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Electrophysiological and speech perception measures of auditory processing in experienced adult cochlear implant users

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Abstract

Objective: This study determined the relationship between auditory evoked potential measures and speech perception in experienced adult cochlear implant (CI) users and compared the CI evoked potential results to those of a group of age- and sex-matched control subjects.

Methods: CI subjects all used the Nucleus CI-22 implant. Middle latency response (MLR), obligatory cortical potentials (CAEP), mismatch negativity (MMN) and P3a auditory evoked potentials were recorded. Speech perception was evaluated using word and sentence tests.

Results: Duration of deafness correlated with speech scores with poor scores reflecting greater years of deafness. Na amplitude correlated negatively with duration of deafness, with small amplitudes reflecting greater duration of deafness. Overall, N1 amplitude was smaller in CI than control subjects. Earlier P2 latencies were associated with shorter durations of deafness and higher speech scores. In general, MMN was absent or degraded in CI subjects with poor speech scores.

Conclusions: Auditory evoked potentials are related to speech perception ability and provide objective evidence of central auditory processing differences across experienced CI users.

Significance: Since auditory evoked potentials relate to CI performance, they may be a useful tool for objectively evaluating the efficacy of speech processing strategies and/or auditory training approaches in both adults and children with cochlear implants.

Keywords: Auditory evoked potential; Cochlear implant; Speech perception; Mismatch negativity

1. Introduction

The range of speech performance among individual CI users varies widely. It is assumed that performance with an implant is strongly associated with the auditory processing abilities of the individual and the integrity of the central auditory pathways from the auditory nerve to the cortex (Kraus et al., 1998; Ponton et al., 1996a,b). Indeed, performance is correlated with duration of deafness, age at onset of deafness (pre, peri, or post-lingual), etiology of deafness, and the number of remaining spiral ganglion cells (Blamey, 1997; Fayad et al., 1991).

The integrity of the central auditory pathways can be studied using evoked potentials (EP). The origins of the various EPs have been difficult to establish but the middle latency and cortical responses have a number of generators including subcortical, thalamo-cortical projections, primary auditory cortex and association areas (McGee and Kraus, 1996; Picton et al., 1974). The relationship between EP and behavioral indices of auditory processing has been established in studies of temporal lobe lesion patients (Allen et al., 1996; Kileny et al., 1987; Kraus et al., 1982; Vaughan and Ritter, 1970; Woods et al., 1987), and children with learning problems (Jirsa and Clontz, 1990; Kraus et al., 1996; Purdy et al., 2002; Tonnquist-Uhlen, 1996). A number of studies (see review by Purdy et al., 2001) have demonstrated plastic changes in the auditory pathways induced by learning in children (Hayes et al., 2003;

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Kujala et al., 2001), adults (Kraus et al., 1995a; Tremblay and Kraus, 2002; Tremblay et al., 1997), and cochlear implant subjects (Jordan et al., 1997; Kaga et al., 1991). By studying subjects who have received an implant, we have a unique model for learning about the effects of profound deafness upon central auditory pathways and to investigate how evoked potentials can reflect performance on behavioral measures of auditory processing.

MLR evoked potential morphology, peak latencies, and absolute peak and interpeak amplitudes are similar in adult CI and normal hearing (NH) subjects (Makhdoum et al., 1998; Miyamoto, 1986; Pelizzone et al., 1989, 1991; Shallop et al., 1990). Groenen et al. (1997) found a relationship between MLR amplitude variation across electrode pairs and suprasegmental speech scores, and between MLR latency measures and segmental aspects of speech scores. Poorer implant users had more variable MLR amplitudes and latencies across electrode pairs than better performers. Groenen et al. (1997) concluded that MLR 'quality' was suggestive of subjective CI performance. In a later study, however, Makhdoum et al. (1998) found no significant correlations between the electrical MLR and speech scores.

CAEPs have been used more widely than the earlier EPs to investigate auditory function in CI users (Groenen et al., 1996b; Kileny et al., 1997; Makhdoum et al., 1998; Micco et al., 1995; Oviatt and Kileny, 1991; Pelizzone et al., 1989; Ponton et al., 1996a,b). Similar CAEP latencies have been reported for CI and NH subjects (Kileny et al., 1997; Micco et al., 1995; Oviatt and Kileny, 1991). Micco et al. (1995) found that N1 amplitude was significantly reduced in CI subjects. Makhdoum et al. (1998) found significant relationships between P2 latency (earlier latencies) and N1–P2 amplitude (greater amplitudes) and higher speech perception scores. Consistent with this, Groenen et al. (1996b) found that 'moderate' CI performers had reduced P2 amplitudes that were outside the normal range. Groenen et al. (1996b) postulated that this could be due to poorer cochleotopic organisation of the auditory cortex. Ponton et al. (1996b) noted that P1 amplitudes were larger in CI children than normal hearing children. They postulated that this may be due to the monaural stimulation provided by the implant leading to excitatory ipsilateral input to the auditory cortex which is greater than occurs with normal binaural stimulation (Kitzes, 1984; Popelar et al., 1994).

Several studies have used MMN to investigate electrophysiological correlates of performance in CI subjects (Ash and Shallop, 1995; Groenen et al., 1996a; Kileny et al., 1997; Kraus et al., 1993, 1995b; Ponton and Don, 1995; Wable et al., 2000). Kraus et al. (1993) conducted an early MMN investigation using speech stimuli in 8 'good' and one 'poor' adult cochlear implant users. Overall, MMN in the CI group was very similar to that found in the normal hearing control subjects. Kraus et al. (1993) concluded that, despite phonemes being processed very differently by the implant, their MMN results indicated that the brain processed the signals in a comparatively normal way in good CI users. Kraus et al. (1993) further concluded that, because of the objective nature of MMN recordings, MMN showed great promise as a tool to investigate the neurophysiological basis of speech discrimination abilities of CI users and was suitable for use with children because of the involuntary nature of the response. Groenen et al. (1996a) found a relationship between MMN elicited by speech phonemes and speech perception performance. Two out of the three 'good' users had a significant MMN that was very similar to the normal hearing control subjects whereas none of 7 moderate performers had a MMN.

Most studies investigating P3 in CI subjects have used an active listening paradigm that requires the subject to attend to the deviant stimuli, and therefore would be measuring P3b (Groenen et al., 1996b; Jordan et al., 1997; Kaga et al., 1991; Kileny, 1991; Mata et al., 1999; Micco et al., 1995; Okusa et al., 1999; Oviatt and Kileny, 1991). Oviatt and Kileny (1991) found a significant difference in P3 latencies between CI and normal hearing subjects for pure tone frequency contrasts (500 versus 1000 Hz, 500 versus 2000 Hz), but latencies were similar across groups for a very large frequency contrast (500 versus 3000 Hz). Kileny (1991) recorded P3 responses in 4 implanted children and found that P3 occurrence depended upon discrimination of the stimulus. A poor performing subject did not have a P3 (an indicator of discrimination), but did have N1–P2 (an indicator of detection). Micco et al. (1995) found that P3 responses to speech phonemes in a group of 'good' CI users were similar to those of normal hearing listeners. Micco et al. (1995) suggested that P3 may be useful in evaluating the cognitive aspects of central auditory processing in CI subjects. Kileny et al. (1997) investigated the relationship between MMN and P3 (recorded using a tonal frequency contrast) and speech scores in implanted children. Significant correlations were found between MMN latency, P3 latency and amplitude, and sentence scores with higher speech scores associated with shorter latencies and greater MMN and P3 amplitudes.

This review of evoked potential findings in CI subjects demonstrates a range of findings. Across studies there is considerable variation in subject numbers, the paradigms used (type of stimuli, active/passive task, mode of stimulus presentation), and the number of control subjects. No study has investigated the characteristics of MLR, CAEP, and MMN in the same group of subjects. The present study was therefore undertaken to investigate a comprehensive set of auditory evoked potentials (AEPs) in implanted subjects to determine those most closely related to implant performance.

2. Methods

2.1. Subjects

The experimental group (Table 1) consisted of 12 experienced adult CI users (mean age 50.3 years, SD 13.7,

Table 1
Details of the CI subjects showing age at implantation in years, duration of profound deafness in years (Dur), duration of implantation in years (Dur CI), number of active electrodes (No. of Elect), stimulation mode (Mode) (Bipolar +1 or Common Ground), and ear of implantation

Subject	Age	Sex	Aetiology	Dur	Dur CI	No. of Elect	Mode	Ear
1	47	F	Otosclerosis	2	3.7	20	bp+1	R
2	45	M	Progressive	5	1.9	14	cg	R
3	60	M	Progressive	6	1.9	20	bp+1	R
4	39	F	Progressive	1	1.7	20	bp+1	L
5	37	F	Progressive	1	5.2	20	bp+1	R
6	42	F	Progressive	2	2.6	20	bp+1	R
7	51	M	Otosclerosis	7	2.25	14	cg	R
8	69	M	Progressive	2	5.2	20	bp+1	L
9	61	F	Progressive	7	1.8	16	bp+1	R
10	27	F	Progressive	9	4.2	20	bp+1	R
11	52	F	Progressive	10	1.3	20	bp+1	R
12	74	M	Progressive	15	2.9	20	bp+1	R

range 27–74 years). The implant subjects had a minimum of 1.3 years CI experience (mean 2.9 years, SD 1.4, range 1.3–5.2 years) and the duration of profound deafness prior to receiving the implant ranged from 1 to 15 years (mean 5.5 years, SD 4.4). All CI subjects used the Nucleus® CI-22M implant and the SPEAK processing strategy. The SPEAK processing strategy uses a stimulation rate of 250 Hz and selects between 6 and 9 maxima from 20 frequency bands. Electrodes corresponding to the frequency band maxima are stimulated from base to apex. All CI subjects were post-lingually deafened and had intelligible speech.

Twelve age-matched (mean 50.2 years, SD 13.9) and sex-matched subjects were used as NH control subjects for the evoked potential section of this study. The test ear used for AEP recordings was the same as the implanted side of the matched CI subject.

2.2. Speech perception

Speech perception was evaluated using HINT© sentence lists (Nilsson et al., 1994) and CNC word lists (Peterson and Lehiste, 1962) which were re-recorded onto a video cassette using a male New Zealand speaker. Speech materials were delivered at 70 dB SPL auditory-alone through a loudspeaker placed at ear level, 50 cm from the implant side at 90° azimuth. Subjects verbally repeated what they heard and received no feedback for right or wrong answers.

2.3. Auditory evoked potentials

Three types of AEPs were recorded in all subjects: (1) the MLR to a tonal stimulus, (2) CAEP responses to 3 pure tone frequencies, and (3) the event related responses, MMN and P3a recorded passively to frequency differences.

The Neuroscan STIM™ system was used to generate acoustic stimuli. A loudspeaker placed at ear level, 50 cm from the implant side at 90° azimuth was used for the CI subjects. For the NH subjects an insert earphone (E-A-R Tone™ 3A) was used to deliver stimuli. The Neuroscan SCAN™ system interfaced with Grass® Model 12A5 amplifiers was used for recording evoked potentials. Custom built radio-frequency filters were placed on the front end of each Grass® amplifier to reduce the CI radio-frequency stimulus artefact. All recordings were made with the Grass® amplifier in AC mode.

AEP testing was conducted within one session in the following order: MMN was recorded first to ensure subjects were alert, followed by MLR, and finally CAEP recordings. Seven Ag–AgCl electrodes were placed at Cz, Fz, Pz, Fpz, the contralateral earlobe, and at the lateral canthus and below the eye opposite to the implant/test ear side. Impedances were kept below 3 kΩ. Subjects were comfortably seated in a reclining chair and watched silent sub-titled videos. They were encouraged to remain alert and to stay awake. Prior to the recording session listening levels for the AEP stimuli were set to a 'loud, but OK' level for individual subjects using the Independent Hearing Aid Fitting Forum (IHAFF) Contour Test 7 point rating scale (Valente and Van Vliet, 1997). Stimulus sound pressure levels ranged from 83 to 113 dB ppeSPL. For MMN recordings the levels for the deviant stimuli were the same as for the frequent stimuli.

Stimulus and recording parameters are listed in Table 2. All AEP responses were amplified with a gain of 50,000 and sampled at 2048 Hz. All recordings were baseline corrected and linear detrended prior to peak identification. Absolute amplitudes were used for all analyses. For MLR and CAEP recordings non-inverting electrodes were placed at Fz and Cz. These were referenced to the contralateral earlobe to minimize CI stimulus artefact. MLR responses were digitally low pass filtered at 200 Hz (12 dB/octave) post hoc to aid in peak identification. The artefact reject level was ±50 μV. The time window was 80 ms, with a 3 ms pre-stimulus baseline. For CAEP recordings the responses were post hoc digitally low pass filtered at 30 Hz (12 dB/octave). Artefact reject levels were 50 μV. The time window was 400 ms, with a 100 ms pre-stimulus baseline.

For MMN/P3a an oddball paradigm was used to present the stimuli. There were 1000 stimulus presentations in pseudo-random order. The first 20 stimulus presentations were all frequent stimuli in order to build a strong memory

Table 2
AEP stimulus and recording parameters: stimulus duration (ms), rise and fall times (ms), frequency (Hz) (dev, deviant stimulus), rate (ms) or ISI (inter-stimulus interval, ms), number of averages, and filter settings

AEP	Duration (ms)	Rise/fall (ms)	Frequency (Hz)	Rate (per second) or ISI (ms)	Number of averages	Filter (Hz)
MLR	60	5	500	1.1/s	2×250	3–300
CAEP	60	20	250, 1000, 4000	1.1/s	2×100	1–100
MMN and P3a		20	Frequent=1000 Small dev=1250 Large dev=1500	600 ms	800 (80%) 100 (10%) 100 (10%)	0.1–100

trace of this stimulus (Schroger, 1998). There was a minimum of 3 standard stimuli between each deviant stimulus. A 500 ms recording time window was used that included a 100 ms pre-stimulus baseline. Non-inverting electrodes were placed at the midline sites Fz, Cz, and Pz, referenced to the contralateral earlobe. Eyeblink activity was monitored in one recording channel by the placement of electrodes at the lateral canthus and below the eye opposite to the implant side. Prior to testing, the CI subjects' frequency allocation tables were checked to ensure standard and deviant tone bursts stimulated different electrodes. All CI subjects could behaviorally discriminate the standard and deviant stimuli (determined psychophysically using a two-alternative forced-choice task). The artefact reject level was set at ± 75 μV to ensure that all eyeblinks were rejected. MMN was judged to be present if there was an area of negativity present on the deviant waveform caused by the deviant waveform crossing the frequent waveform at a latency no earlier than the latency of the N1 cortical peak for the individual subject.

Repeated measures ANOVAs were performed to determine whether group and montage effects were significant for MLR, CAEP, MMN, and P3a responses. Separate repeated measures ANOVAs were performed for each type of evoked potential, with montage, deviant type and the waveform parameter (latencies, amplitudes, etc.) as repeated measures. A statistical significance level of 0.05 was used for all analyses. *F* statistics and *P* values are shown for all significant findings.

3. Results

3.1. Speech perception

Mean speech perception scores of the CI group were 69.2% for HINT sentences (SD=32.3), 60.8% for CNC phonemes (SD=18.8), and 37.0% for CNC words (SD=21.5). As shown in Fig. 1, the CI subjects could be separated into two groups based on their speech perception scores.

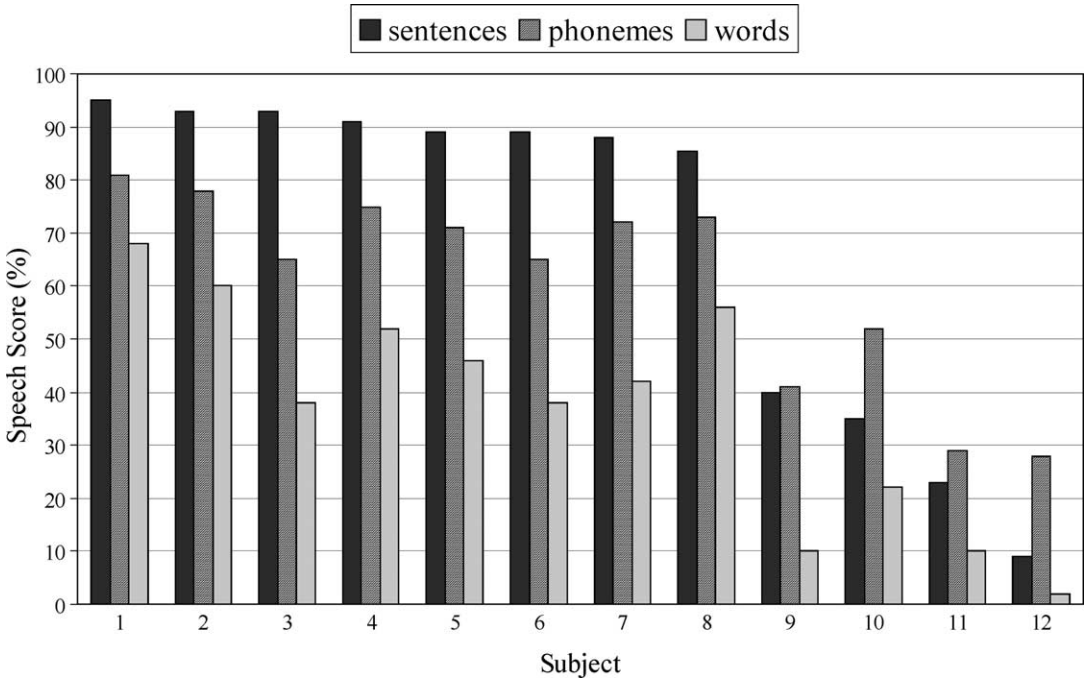

Fig. 1. Individual speech recognition scores (% correct) for HINT sentences and CNC words (word and phoneme scores) rank ordered from best to worst sentence score for 12 CI subjects. The subjects can be divided into 8 'better' (>85%) users (subjects 1–8) and 4 'poorer' (<40%) users (subjects 9–12) based on their HINT sentence scores.

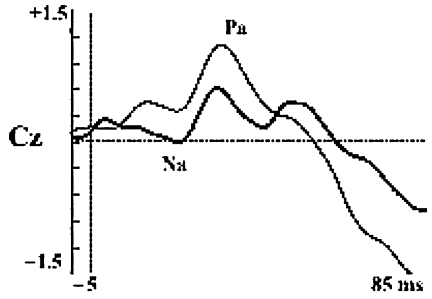

Fig. 2. Grand mean MLR waveforms ($n=12$ per group) for the CI (thick line) and NH (thin line) groups for the Cz electrode montage. Vertical scale is in microvolts.

Eight subjects who had scores greater than 85% on HINT sentences, over 60% for CNC phonemes, and over 35% for CNC whole words were classified as 'better' users (subjects 1–8). The other 4 subjects were classified as 'poorer' users with scores less then 40% for CNC words and HINT sentences (subjects 9–12). Poorer subjects had been profoundly deaf for longer prior to implantation. The mean duration of profound deafness for the better group was 3.1 years (SD = 2.5 years) and the mean duration of implant use was 3.1 years (SD = 1.5 years). In contrast, the mean duration of profound deafness for the poorer CI group was 10.3 years (SD = 3.4 years), but they had a similar mean duration of implant use (2.6 years, SD = 1.3 years).

There were significant negative correlations between duration of profound deafness and HINT sentence scores ($r=-0.853$, $P<0.001$), CNC phoneme scores ($r=-0.825$, $P=0.001$), and CNC word scores ($r=-0.809$, $P=0.001$). Thus poorer speech recognition scores were associated with longer duration of profound deafness.

3.2. Middle latency response

The grand mean waveforms for the CI and NH groups recorded at Cz are illustrated in Fig. 2. There were no significant group or montage effects on MLR latencies or amplitudes. A RMS power analysis performed on the Cz data across the latency range 15–60 ms showed no significant group difference.

3.3. Cortical responses

The CAEP responses had well-defined morphology for both subject groups (see Fig. 3). N1 amplitude was significantly greater at Cz than Fz [$F(1,22)=7.752$, $P=0.011$]. There was a significant frequency by group interaction for N1 amplitude [$F(2,21)=4.944$, $P=0.017$]. Fig. 4 shows that N1 amplitude reduced with increasing frequency from 250 Hz to 4 kHz in CI subjects, whereas N1 was the greatest at 1 kHz in the NH group. N1 was much larger in the NH group compared to the CI group at 1 and 4 kHz. As for N1 amplitude, P1 amplitude in the CI group was largest at 250 Hz. P1 amplitude was smaller at 4 kHz in the CI group than in the NH group. At 1 kHz P1 amplitudes were similar for the two groups.

3.4. Mismatch negativity and P3a

All NH subjects had MMN to both deviant stimuli. In contrast, only 75 and 58% of the CI subjects had MMN to large and small deviants, respectively. The pattern of responses varied across montage, with larger MMN amplitude and area and larger P3a amplitude at Fz. There was a trend for the CI subjects with poorer speech scores to have either a greatly reduced MMN than the better performers or no identifiable MMN (Fig. 5).

To compare NH versus CI groups analysis of the MMN and P3a data was performed separately for each deviant stimulus at each electrode montage. The following measures were analyzed: MMN and P3a peak latency and amplitude; MMN onset latency and amplitude, offset latency and amplitude, duration (offset–onset), area (duration × amplitude measured using the Neuroscan area algorithm). MMN onset was identified using the difference waveform (standard-deviant) and was defined as the point at which the deviant waveform became consistently more negative than the standard waveform within the latency range 50–150 ms. MMN offset was defined as the point at which the deviant and standard waveforms converged within the latency range 150–350 ms. Means and standard deviations of MMN parameters are listed in Table 3. Significant group effects were found at Fz only. For the large deviant MMN

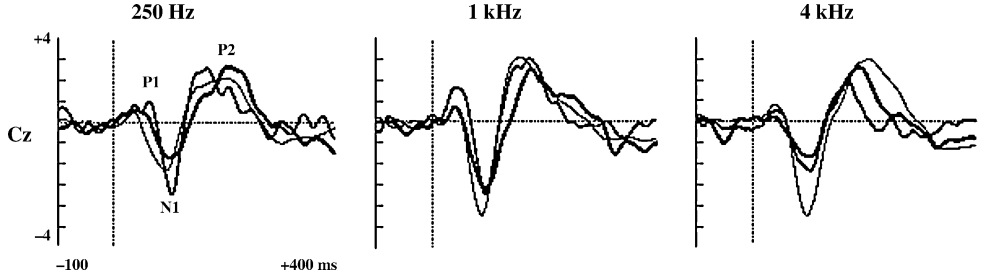

Fig. 3. Grand mean CAEP waveforms for 250 Hz, 1 and 4 kHz stimuli for the better CI (thick line), poorer CI (medium line), and NH (thin line) groups at the Cz electrode montage. Vertical scale is in microvolts.

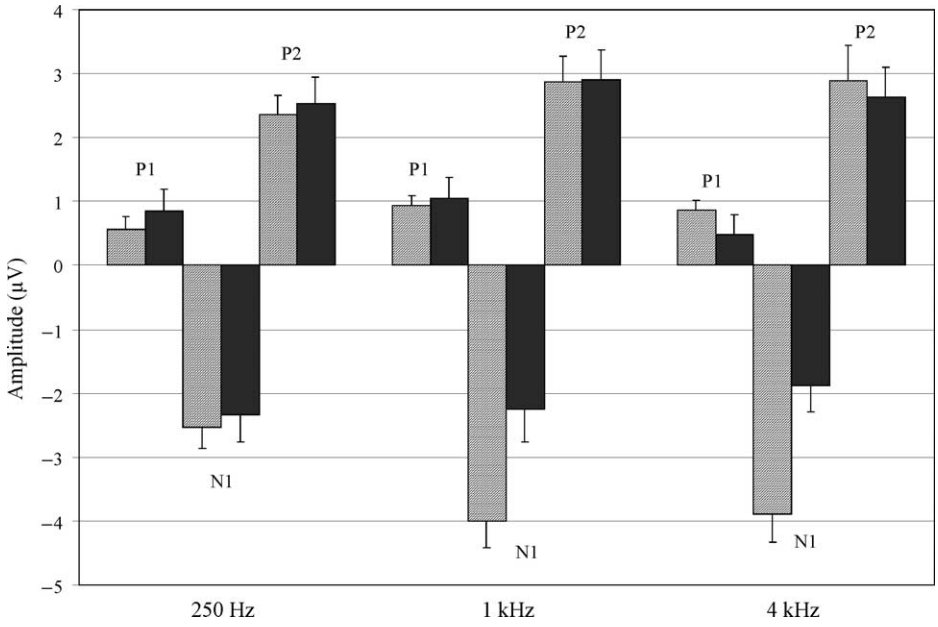

Fig. 4. Mean CAEP P1, N1, and P2 amplitudes (μV) for the NH group (striped) and CI group (solid) for 3 stimulus frequencies (250 Hz, 1 and 4 kHz) at the Cz electrode montage. Error bars show standard errors of the mean.

duration at Fz was significantly shorter [$F(1,19)=11.027$, $P=0.004$] and onset latency was significantly later [$F(1,19)=6.749$, $P=0.018$] in CI subjects. There were no MMN group effects for the small deviant stimulus. P3a amplitudes were significantly larger in the CI group for both small [$F(1,15)=5.873$, $P=0.028$] and large [$F(1,18)=5.861$, $P=0.026$] deviants at Fz.

3.5. Objective versus subjective measures of implant performance

Bivariate correlations were performed to investigate possible relationships between the electrophysiological measures of implant performance in the CI group and speech scores. The effects of subject characteristics (duration of profound deafness, duration of implant use, and age) on evoked potentials were also investigated. A large number of correlations were calculated and no corrections were made for multiple comparisons, hence significant findings should be interpreted cautiously. Table 4 lists the Pearson's r and P values for the correlations with $P<0.05$.

Na amplitude at Cz was correlated with duration of deafness, and Pb amplitude was correlated with age. Na amplitudes decreased with increasing duration of deafness, and Pb amplitude increased with subject age, as reported by Amenedo and Díaz (1998) and Pfefferbaum et al. (1979).

The correlation analysis of the CAEP data was performed separately for each stimulus frequency. For 250 Hz stimulus, significant negative correlations were found between duration of CI use and N1 latency and P1 amplitude at Cz. N1 was earlier and P1 was smaller in subjects who had used an implant longer, indicating a change in cortical response morphology associated with implant use. P2 latency at Cz was correlated with duration of deafness and negatively correlated with speech scores, consistent with Makhdoum et al. (1998). Thus shorter P2 latencies were associated with higher speech scores and shorter durations of deafness.

There was only one significant correlation for 1 kHz stimulus, between N1 amplitude and CI use. N1 amplitude was larger with greater CI use. No significant correlations were found for 4 kHz stimulus.

3.6. MMN and P3a

For the large deviant stimulus MMN duration and speech scores were correlated. Subjects with better speech scores had greater MMN duration. For the large deviant there was a correlation between P3a amplitude and duration of CI use, with greater amplitudes being found with longer CI use. For the small deviant stimulus P3a latency was also correlated with duration of CI use. Overall, P3a amplitude was larger and P3a latency was shorter in subjects using an implant longer.

4. Discussion

The present study was undertaken to investigate the characteristics of electrophysiological measures of auditory processing in adult experienced CI users and to compare these to speech perception measures. The MLR results were consistent with the findings of Gardi (1985) who reported

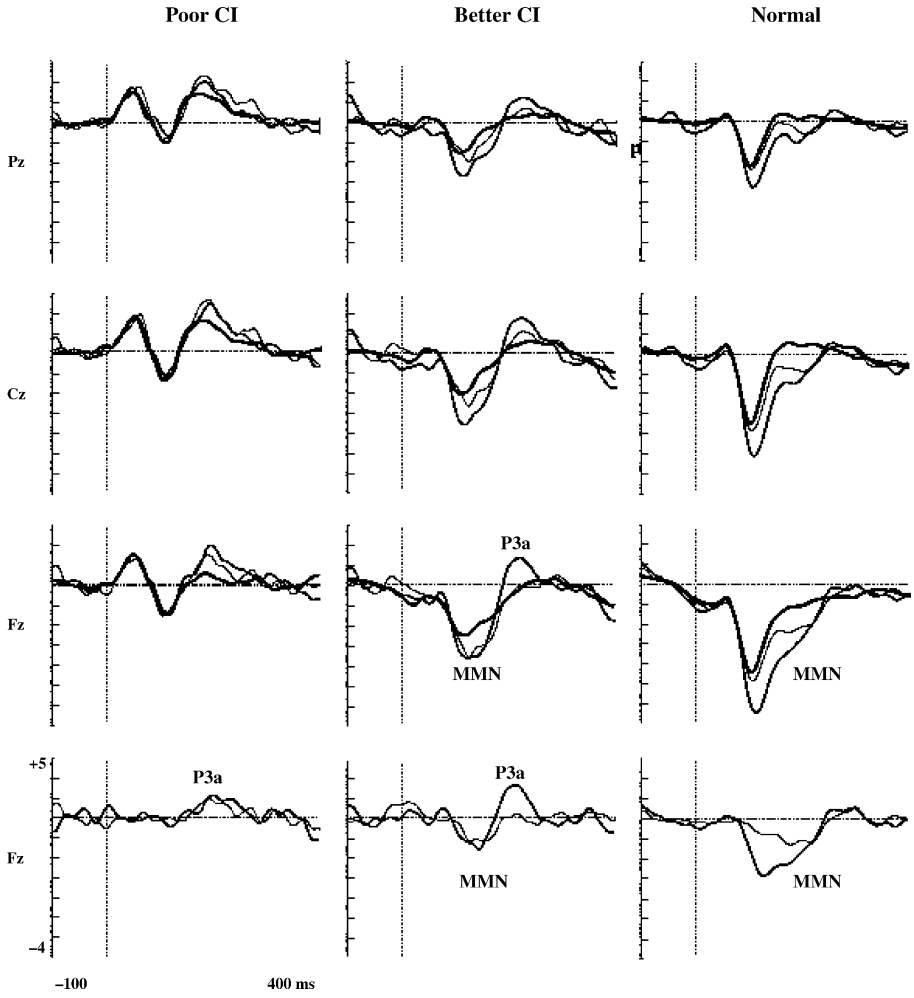

Fig. 5. Top 3 rows show MMN grand mean waveforms at Pz, Cz, and Fz for (a) NH group ($n=12$), (b) better CI group ($n=8$), and (c) poorer CI group ($n=4$). The frequent waveform (1000 Hz, thick line), small deviant waveform (1250 Hz, thin line) and large deviant waveform (1500 Hz, medium line) are shown. Bottom row shows the corresponding MMN grand mean difference waveforms for the small deviant (thin line) and large deviant (thicker line) stimuli. Vertical scale is in microvolts.

similar latencies and amplitudes in NH and CI subjects. The MLR showed considerable inter-subject variability in both NH and CI groups and there was no relationship between the MLR and CI performance. In contrast, obligatory and discriminative cortical response latencies and amplitudes differed significantly between the groups and poorer responses were associated with poorer CI performance. Poorer CI subjects had either absent or small amplitude MMN. Surprisingly, the majority of the CI subjects had a robust P3a response.

4.1. Speech perception

The speech results indicate that duration of deafness is critical in determining successful speech perception outcomes with an implant. This is consistent with many studies in the implant literature showing that duration of profound deafness prior to implantation is predictive of implant outcome (Blamey et al., 1992; Gantz et al., 1993; Shipp and Nedzelski, 1994; Shepherd et al., 1997; van Dijk et al., 1999).

Table 3
Means and standard deviations of MMN onset latency and amplitude, peak latency and amplitude, offset latency and amplitude, and area. Standard deviations are shown in parentheses

		Onset latency (ms)	Onset amplitude (μV)	Peak latency (ms)	Peak amplitude (μV)	Offset latency (ms)	Offset amplitude (μV)	Area (ms μV)
Large deviant	NH	84.77 (26.91)	−0.22 (0.34)	162.30 (36.16)	−30.05 (10.64)	243.85 (46.70)	−0.05 (0.06)	1.66 (0.84)
	CI	148.92 (84.36)	−0.17 (0.52)	197.85 (83.35)	−20.66 (10.51)	243.85 (46.70)	0.14 (0.26)	1.46 (0.77)
Small deviant	NH	120.12 (47.12)	−0.34 (0.60)	170.02 (43.44)	−20.09 (10.15)	231.44 (32.48)	−0.23 (0.39)	1.24 (0.66)
	CI	123.24 (51.98)	−0.36 (0.38)	169.14 (39.75)	−20.60 (10.46)	250.39 (48.46)	−0.23 (0.39)	1.57 (1.06)

Table 4
Significant correlations ($P<0.05$) between the evoked potential measures and subject characteristics and speech scores. Correlations were calculated using Cz recordings for MLR and CAEP and Fz recordings for MMN and P3a

	Measure	Duration deafness	Duration of CI use	Age	Speech scores (% phonemes)
MLR	Na ampl	$r=-0.678$ $P=0.015$			
	Pb ampl			$r=-0.683$ $P=0.014$	
250 Hz CAEP	P1 ampl		$r=-0.634$ $P=0.027$		
	N1 lat		$r=-0.712$ $P=0.009$		
	P2 lat	$r=0.664$ $P=0.019$			$r=-0.629$ $P=0.029$
1 kHz CAEP	N1 ampl		$r=0.689$ $P=0.013$		
MMN	Duration (large dev)				$r=0.681$ $P=0.044$
P3a	Ampl (large dev)		$r=0.788$ $P=0.002$		
	Lat (small dev)		$r=-0.873$ $P=0.023$		

4.2. Electrophysiological measures

P1 amplitudes were negatively correlated and N1 amplitudes were positively correlated with duration of CI use. This is consistent with Tremblay and Kraus's (2002) recent report of decreased P1 and increased N1 amplitudes in normal-hearing adult listeners receiving auditory training. Thus, the changes in auditory pathways associated with implant use in adults with acquired profound deafness seem to parallel the changes that occur with auditory experience in normal hearing adults. P1 amplitude decreases and N1 amplitude increases with CI experience may be manifestations of the same phenomenon. Since P1 and N1 are phase opposite and partially temporally overlap, an increase in N1 amplitude will likely result in a decrease in P1 amplitude by phase cancellation (Ponton and Eggermont, 2001).

Tremblay and Kraus (2002) found differences in auditory training effects for midline versus lateral electrode montages. Auditory experience with an implant also seems to change the scalp distribution of auditory activity, as Ponton et al. (1999) found reduced cortical response hemispheric asymmetry for adult CI users compared to normal listeners. One limitation of the current study is that only midline recordings were made and differences in hemispheric responses may have been missed.

Previous studies have reported significant frequency effects on cortical response latencies and amplitudes (Jacobson et al., 1992; Rapin et al., 1966; Verkindt et al., 1995). In NH listeners N1 was larger at 1 kHz than at 250 Hz, consistent with the findings of Rapin et al. (1966). In contrast, N1 was largest at 250 Hz and declined with frequency in the CI group. This trend for poorer high frequency cortical responses in the CI group was also evident when the CI subjects were divided into 'better' and 'poorer' CI users, with the poorer group having smaller cortical responses than the better users. Micco et al. (1995) also reported smaller than normal N1 amplitudes in CI users.

The pattern of delayed P1 latencies and greater P1 amplitudes in the CI group is consistent with Ponton and colleagues' finding that maturation of cortical responses in profoundly deaf children receiving an implant may not match that of children with normal hearing. Later-implanted CI children have later and larger P1 responses than their NH peers (Eggermont et al., 1997; Ponton et al., 1996a,b, 1999, 2000). These studies have shown that the relationship between the period of prolonged deafness, chronological age prior to receiving the implant, and the period of time since receiving the implant will determine how 'normal' the cortical responses become.

The similarity between CI and NH cortical responses at 250 Hz may reflect the typical pattern of better residual low frequency and poorer high frequency hearing prior to cochlear implantation in individuals with profound hearing loss, and suggests equal activation levels. For both CI and NH listeners, tones were presented at a 'loud, but OK' level which corresponded to levels in excess of 80 dB SPL for all subjects. At these levels cortical response amplitudes should be saturated or almost saturated (Naatanen and Picton, 1987). It is difficult to be certain that presentation levels were equated for the CI and NH subjects, but the high presentation levels would have produced high levels of neural excitation in all subjects.

Profound deafness is generally associated with greater high frequency loss, and it is not usually possible to adequately amplify high frequencies (including 4 kHz) into the speech spectrum (Snik et al., 1995). Thus poor high frequency cortical responses in the CI subjects may be due

to inadequate peripheral stimulation over a period of many years prior to implantation. Expanded low frequency representation in the auditory cortex occurs with peripheral damage to high frequency regions (Harrison et al., 1991; Reale et al., 1987; Recanzone et al., 1993; Robertson and Irvine, 1989). Despite high frequency hearing being restored by the implant, the CI cortical responses were still poorer than those of their age-matched peers. The 4 kHz data indicate that high frequency stimulation provided by the implant had not resulted in a full recovery of central auditory function, despite hearing thresholds with the implant being near-normal at this frequency.

The two-deviant oddball paradigm was successful in demonstrating electrophysiological differences between the better and poorer CI groups, a difference that was also found in the speech perception results. In general the CI subjects with poorer speech scores had small or absent MMN to the smaller deviant stimulus. Several studies using either tonal or speech stimuli have also found no measurable MMN in poorer performing CI subjects (Ash and Shallop, 1995; Groenen et al., 1996a; Kraus et al., 1993). Similarly, Beynon et al. (2002) found that P3 measured using an active listening paradigm was significantly later in implanted children with poorer speech perception scores. The expected effect of prolonged latency and reduced MMN amplitude with more difficult discrimination tasks occurred for both NH and CI groups, with MMN to the small deviant stimulus being smaller and later than for the large deviant stimulus (Naatanen, 1992; Sams et al., 1985).

The increase in MMN amplitude, area, and duration for both NH and CI groups at Fz is consistent with previous studies which have shown larger MMN at frontal electrodes (e.g. Jacobsen et al., 2004; Naatanen, 1992). It has been proposed that MMN is associated with involuntary orienting of attention following the detection of stimulus change, and that this orienting of attention is mediated by prefrontal motor areas (Alho et al., 1994; Giard et al., 1990; Naatanen, 1990, 1992).

The presence of a robust P3a, especially for the large deviant, in the poorer CI group without the presence of MMN suggests separate generator systems for these two responses (Naatanen et al., 1982; Paavilainen et al., 1991; Sams et al., 1985). The NH group results are also consistent with separate generators since the NH group had substantial MMN without a large P3a response, even for the large deviant stimulus, as has been found previously in NH listeners (Naatanen et al., 1980, 1982; Sams et al., 1984). Another possible explanation, suggested by Wunderlich and Cone-Wesson (2001), is that the negative MMN and positive P3a have a phase-cancelling effect, with a large P3a being most evident on waveforms where MMN is not present. However, as illustrated in Fig. 5(b), some subjects had a robust MMN and P3a for the same stimulus which suggests that the presence of a large P3a in some CI subjects was not simply due to MMN absence.

Kaga et al. (1991) reported small P3 amplitudes initially in a single CI subject that increased in size in the following 3 months as discrimination ability improved. In the present study duration of implant use and P3a amplitude were correlated, with P3a amplitude increasing as duration of implant use increased, consistent with Kaga et al.'s (1991) results. Thus, although the present study used a passive listening paradigm there are some similarities with previously reported P3 results obtained using an active stimulus paradigm.

P3a amplitudes were greater at Fz and with larger deviance, consistent with previous studies (Comerchero and Polich, 1998; Ivey and Schmidt, 1993; Katayama and Polich, 1998; Polich, 1998). The CI responses were unusual, however, in that a robust P3a was recorded from both the poorer and better CI subjects without a preceding MMN response in some instances. There are no previous reports of an enhanced P3a in CI subjects. This is an interesting finding in the light of some evidence from the psychiatric literature that P3 reflects the sensory gating abilities of an individual (Bender et al., 1999; Schall and Ward, 1996). The scalp distribution of P3a is predominantly in the frontal areas of the cortex (Polich, 1989; Squires et al., 1975). The prefrontal cortex provides both inhibitory and excitatory input to distributed neural circuits required to support performance in diverse tasks. Prefrontal damage can disrupt the inhibitory modulation of inputs to primary sensory cortex, perhaps through abnormalities in a prefrontal-thalamic sensory gating system (Kaas and Hackett, 1998). The P3a results suggest that an investigation of sensory gating using the more usual sensory gating paradigm of P1 suppression (e.g. Boutros and Belger, 1999) may be an interesting area of future research in adults with cochlear implants.

4.3. Electrophysiological versus speech perception results

The finding of significant correlations between P2 latencies and speech recognition scores is consistent with Makhdoum et al. (1998) who also found that P2 latency was strongly correlated to spondee speech perception score in experienced CI users. P2 appears to reach adult latency and amplitude values by about age 5 years (Ponton et al., 2000). All the CI subjects in the present study were post-linguistically deafened or had progressive losses and it is likely that they would all have had mature P2 responses (at least to low frequency stimuli) from an early age. Therefore, the relationship between P2 latency and speech scores should reflect changes in central auditory function due to the CI subjects' hearing deteriorating prior to receiving an implant and improvements after implantation.

5. Conclusions

The general similarity of MLR and CAEP responses in the CI and NH subjects indicates that central auditory

processing up to the level of the auditory cortex is relatively intact in post-linguistically deafened adults who have some implant experience, despite the abnormal peripheral stimulation provided by the implant. Speech perception correlated significantly with the cortical P2 responses. This, combined with the ease of recording and robustness of cortical responses, suggests that CAEP responses may be useful in both clinical and research applications as a predictor and objective indicator of implant performance. The MMN results support previous studies showing that MMN is a useful tool for objectively investigating auditory discrimination in CI subjects. The results indicate some limitations to the usefulness of MMN, however. Although all of the subjects, both NH and CI, could detect the differences between the standard and deviant tones, not all subjects had a measurable MMN. Previous studies have also reported absent MMN for stimulus contrasts that are clearly discriminated behaviorally (e.g. Dalebout and Stack, 1999). Analyzing a range of electrophysiological measures in the same subjects reveals that the major effects are occurring at the cortical level. Furthermore, these effects correlate with speech perception ability and duration of deafness. This is consistent with animal studies of plasticity after cochlear lesions which show greater cortical than sub-cortical reorganization in mature animals (Harrison, 2001). The presence of P3a in many CI subjects possibly reflects their need to devote greater attentional resources to processing auditory information.

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