.

Exploring the source of neural responses of different latency obtained from different recording electrodes in cochlear implant users

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Abstract

In this study we measured the electrically evoked compound action potential (ECAP) from different recording electrodes in cochlea. Under the assumption that different response latency may be the result of differences in the neural population contributing to the response, we assessed the relationship between neural response latency and spread of excitation. First, we evaluated changes in N1 latency when the recording electrode site was varied. Second, we recorded channel interaction functions using a forward masking technique but with recording electrodes at different intra-cochlear locations. For most individuals, N1 latency was similar across recording electrodes. However, reduced N1 latencies were observed in 21% of CI users when ECAPs were recorded using a remote recording electrode. We hypothesized that if recordings from different electrodes represented contributions from different populations of neurons, then one might expect that channel interaction functions would be different. However, we did not observe consistent differences in channel interaction functions (neither peak location nor breadth of the functions), and further, any variation in channel interaction functions were not correlated with ECAP latency. These results suggest that ECAPs from different recording electrodes with different latency originate from similar neural populations.

Keywords

“electrically evoked potential”; “compound action potential”; “auditory system”; “auditory evoked potentials”; “cochlear implant”

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Conflicts of interest

None of the authors report a conflict of interest.

Introduction

Multichannel cochlear implants (CIs) produce dramatic improvements in speech perception for patients with severe-to-profound sensorineural hearing loss [Gantz et al., 1998]. However, the information conveyed by the CI is temporally and spectrally degraded relative to the acoustic signal [Loizou et al., 1998; Wilson and Dorman, 2008]. This degradation arises in part due to limitations in the implant itself such as the number of channels. It may also be due to spread of neural excitation away from the stimulating electrode. Spread of current across the cochlear duct results in channel interaction, i.e., multiple electrodes stimulate overlapping neural populations. Channel interaction, in turn, can cause spectral or temporal smearing. It has been shown to produce perceptual changes in loudness [Shannon, 1983; White et al., 1984] and elevate detection thresholds in psychophysical forward-masking experiments [Boex et al., 2003; Chatterjee and Shannon, 1998; Throckmorton and Collins, 1998]. It seems likely that excess channel interaction could have a negative impact on speech perception. Additionally, recent computational models of electrical potential distributions have shown that spread of excitation can occur not only in adjacent neural populations, but also in more distant neural populations because of electrode current spreading across cochlear turns (i.e. cross-turn or ectopic stimulation) [Briaire and Frijns, 2006; Hanekom, 2001].

Electrically evoked compound action potentials (ECAP) have been used to try to measure the way current spreads across the cochlear duct in multichannel cochlear implant users. ECAPs are recordings of the synchronous firing of a large number of auditory nerve fibers to an electrical stimulus. Typically ECAPs are measured using the reverse telemetry capabilities of the cochlear implant; current is delivered to one electrode and a neural response is recorded from another electrode. In this study we used a standard masker-probe paradigm to eliminate stimulus artifact. The morphology of the response consists of a negative peak (N1) followed by a positive peak (P2), with typical latencies of 200–400 μ s and 600–800 μ s, respectively [Abbas et al., 1999; Brown et al., 1998].

There are two different ways to use ECAPs to assess spread of excitation across the cochlea. One method is to fix the location of the stimulating electrode and record a series of ECAPs while systematically varying the location of the recording electrode [Cohen et al., 2004; Hughes and Stille, 2010; Van der Beek et al., 2012]. As the recording electrode is moved away from the stimulating electrode, ECAP amplitudes generally decrease. These spread of excitation functions are often used to describe how current spreads across the length of the cochlea. An inherent assumption associated with this method is that only neural activity from neurons located very close to the recording electrode are sampled. Clearly, that may not always be the case. Each of the intracochlear electrodes can record neural activity from a range of neurons.

A second method that can be used to measure spread of excitation across the cochlea is to fix the stimulating and recording electrodes but to record a series of ECAPs while systematically varying the masker electrode across the intracochlear electrode array. If there is overlap between the neurons responding to the masker and probe, then due to refractory properties of the auditory neurons, the response to the probe will be reduced. ECAP

amplitude should provide an indication of the relative overlap between neural populations responding to the masker and the probe. These functions are generally called channel interaction functions and they represent an alternative approach to measuring spread of neural excitation in the cochlea [Abbas et al., 2004; Cohen et al., 2003; Peeters et al., 1998]. When channel interaction functions are recorded, the recording electrode is typically fixed, usually near the probe electrode. The assumption is that the recording electrode samples neural activity from a wide range within the cochlea. Channel interaction functions provide an indirect estimate of the spread of neural excitation [Hughes and Stille, 2010].

Finley et al. [2013] recently reported ECAPs from a group of Advanced Bionics cochlear implant users where they fixed the stimulating electrode and systematically varied the recording electrode across the array. They described changes in ECAP latency as the recording electrode was varied. For some cochlear implant users, N1 latencies became shorter as the recording electrode was moved away from the stimulating electrode. For apical stimulation sites, the latency became shorter as the recording site was moved basally and conversely, for basal stimulation sites shorter latencies were observed as the recording electrode was moved apically. The authors proposed that the responses with two different latencies may represent recordings that were dominated by neural response from neurons located in two anatomically distinct regions. One region may be the ganglion cell region located near the stimulating electrode, which responds with a long N1 latency. The other is located in the modiolus or at the base of the cochlea, where the VIIIth cranial nerve exits the modiolus to the internal auditory meatus. In theory, neurons from this location may be expected to have shorter N1 latencies. We would expect that stimulation at the ganglion cell site is more spatially specific due to the relatively diffuse arrangement of auditory fibers, whereas modiolus stimulation likely results in a larger group of neurons, or at least a different population of neurons, being stimulated due to more compact arrangement of auditory fibers.

In this study, we evaluated changes in N1 latency when the recording electrode site was varied, essentially replicating the experiment of Finley et al. [2013]. However, we also recorded a series of channel interaction functions using recording electrodes at different intracochlear locations. Our goal was to test the hypothesis that ECAPs with long peak latencies are dominated by peripherally excited, spatially specific neural populations, and alternatively, that ECAPs with relatively short peak latencies are dominated by centrally excited, spatially compact neural populations. We hypothesized that if ECAPs with a long-latency N1 peak were dominated by activity of spatially specific neural populations, then they would demonstrate relatively narrow channel interaction functions with the peak at or near the probe stimulating electrode if the recording was near the probe electrode. For recording electrodes more remote to the probe (specifically those associated with the shorter-latency responses presumably originating from modiolar stimulation), we hypothesized that the channel interaction functions would be broader and/or centered on a different electrode, reflecting the different population of stimulated neurons dominating that response.

Materials and Methods

Subjects

Thirty-four cochlear implant recipients ranging from four to 79 years of age (mean age = 47.4) participated in the study. All CI recipients were implanted at the University of Iowa. Demographic data about each study participant is presented in Table 1. Eight ears were implanted with the Advanced Bionics CI system and 28 ears had the Nucleus CI device. Five individuals had bilateral cochlear implants. Both ears of these individuals were tested on the same day bringing the total number of ears tested to 39. Small amplitude ECAPs were recorded from five ears, which limited the amount of useful data we could record (i.e., only one apical or basal probe).

The University of Iowa Institutional Review Board approved the protocols used in this study. All participants or their guardians signed an informed consent document after being provided detailed information on the scope, potential risks, and benefits of the procedures used.

Stimuli

For subjects who used a Nucleus CI, experimental stimuli were generated using the Cochlear NRT (version 3.1) or Custom Sound EP (version 4.2) software. Subjects who had an Advanced Bionics CI were tested using the Bionic Ear Data Collection System (BEDCS) as well as SoundWave. BEDCS is a research software platform provided by Advanced Bionics for use with CII and 90k cochlear implants. The stimulation rate used for the Nucleus CI recipients was 80 pps. A stimulation rate of approximately 30 pps was used for Advanced Bionics CI users. Biphasic pulses with pulse widths of 25, 37, or 50 μ s/phase were used for Cochlear devices as needed to optimize ECAP responses for the individual. For all Advanced Bionics CI users, a pulse width of 32 μ s/phase was used. All stimuli were cathodic leading biphasic pulses.

Stimulation levels

For each adult participant, the stimulus used for obtaining ECAP recordings was presented to the patient and he/she was asked to identify the maximum comfort stimulation level (C_{exp}). C_{exp} levels were measured for each electrode that the subject used in his/her clinical or “everyday” MAP. Electrodes deactivated in the individual’s clinical MAP were not tested. The stimulation level was initially low but was increased in steps of 5 CL for individuals with Cochlear devices and 32 μ A for individuals with Advanced Bionics devices. The C_{exp} was defined as the stimulus level one step size below the level the subject identified as being uncomfortable. The lowest C_{exp} across all electrodes in a given ear and associated electrode were noted.

C_{exp} was used along with the participants’ clinical MAP, specifically loudest comfortable (C_{MAP}) levels for Cochlear devices and most comfortable (M) levels for AB devices, to determine experimental stimulation levels. MAP levels are associated with higher stimulation rates than the experimental stimuli used to elicit ECAPs. The low-rate experimental stimulation levels were initially set equal to C_{MAP} /M levels, and then elevated

in equal amounts across the entire electrode array until the level of the electrode associated with the lowest C_{exp} was set to that level. This method of setting stimulation levels allows us to keep the MAP contour intact but increase level appropriate to the lower rate stimulus used to measure ECAPs.

For children who could not provide loudness ratings behaviorally (CE79L, CE102R CE54R, CE66R, CS12R, CS12L, CF23L, CL5R, CL7L, CL6, CZ33L, CZ33R), automated neural response telemetry (Custom Sound AutoNRT for Cochlear devices and SoundWave NRI for Advanced Bionics) was used to measure ECAP thresholds for all of the intracochlear electrodes. This automated algorithm identifies the lowest current level needed to obtain an ECAP using a 25 μ s pulse-width stimulus (for Cochlear devices) and a 32 μ s pulse-width stimulus (for Advanced Bionics devices). For these younger subjects, experimental stimulation levels were initially set equal to MAP C levels, then, stimulating at the lower rate used for ECAP measures, levels were elevated (keeping the same contour across electrodes) until the participant showed some sign of discomfort. Final stimulation levels were set below that value. Due to variations of ECAP thresholds and stimulus uncomfortable levels, the experimental stimulation levels ranged from 19 CL below to 69 CL above ECAP thresholds for the Cochlear devices and 138.5 to 487.5 μ A above ECAP thresholds for Advanced Bionics devices. Neither the experimenters nor the children's guardians noted any signs of discomfort when the electrical stimuli were presented at the final stimulation levels.

Recording parameters

ECAPs were recorded using NRT, Custom Sound EP, or BEDCS software as appropriate. In each case, ECAP recording parameters (e.g., amplifier gain, recording delay) were optimized and electrical stimulus artifact was removed using a subtraction method described previously [Abbas et al., 1999; Brown et al., 1998; Cohen et al., 2003; Dillier et al., 2002]. The recording electrode was typically two electrodes apical to the probe. Electrodes that did not show clear responses were excluded from use either as a probe or masker electrodes from the study. From the electrodes with clear ECAPs, the second most apical and second most basal electrodes were chosen as probe stimulus electrodes (Table 1). For each probe electrode, spread of excitation and channel interaction functions were both measured.

To measure spread of excitation, the same electrode was used for both the masker and probe pulses and the recording electrode was varied. The probe level was determined by the procedure described previously and the masker level was fixed 10 CL units above the probe level for Cochlear devices and 64 μ A above the probe level for AB devices. The higher level masker allows us to obtain a recording of the probe artifact which is ultimately used to calculate the ECAP response to the probe [Brown et al., 1998]. A standard subtraction method was used to extract the neural response from the stimulus artifact [Abbas et al, 1999].

Figure 1 shows typical ECAP waveforms recorded from different recording electrodes in two different CI users. In both cases the largest amplitude responses tend to occur when the recording electrode is closer to the stimulating electrode. ECAP amplitudes decrease as the recording electrode becomes more distant from the stimulating electrode. Figure 1A shows an example of a response where the latency of the N1 peak changes little as the recording

electrode is varied. This pattern is typical of approximately 80% of our recordings. Figure 1B illustrates a case where there is a clear shift in N1 latency as the recording electrode is moved farther from the probe electrode. The indicated N1 peak (vertical bar) on each trace shows a decrease in latency of approximately 100 μ s going from electrode 2 to 22. Although we observed similar patterns of latency change in several individuals, the degree of change was variable.

N1 latency and N1-P2 amplitude were measured offline using a custom written MATLAB script. Since we were interested in characterizing the effects of recording electrode location on latency, N1 latency of the two recording electrodes located farthest from the probe were averaged and N1 latencies recorded from three recording electrodes closest to the probe were averaged. The latency shift was calculated as the difference between averaged N1 latency for near and remote recording electrodes.

Channel interaction functions were also measured for both probe electrodes. The probe electrode and stimulation level was fixed and the masker electrode was varied across all other active electrodes in the array. This method results in larger amplitude responses for masker-probe pairs that result in greater overlap (greater channel interaction). A total of four channel interaction functions were measured for each probe electrode, each with a different recording electrode. Two recording electrodes were close to the probe and two recording electrodes were far from the probe in either basal or apical directions. For example, for Cochlear devices, channel interaction functions were typically measured on probe electrode 2 using recording electrodes 4, 5, 20, and 21. For probe electrode 21, we used recording electrodes 4, 5, 18, and 19. Similar choices were made for the 16-electrode Advanced Bionics implant. If proximal and remote recording electrodes were dominated by peripheral vs modiolar activity, respectively, we might expect that channel interaction functions would display different peak locations and/or exhibit different amounts of spread of excitation across electrodes.

To analyze channel interaction functions, ECAP amplitudes for each masker electrode were normalized to the maximum amplitude obtained across all masker electrodes. The two normalized channel interaction functions obtained for the near recording electrodes were averaged and the two normalized functions obtained from remote recording electrodes were also averaged. An example is shown in Figure 2. Channel interaction for probe 2 was recorded using electrodes 4, 5, 20, and 21. This example is typical of our data in that we generally observed only small differences in the channel interaction functions for near and remote recording electrodes.

Finally, to quantify differences between channel interaction functions, we calculated both the area under the functions as well as determined the electrode at which the function was at a maximum. The peak of the function was determined as the masker electrode that elicited the highest ECAP amplitude for each channel interaction function. The area of the function was calculated as the sum of the normalized amplitudes across masker electrodes for a particular probe electrode. We hypothesized that peak location and spread (represented by area) would differ with peripheral and modiolar stimulation. We reasoned that if the response obtained using a remote recording electrode is dominated by activity due to ectopic

stimulation, then the peak location may be shifted from the probe electrode site and the spread would be broader.

Data analysis

SPSS (version 16.0, SPSS Inc. Chicago, IL, USA) software was used for data analysis. Pearson's Product-Moment Correlation was used to examine correlations between near and remote channel interaction function peak location and excitation area, and between channel-interaction function metrics and N1 latency. Student's t-tests were also used on the electrodes showing a significant latency shift to examine the effect of recording electrode location on channel interaction function areas and peak locations. P values < 0.05 were considered significant.

Results

N1 latency shift

We analyzed results from 39 ears (73 total electrodes) and calculated the N1 latency shift, the difference between the average N1 latency recorded using near electrodes and average N1 latency recorded using remote electrodes. For the 73 total electrodes tested, we observed latency shifts that ranged from -105.7 to 233.5 μ s, where positive differences represent a shorter latency with a remote recording electrode. Taking into account sampling rate of the data collection systems (20 kHz, or 50 μ s/sample for Cochlear devices and 56 kHz, or ~18 μ s/sample for AB devices), we set a threshold of N1 latency shifts of 100 μ s to be considered different. In 58 of 73 cases the absolute value of the latency shift was less than or equal to 100 μ s (i.e. not significant). In 15 cases the latency shift was greater than 100 μ s and in only one case it was less than -100 μ s (i.e., opposite of the hypothesized direction). Of the five bilaterally implanted patients, three demonstrated a latency shift but only one demonstrated a latency shift in both ears.

Differences in channel interaction functions with different recording electrodes

Figure 3A is a scatterplot of the peaks of the channel interaction functions measured with remote and near recording electrodes. Channel interaction functions often peak at or near the probe electrode location. Thus, there is a general clustering of points around apical and basal electrodes due to the choice of probe electrodes for this study. The peaks across electrode locations, however, illustrate several exceptions. More importantly, the near and remote peak locations are highly correlated and show no consistent deviation from the diagonal (equal for near and remote recording electrodes). Furthermore, the 15 probe electrodes associated with the greatest latency shifts with recording electrode location (open circles) are no different from the electrodes with smaller or no latency shifts (filled circles). There was no significant difference in peak location of the channel interaction function between the near and remote recording when all 73 cases are considered ($p=0.58$) or when the analysis is limited to the 15 probe electrodes ($p=0.59$).

Figure 3B shows a similar scatterplot for the area under the channel interaction function. The area for near and remote electrodes show a similar pattern, i.e., both variables are highly correlated. Moreover, the area associated with the remote electrode is not larger than the area

associated with the near electrode. This is true if we take into account all probe electrodes ($p=0.08$) or if we limit the analysis to the 15 electrodes associated with the greatest latency shifts with recording electrode location (white circles, $p=0.20$).

If an individual shows a latency shift with changes in recording electrode (e.g. Figure 1B) we hypothesized that stimulation of the modiolus would result in a different pattern of channel interaction. However, except for one case, no differences in peak electrode location were observed for interaction functions measured with near or remote recording electrodes (Figure 3A). To investigate this relationship further, Figure 3C plots peak differences of the channel interaction function relative to N1 latency shift. If shorter N1 latencies recorded from a remote electrode reflect modiolar stimulation, then larger changes in peak masker electrode location of the channel interaction functions would be expected to correlate with larger N1 latency differences. Peak location difference was calculated by subtracting averaged peak masker electrodes for near recording electrodes compared to remote electrodes. There was only one case where there were significant differences in the location of the peak and no correlation was observed between the N1 latency shift and the channel interaction function peak differences ($r = 0.034$, $p = 0.815$). The one participant who had a large difference in the peak also had a very noisy channel interaction function, which may preclude meaningful conclusions.

Channel interaction functions reflecting modiolar stimulation would be expected to be broader, reflecting stimulation of a more diverse neural population. We calculated the difference in area of channel interaction functions measured with a remote recording electrode and a near recording electrode normalized to the area of the near electrode and plotted those values relative to N1 latency difference (Figure 3D). Since two channel interaction functions each were used for near and remote recording electrodes, the area reflects the average channel interaction function. Positive numbers reflect broader channel interaction functions at remote recording sites. Changes in area are generally small and there was no significant correlation between N1 latency shift and area of channel interaction function difference ($r = 0.252$, $p = 0.08$).

Discussion

In this study, we showed that some electrodes (15/73) had reduced N1 latency when ECAPs were recorded from a remote electrode compared to that recorded from an electrode closer to the stimulus. This phenomenon was observed both for basal and apical probe electrodes and is consistent with previous data reported by Finley et al. [2013]. A similar observation has also been noted in Med-El Standard and Med-El Flexsoft implants [Schwarz et al., 2012], especially in cases where the stimulating electrode was located near the apex of the cochlea. One observation made by these investigators was an increased presence of double peaks with decreased N1 latencies. In the present data set, only 2 of 15 subjects with clear latency shifts also showed double-peaked ECAP responses. Both previous studies also documented reports of echo-like sensation in subjects showing latency shifts; unfortunately, we did not systemically gather that data from our subjects. The subjects/ears with a change in latency were not obviously different from subjects/ears without latency shifts in other aspects. We retrospectively examined correlations with the electrode site (modiolar hugging

vs. lateral wall), insertion depth, electrode array type, and the duration of the deafness, but we did not observe any significant correlations.

We hypothesized that recording electrodes used to obtain ECAPs with shorter N1 peak latencies would have broader, or at least different, channel interaction functions than electrodes from which ECAPs with longer peak latencies were recorded. This hypothesis was based on an assumption that the different latencies resulted from modiolar vs more peripherally stimulated neurons dominating the responses. However, we did not observe consistent differences in channel interaction functions, and any variation in channel interaction functions that was observed was not correlated with ECAP latency shifts. Moreover, while these observations do not preclude an ectopic stimulation site, it does suggest that the populations of responding neurons at both the adjacent and more remote recording sites were similar.

Several previous reports have assessed channel interaction functions obtained from different recording electrodes. Cohen et al. [2003] compared channel interaction functions recorded at two positions basal and two apical from the probe electrode, and found no differences in peak or width. Our findings are consistent with this previous report. Hughes and Stille [2010] assessed channel interaction functions recorded on up to four electrode positions relative to the probe for AB subjects, and up to six electrode positions relative to the probe for Cochlear subjects. Consistent with the trends reported here, in most cases they observed no difference in channel interaction functions across different recording electrode positions. They did note, however, that in a minority of cases (12%), narrower channel interaction functions were observed when the recording electrode was located close to the probe electrode. Van der Beek et al. [2012] performed similar recordings in five patients, and found that the width of the channel interaction function increased when more remote recording electrode sites were used. However, their measurements were done intraoperatively with high stimulation levels (up to 1.2 mA), which normally exceed loudness comfort levels of awake patients and can cause significant spread of excitation and broader interaction functions [Hughes and Stille, 2010]. In neither case, however, were comparisons to ECAP latency measures reported.

The relationship between single neuron response properties and ECAP measures is complicated by differences in response latency among fibers as well as relative contributions of orthodromic and antidromic propagated actions potentials to the recorded potential from an intracochlear electrode. Data from single fiber recordings in animal models have demonstrated evidence for differences in stimulation site (both peripheral and central to the cell body) consistent with differences in latency of centrally recorded action potentials [Miller et al., 2003; Javel and Shepherd, 2000; Stypulkowski and van den Honert, 1984]. These reports showed that variations in stimulus configuration could alter the site of stimulation within the same neuron. They also suggested that as the intensity of the stimulus increased, the place where the spikes originated tended to move from a more peripheral site to a site more central to the cell body. In addition, there is evidence that action potentials can propagate both orthodromically and antidromically in the stimulated auditory nerve fiber. Miller et al. [2004] compared ECAPs recorded from cat ears obtained from recording electrodes in the cochlea close to the probe and from recording electrodes located at the

nerve trunk. They suggested that the central site of action potential initiation produced both orthodromic and antidromic action potentials, and that the use of intracochlear recording electrodes can more effectively detect the antidromic action potentials. Briare and Frijns [2005] used a computer model to study the contributions of orthodromic and antidromic action potentials in the cochlea. That work demonstrated the complex nature of the ECAP waveform in that the contribution of different neuron populations to the recorded ECAP could be dependent on stimulus waveform (anodic vs cathodic) as well as stimulus level. Given that the ECAP can have contributions from neurons with different response latencies as well as both antidromic and orthodromic action potentials, the shift in latency observed in some individuals suggests that either a different population or perhaps a different place of action potential generation is dominating the response recorded at a particular location. The similarity of the channel interaction function shapes, however, suggests that the distribution of neurons contributing to the ECAP is similar for the different recording conditions.

One important limitation of the methods employed in this study is the use of the two-pulse channel interaction paradigm to assess spread of neurons contributing to the response [Abbas et al., 2004]. The method relies on refractory properties of the neurons to demonstrate overlap in the population of neurons responding to a masker and probe pulse. As a result the “spread” is dependent on the masker pulse as well as the probe [Consentino et al, in press]. In this study, we chose to leave masker and probe the same and compare channel interaction when only recording electrode is varied, under the assumption that differences in neurons contributing to the responses would result in changes in channel interaction function. Nevertheless, a broad spread of excitation to the masker could limit the sensitivity of the measure to different contributions to the probe response with changes in recording electrode.

Conclusion

In 21% of the ears tested, use of a recording electrode located at a site that was remote relative to the stimulating electrode produced ECAPs with shorter N1 latencies compared to similar measures obtained using a recording electrode located closer to the stimulating electrode. The hypothesis that differences in latency of response would be correlated with changes in channel interaction functions was not supported. Different response latency suggests a different site of action potential initiation, but this could be within the same neuron (peripheral process vs. central to the soma) rather than a different group of neurons (those with close to the stimulating electrode vs those clustered in the modiolus, excited by ectopic stimulation). Since no clear shift in peak or width of channel interaction functions was evident in our data, we suggest that ECAPs from different recording electrodes likely originate from similar neural populations.

Acknowledgments

Sources of funding

This study was funded by grants from the NIH/NIDCD (R01 DC012082; P50 DC000242).

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Figure 1.

Examples of ECAPs measured using different recording electrodes. Averaged recorded voltage is plotted as a function of time after probe offset (see text for details relative to artifact reduction).

(A) The ECAPs of probe electrode 21 for E95R (current level 161 μA). The recording electrode is indicated at the right of each trace. The amplitude responses tend to be larger when recording closer to the stimulating electrode. Short vertical bars on each trace indicate the N1 peak. In this case little or no change in N1 latency was observed.

(B) The ECAPs of probe electrode 2 for CE79L (current level of 187 μA). Response amplitude was larger when recording closer to the stimulating electrode. The N1 latency was shorter when the recording electrode was remote from stimulating electrode.

Figure 2.

Examples of averaged channel interaction functions of CE79L recorded from remote and near recording electrodes. Normalized amplitude of the recorded ECAP is plotted as a function of masker electrode. Channel interaction for probe 2 was recorded from electrodes 4, 5, 20 and 21. The average of the functions recorded by electrodes 4 and 5 is plotted using filled circles connected with a solid line. The average of the functions recorded by electrodes 20 and 21 is plotted using open circles connected with a dotted line.

Figure 3.

(A) Scatter plot showing averaged peak location of channel interaction functions recorded from near and remote electrodes. Most comparisons showed little differences in peak location.

(B) Scatter plot showing averaged normalized area of channel interaction functions recorded from near and remote electrodes. The areas (spread of excitation) tended to be similar for the two recording locations.

(C) Scatter plot showing the relationship between the N1 latency difference and peak difference (near vs remote recording location) depending on the remote and near recording sites. Peak difference was calculated by subtracting averaged peak of near electrode from that of the remote electrode. No correlation was observed between the two variables.

(D) Scatter plot showing difference in the area of channel interaction function recorded from near and remote recording electrodes as a function of N1 latency difference. Channel interaction function area difference was calculated as the difference of near recording area from far recording area, normalized by the area of the near electrode. There was no correlation evident between the N1 latency difference and the area difference. In every figure, filled circles represent the probe electrodes associated with the latency shifts less than 100μs and open circles represent the probe electrodes associated with the latency shifts more than 100μs.

Table 1

Subject Demographic Information

Subject	Gender	Device	Ear	Electrode Tested		Age (yrs)	Duration of CI use (mo)
				Basal	Apical		
M4	M	24M	Left		○	75	205
R47L	F	24R	Left		○	67	135
CR44	M	24R	Right	○	○	12	131
CE79L	M	24RECA	Left	○	○	12	52
E96L	M	24RECA	Left	○	○	70	62
E18	M	24RECA	Right	○	○	76	97
E39	M	24RECA	Left	○	○	31	86
E95R	M	24RECA	Right	○	○	65	25
CE102	M	24RECA	Right	○	○	6	25
CE54R	M	24RECA	Right	○	○	7	71
CE66R	M	24RECA	Right	○		13	60
E94R	F	24RECA	Right		○	58	25
S3L	M	CI422	Left	○	○	62	12
S5L	F	CI422	Left	○	○	65	12
S6R	F	CI422	Right	○	○	64	12
S4L	M	CI422	Left	○	○	59	11
S7L	F	CI422	Left	○	○	64	13
CS12R	M	CI422	Right	○	○	12	16
CS12L	M	CI422	Left	○	○	12	4
F27L	M	CI512	Left	○	○	64	48
F27R	M	CI512	Right	○	○	64	48
CF23L	M	CI512	Left	○	○	6	34
F22R	F	CI512	Right	○	○	58	45
F24L	F	CI512	Left	○	○	79	35
L7L	M	L24	Left	○	○	64	22
CL5R	F	L24	Right	○	○	13	18

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Subject	Gender	Device	Ear	Electrode Tested		Age (yrs)	Duration of CI use (mo)
				Basal	Apical		
CL7L	F	L24	Left	○	○	16	3
L15R	M	L24	Right	○	○	59	7
L13L	F	L24	Left	○	○	47	13
CL1L	M	L24	Left	○	○	13	24
CL6	F	L24	Left	○	○	14	9
H36L	F	CII	Left	○		70	144
H37	F	CII	Left	○	○	48	144
Z158L	M	90K	Left	○	○	63	11
Z157R	M	90K	Right	○	○	54	12
Z17R	M	90K	Right	○	○	75	120
CZ33L	F	90K	Left	○	○	4	39
CZ33R	F	90K	Right	○	○	4	39
Z156R	M	90K	Right	○	○	67	11

Bilateral Users:	CR44 / CE79L
	CE102 / CF23L
	F27L / F27R
	CS12R / CS12L
	CZ33R / CZ33L