Neurophysiology of Cochlear Implant Users I: Effects of Stimulus Current Level and Electrode Site on the Electrical ABR, MLR, and N1-P2 Response

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Objective: **As the need for objective measures with cochlear implant users increases, it is critical to understand how electrical potentials behave when stimulus parameters are systematically varied. The purpose of this study was to record and evaluate the effects of implanted electrode site and stimulus current level on latency, amplitude, and threshold measures of electrically evoked auditory potentials, representing brainstem and cortical levels of the auditory system.**

Design: **The electrical auditory brainstem response (EABR), electrical auditory middle latency response (EAMLR), and the electrical late auditory response (ELAR) were recorded from the same experimental subjects, 11 adult Clarion cochlear implant users. The Waves II, III, and V of the EABR, the Na-Pa complex of the EAMLR and the N1-P2 complex of the ELAR were investigated relative to electrode site (along the intra-cochlear electrode array) and stimulus current level. Evoked potential measures were examined for statistical significance using analysis of variance (ANOVA) for repeated measures.**

Results: **For the EABR, Wave V latency was significantly longer for the basal electrode (7) compared with the mid (4) and apical (1) electrodes. For the EAMLR and ELAR, there were no significant differences in latency by electrode site. For all subjects and each of the evoked potentials, the apical electrodes tended to have the largest amplitude and the basal electrodes the smallest amplitude, although amplitude differences did not reach statistical significance. In general, decreases in stimulus current level resulted in statistically significant decreases in the amplitude of Wave V, Na-Pa and N1-P2. The evoked potential thresholds for Wave V, Na-Pa, and N1-P2 were significantly higher for the basal Electrode 7 than for Electrodes 4 and 1.**

Conclusions: **Electrophysiologic responses of Waves II, III, and V of the EABR, Na-Pa of the EAMLR, and N1-P2 of the ELAR were characterized as functions of current level and electrode site. Data from this study may serve as a normative reference for expected latency, amplitude and threshold values for the recording of electrically evoked auditory brainstem and cortical potentials. Responses recorded from cochlear implant users show many similar patterns, yet important distinctions, compared with auditory potentials elicited with acoustic signals.**

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Electrically evoked auditory potentials have been recorded through a variety of implanted electrode arrays. These potentials include the electrical auditory brainstem response (EABR), the electrical auditory middle latency response (EAMLR) and electrical late auditory responses (ELAR). The earliest studies used experimental devices in deaf adult subjects (Gardi, 1985; Starr & Brackmann, 1979) and were fraught with technical recording difficulties due to increased stimulus artifact produced by electrical stimulation. Later studies included FDA approved and clinical-trial devices such as the 3M single-channel device (Miyamoto, 1986), the Ineraid system (Abbas & Brown, 1988, 1991), the Nucleus device (Abbas & Brown, 1991; Brown, Abbas, Fryauf-Bertschy, Kelsay, & Gantz, 1994; Hodges, Ruth, Lambert, & Balkany, 1994; Mason, Sheppard, Garnham, Lutman, O'Donoghue, & Gibbin, 1993; Shallop, Beiter, Goin, & Mischke, 1990; Shallop, Van Dyke, Goin, & Mischke, 1991), the Clarion implant (Brown, Hughes, Lopez, & Abbas, 1999; Firszt, Rotz, Chambers, & Novak, 1999) and the Med-El system (Firszt, Gaggl, Wackym, & Reeder, Reference Note 1).

Results of EABR measures have not been reported consistently in published studies. Thresholds, amplitudes and waveform morphologies have differed across and within subjects for different electrodes (Gardi, 1985; Hodges, et al., 1994; Shallop et al., 1990). Some animal studies show a relation between amplitude growth functions and surviving ganglion cells (Simmons & Smith, 1983; Walsh & Leake-Jones, 1982), whereas other investigations

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have not confirmed these findings (van den Honert & Stypulkowski, 1986).

The EAMLR is an attractive alternative to shorter latency potentials. It has a longer latency (positive peak at 27 to 37 msec), making it less likely to be contaminated by the stimulus artifact that occurs early in the response. Recordings in animals and humans indicate that it is reliable (Burton, Miller, & Kileny, 1989; Gardi, 1985; Groenen, Snik & van den Broek, 1997; Kileny, Kemink, & Miller, 1989; Miyamoto, 1986) and may correlate with neural survival (Jyung, Miller, & Cannon, 1989). One study of the electrical and acoustic MLR within the same subject suggests that both responses are activated by the same neural generators of the central system (Kileny et al., 1989). In general, there are few published studies of the EAMLR in cochlear implant users.

Reports of late auditory potentials evoked with electrical stimulation have included recordings of the N1, P2, P300, and mismatch negativity (MMN) response elicited with pulsed tones (Oviatt & Kileny, 1991), stimulated electrode pairs (Makhdoum, Groenen, Snik, & van den Broek, 1997; Ponton & Don, 1995), and speech (Kaga, Kodera, Hirota, & Tsuzuka, 1991; Kraus et al., 1993; Micco et al., 1995). Results suggest that cortical responses provide a mechanism for understanding how electrical stimuli are registered by the central auditory system.

The need for objective measures with cochlear implant users has increased due to unexplained variation in patient performance, implantation of children at younger ages, and an increase in programming options. With this in mind, it is critical to understand how electrical potentials behave when stimulus parameters are systematically varied. It is often difficult to compare published findings of early, middle, and late-latency potentials across studies due to differences in electrode arrays (longitudinal, radial), stimulation mode (bipolar, monopolar), time and location of recordings (operating room, clinic, intra-operative, postoperative), subjects (pediatric, adult) and stimulus and recording parameters. There are no published studies that characterize the latency, amplitude and threshold for EABR, EAMLR, and ELAR recorded across the electrical dynamic range from the same experimental cochlear implant subjects.

The goals of this study were 1) to record Wave V of the ABR, the Na-Pa complex of the MLR and the cortical N1-P2 complex from intracochlear electrodes, and 2) to evaluate the effects of implanted electrode site and stimulus level on electrically evoked auditory potentials that represent the brainstem and cortical levels of the auditory system from the same subject sample. Specifically, the EABR, EAMLR, and ELAR were recorded for individual subjects from three electrodes that represent basal, mid, and apical positions along the Clarion electrode array. Latency, amplitude, general morphology, and threshold were analyzed across stimulus level and electrode site within subjects for each evoked potential. In a companion paper in this issue, the analysis of evoked potential measures in relation to speech perception performance for this subject sample is presented (Firszt, Chambers, & Kraus, 2002).

METHODS AND PROCEDURES

Subjects

Eleven adults (five women and six men) who received the Clarion 1.2 radial electrode array participated in the study. All subjects had full electrode insertions, used monopolar stimulation of the medial electrode contacts, and used the continuous interleaved sampler speech-processing strategy. At the time of study, the subjects ranged in age from 29 to 75 yr, with a mean of 56 yr. At the time of cochlear implantation, the subjects ranged in age from 24 to 70 yr, with a mean of 53 yr. Subjects had used their cochlear implants for at least 3 mo, with a maximum length of use of 5 yr and a mean across subjects of 2.7 yr. Subjects were full-time users of their cochlear implants with a range of daily wear between 10 to 15 hr. Table 1 provides background information for each subject.

General Procedures

The EABR, EAMLR, and ELAR were recorded on Electrode 1 (apical), Electrode 4 (mid), and Electrode 7 (basal) within the cochlea. Before recording these potentials, behavioral data were collected that defined the subject's electrical threshold and dynamic range (number of electrical steps between threshold and upper limit of comfortable loudness) on each tested electrode. Because subjects were evaluated while awake in the clinic, it was important that stimuli were comfortably loud. Electrical stimulus levels within the subject's behavioral dynamic range (BDR) were presented for each electrode and response type (e.g., EABR, EAMLR, ELAR).

Behavioral Measures

Stimuli, Equipment, and Procedures • Stimuli for behavioral measures were biphasic current pulse trains generated by a computer-controlled interface unit and SCLIN for Windows software version 1.2. Pulses were 75 μsec in duration and negative-leading in polarity. Stimulus amplitude was expressed in clinical units (CU) relative to microamperes (μA)

TABLE 1. Demographic information on subjects.

Subject	Ear	AAO/HL-Degree	AAO/PHL	AAI	LOU-CI	AAT	Etiology	Onset
1	R	32-Mild-Moderate	50	53	2-2	55	Inherited	Progressive
	L	32-Mild-Moderate	45					
2	**R**	38-Moderate	47	52	3-5	55	RE possible Meniere's; LE congenital unknown	Progressive
	L	5-Severe/Profound	5					
3	R	17-Mild	44	46	3-0	49	Unknown	Progressive
	L	18-Mild	38					
4	R	18-Moderate	40	64	4-0	68	Possible cochlear otosclerosis	Progressive
	L	18-Moderate	40					
5	**R**	20-Mild	64	64	1-0	65	Unknown	Progressive
	L	20-Severe	64					
6	**R**	61-Severe	61	67	3-0	70	RE unknown; LE chronic suppurative otitis media	Sudden
	L	18-Severe	61					
7	**R**	30-Moderate	31	32	1-6	33	Autoimmune inner ear disease	Sudden
	L	30-Moderate	31					
8	**R**	3-Severe	10	24	5-0	29	Congenital hereditary (twin sister w/ HL)	Progressive
	L	3-Mild	12					
9	R	41 Moderate	58	70	5-0	75	Meniere's disease	Progressive
	L	48-Moderate	64					
10	**R**	46-Mild	66	66	0-7	67	Meniere's disease	Progressive
	L	48-Mild	66					
11	R	46-Moderate	47	47	0-3	47	RE hemangioma of internal auditory artery; LE mumps	Sudden
	L	7-Profound	7					

Ear indicated in bold = implanted ear. AAO/HL-Degree = age at onset (years) and degree of hearing loss; AAO/PHL = age at onset (years) of profound hearing loss; AAI = age at implant; LOU-CI = length of use (years-months) with cochlear implant; AAT = age at test.

and was varied between 3 and 3000 CU.* Procedures for the measures of behavioral threshold (BT), most comfortable loudness level (MCL), and upper limit of comfort level (ULCL) are described in detail in the companion paper in this issue.

Electrophysiologic Measures

Stimuli, Equipment, and Procedures • Stimuli used to elicit the electrical evoked potentials were identical to those used to obtain BT, MCL, and ULCL and were within the subject's BDR for each electrode. Electrically evoked potentials were recorded at levels that corresponded to a) 100%, 75%, 50%, and 25% of the BDR, b) the BT, and c) the evoked potential threshold. As a final control, EABR, EAMLR, and ELAR responses were recorded at the lowest amplitude input (3 CU) generated by the stimulus software to evaluate response identification against the subject's baseline EEG activity.

Responses were recorded with a Nicolet Compact 4 (C4) electrodiagnostic system externally triggered by the stimulus output of the SCLIN for Windows software and the interface unit. The interface unit connected to a pulse stretcher that extended the duration of the pulse (200 μsec) sent from the interface unit to trigger the Nicolet C4 averaging computer. The interface unit also connected to a stock speech processor and the subject's headpiece, transmitting the stimulus signal across the skin to the implanted device. The amplifier connected to electrodes that were positioned through a radio frequency filter with a low-pass cutoff of 32.125 kHz preceding the C4 amplifier.

The electrical evoked potentials were recorded using standard gold cup surface electrodes placed on the forehead (+), nape of the neck (−), and contralateral earlobe (common). Recording of electrical activity included two or three replications of 1000 sweeps (EABR), 500 sweeps (EAMLR), and 300 sweeps (ELAR) at each stimulus level with a time window of 10 msec (EABR), 50 msec (EAMLR), and 300 msec (ELAR) for each stimulus condition. For the ELAR, eye movements were monitored using electrodes located on the superior and lateral canthus of one eye (Kraus et al., 1993). Artifact rejection eliminated trials that included eye movement and interfered with the recording of the response. Responses were amplified 100,000 times. Frequency

*Electrical charge is the product of current amplitude and pulse duration, and interacts with electrode impedance. For the Clarion device used in this study, the charge is expressed in CUs. For this subject sample and tested electrodes, the range of impedance values was narrow (7–28 kΩ, mean = 18.6 kΩ). Actual electrical charge was calculated for each current level using a function that incorporated electrode number, measured impedance, and current for one internal cochlear stimulator. The estimated actual current values (electrical charge) using this correction function resulted in minimal current differences due to the low impedance values for these subjects. The analyses described in our study do not depend on the absolute current level, but rather proportions within the subject's dynamic range. It should be noted, however, that CUs may not be directly comparable across subjects and electrodes.

cutoffs of 100 and 3000 Hz (EABR), 5 and 500 Hz (EAMLR), and 1 and 100 Hz (ELAR) were used (Butterworth, 12 dB/octave). For each evoked potential type, subjects were tested in a quiet exam room in a reclining chair. For EABR testing, subjects were instructed to relax and encouraged to sleep during the recording session. For the EAMLR and ELAR testing, subjects were instructed to remain awake and watch a captioned videotape.

Identification and Measures of Waveforms

The waveforms for early, middle and late responses were identified according to criteria that were based on animal and human research with acoustic and electrical stimulation. For the EABR, it was expected that 1) a series of positive peaks would occur between approximately 1 to 4 msec; 2) the latency of Wave V would occur at approximately 3.5 to 4.0 msec at higher stimulus current levels and increase with decreases in stimulus level; and 3) Wave I would not be visible because it would have a latency of approximately 0.75 msec (as seen in recordings of the electrically evoked action potential, Abbas et al., 1998; Franck & Norton, 2001), and would be embedded in stimulus artifact (Abbas & Brown, 1988, 1991; Picton, Hillyard, Krausz, & Galambos, 1974; van den Honert & Stypulkowski, 1986). For the EAMLR, a negative trough, Na, at approximately 15 to 18 msec, would be followed by a positive peak, Pa, at approximately 25 to 30 msec from the onset of the stimulus at suprathreshold levels (Jyung et al., 1989; Kileny & Kemink, 1987; Özdamar & Kraus, 1983; Picton et al., 1974). Electrical N1-P2 cortical responses would consist of a negative trough, N1, at approximately 80 to 110 msec, followed by a positive peak, P2, at approximately 180 to 210 msec (Kraus et al., 1993; Näätänen & Picton, 1987; Picton et al., 1974; Ponton & Don, 1995). For all evoked potentials, each waveform was compared with that generated with a minimum current level during the control run.

The latencies were measures (in msec) at the midpoint for the peak (Wave V, Pa, P2), or the midpoint of the trough (Na, N1). The evoked potential threshold was defined as the lowest current level that a repeatable response could be visually detected. Amplitudes were calculated based on the difference (in μV) between the positive peak and the following trough for Waves II, III and V, between the Na trough and Pa peak for the Na-Pa complex, and between the N1 trough and P2 peak for the N1-P2 complex.

RESULTS

Effects of electrode location and stimulus level on evoked potential amplitudes and latencies were evaluated with separate repeated-measures analyses of variance (ANOVAs). The three stimulus levels of 100% and 75% of the BDR and the evoked potential threshold were chosen because almost all subjects produced waveforms at these levels. The threshold of Wave V, the Na-Pa complex, and the N1-P2 complex were each examined with separate 1-way repeated-measure ANOVA to examine the effect of the implanted electrode location. None of the subjects had EABR, EAMLR, or ELAR present at the BT, and therefore the recordings obtained at BT are not discussed in the results.

EABR

EABR waveforms were recorded from 9 of 11 subjects. Two subjects had no measurable EABRs on any of the three electrodes at any stimulus presentation level. For those subjects with EABRs, recordings contained one to three positive peaks, Waves II, III, and V.

Morphology • Figure 1 displays EABR tracings recorded from Electrodes 1, 4, and 7 for two subjects at the stimulus current level equal to 100% of the subject's BDR. Subject 1 had Waves II, III, and V present on each tested electrode. Subject 5 had Waves II, III and V present on Electrode 1, but only Waves III and V on Electrodes 4 and 7. For all subjects, Electrode 1 (apical location) tended to produce the best responses, i.e., those with the largest amplitude, best morphology, and greatest number of positive peaks. Recordings from the basal end of the electrode array (Electrode 7) tended to have the poorest morphology and smallest amplitudes.

Wave V was present at the upper portion of the BDR (100% and 75%) for all subjects. Wave III was present in the majority of subjects on Electrode 1 at 100% and 75% of the BDR. Wave II was present in only 55% of subjects on Electrode 1 at 100% and 75% of the BDR. Recordings on Electrode 7 showed fewer EABR components overall, especially when the stimulus level was at 50% or less of the BDR.

Absolute Latency and Interpeak Interval Measures • Table 2 displays the mean absolute latency values and one standard deviation from the mean for Waves II, III, and V for all subjects at each stimulus level and each electrode, and the mean latency across all electrodes. Wave V latency increased slightly as stimulus level was decreased. Wave V latency is somewhat longer for the basal Electrode 7 than either Electrode 1 or 4 at 100% or 75% of the BDR, and at Wave V threshold (WVT). The mean latency values for all electrodes across

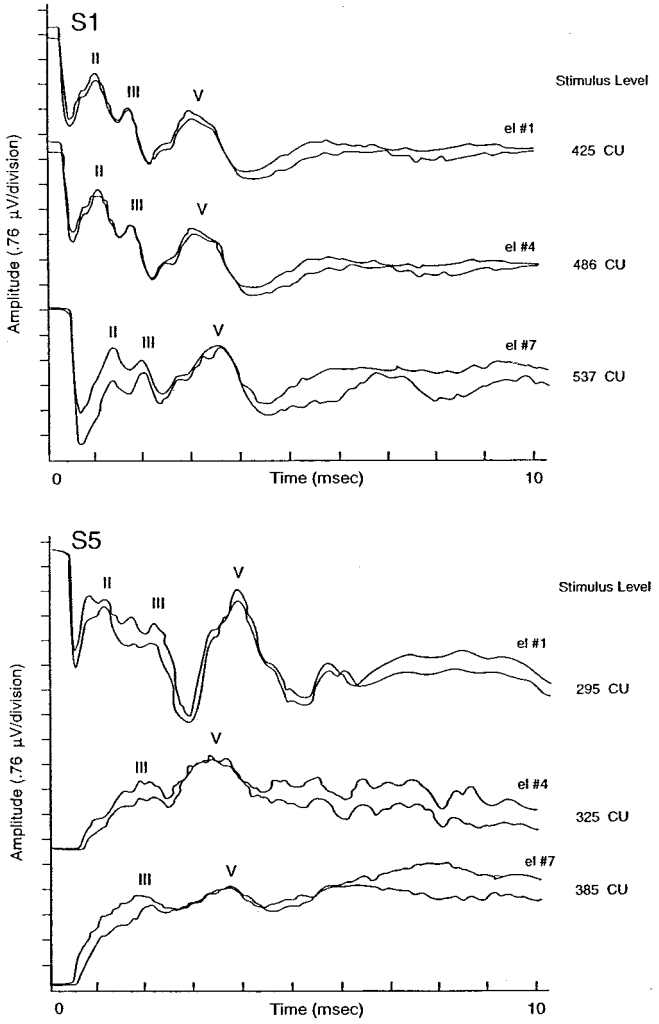

Figure 1. EABR tracings from two subjects at the stimulus level that corresponded to 100% of the BDR. Recordings from electrodes that represent apical (el 1), mid (el 4), and basal (el 7) stimulation are displayed for Subject 1 (upper panel) and Subject 5 (lower panel).

stimulus level increased on average 0.4 msec from 100% of the BDR to WVT.

For Wave V latency, ANOVAs with repeated measures for electrode site and stimulus level were completed. There were significant effects for both electrode ($F = 8.044$, $df = 2,16$, $p = 0.004$) and stimulus level ($F = 36.719$, $df = 2,16$, $p < 0.001$). Post hoc comparisons for electrode site alone were significant for Electrodes 1 versus 7 ($F = 10.912$, $df = 1,8$, $p = 0.011$) and Electrodes 4 versus 7 ($F = 10.630$, $df = 1,8$, $p = 0.012$). Post hoc comparisons for stimulus level were significant for all comparisons ($p \leq 0.05$). There were no significant interactions between electrode site and stimulus level for Wave V latency. The mean interpeak latencies at 100% of the BDR averaged across electrodes for the intervals II-III, III-V, and II-V were 0.79 msec, 1.62 msec, and 2.41 msec, respectively.

Amplitude Measures • The mean Wave V amplitudes at 100% of the BDR for Electrode 1, 4, and 7 were 1.46 μV, 1.42 μV, and 1.16 μV, respectively. The differences in mean Wave V amplitude across stimulus levels and across electrodes are illustrated in Figure 2. The mean amplitude of Wave V decreased with decrease in stimulus current for each electrode. Electrode 1 tended to have the largest Wave V amplitude, followed by Electrode 4 and then 7, although these differences were not statistically significant.

There were significant effects for electrode stimulus level ($F = 17.680$, $df = 2, 16$, $p < 0.001$). Post hoc tests for stimulus level were significant for comparison of 100% and 75% of the BDR ($F = 8.844$, $df = 1,8$, $p = 0.018$), 100% of the BDR and WVT ($F = 19.687$, $df = 1,8$, $p = 0.002$), and for 75% of the BDR and WVT ($F = 21.113$, $df = 1,8$, $p = 0.002$). There were no significant interactions between electrode site and stimulus level for Wave V amplitude.

The mean Wave II and III amplitude values for all subjects at each tested electrode and stimulus level, and the mean amplitude across all electrodes is shown in Table 3. Amplitudes for Waves II and III were smaller than those for Wave V, decreased with decreasing stimulus level, and were substantially smaller for Electrode 7 than for Electrodes 1 or 4.

Threshold Measures • Wave V was the most robust EABR component. EABR tracings from Subject 2 on Electrode 4 recorded at seven stimulus levels are shown in Figure 3. The levels include 100%, 75%, 50%, and 25% of the BDR, as well as the level producing WVT, and the stimulus level one step below that needed for threshold. The waveform elicited during a control run with a stimulus level of 3 CU is also shown.

Stimulation of Electrode 1 resulted in the lowest mean WVT, followed by Electrode 4, and then Electrode 7 (highest mean threshold). The difference in threshold across electrodes was statistically significant ($F = 10.211$, $df = 2,16$, $p = 0.001$) as were post hoc tests for all WVT electrode comparisons ($p \leq 0.05$).

EAMLR

EAMLR waveforms were recorded on at least two electrodes from 8 of 11 subjects. Three subjects had no measurable EAMLRs on any electrodes at any stimulus level. One subject had responses for Electrodes 1 and 4, but no response for Electrode 7.

Morphology • In Figure 4, EAMLR responses at 100% of the BDR from Subjects 5 and 6 for Electrodes 1, 4, and 7 are displayed. EAMLR waveforms were similar in morphology across electrodes within subjects. The Na-Pa complex tended to decrease in

TABLE 2. Means and standard deviations for EABR Waves II, III, and V absolute latencies across electrodes and stimulus levels

	Wave II Absolute Latency											
	Electrode 1			Electrode 4			Electrode 7			All Electrodes		
Percent of BDR	Mean	SD	*n*	Mean	SD	*n*	Mean	SD	*n*	Mean	SD	*n**
100	1.29	0.11	5	1.31	0.08	3	1.29	0.07	2	1.30	0.09	10
75	1.31	0.11	5	1.33	0.07	3	1.29	0.07	2	1.31	0.08	10
50	1.37	0.06	3	1.34	0.08	3				1.35	0.07	6
25	1.38	0.08	2							1.38	0.08	2
	Wave III Absolute Latency											
	Electrode 1			Electrode 4			Electrode 7			All Electrodes		
Percent of BDR	Mean	SD	*n*	Mean	SD	*n*	Mean	SD	*n*	Mean	SD	*n**
100	2.12	0.16	9	2.08	0.16	8	2.07	0.41	7	2.09	0.17	24
75	2.09	0.13	8	2.18	0.24	7	2.14	0.34	7	2.14	0.19	22
50	2.16	0.13	6	2.04	0.05	4	2.04	0.30	3	2.08	0.08	13
25	2.22	0.14	5	2.06	0.06	2				2.15	0.10	7
	Wave V Absolute Latency											
	Electrode 1			Electrode 4			Electrode 7			All Electrodes		
Percent of BDR	Mean	SD	*n*	Mean	SD	*n*	Mean	SD	*n*	Mean	SD	*n**
100	3.69	0.14	8	3.68	0.16	8	3.76	0.20	9	3.71	0.17	25
75	3.70	0.16	9	3.70	0.18	9	3.87	0.20	9	3.76	0.18	27
50	3.83	0.21	9	3.80	0.17	9	3.87	0.19	5	3.83	0.19	23
25	3.86	0.27	6	3.84	0.12	5	3.86	0.12	3	3.85	0.17	14
WVT	4.07	0.12	9	4.08	0.17	9	4.17	0.19	9	4.11	0.16	27

*Latency values are expressed in msec. BDR = behavioral dynamic range; WVT = wave V threshold; n** value represents total electrodes across subjects. The percentage of the behavioral dynamic range at which wave V threshold occurred was not always less than 25% of the BDR for individual subjects.

amplitude with decrease in stimulus current level. However, at lower stimulus levels, there was less change in amplitude and the response disappears rapidly as the visual detection threshold is crossed. This characterization has been reported with acoustic recordings of the MLR in normal-hearing subjects, where the growth of Pa amplitude occurs at stimulus levels between 40 to 80 dB HL, with relatively small change in amplitude for stimuli below 40 dB HL (Özdamar & Kraus, 1983). Components Na and Pa were present in all subjects with responses on Electrode 1 and 7 at 100% and 75% of the BDR, and on Electrode 4 at 100%, 75%, and 50% of the BDR. At least 50% of the subjects had Na-Pa responses at 25% of the BDR for Electrodes 1 and 4.

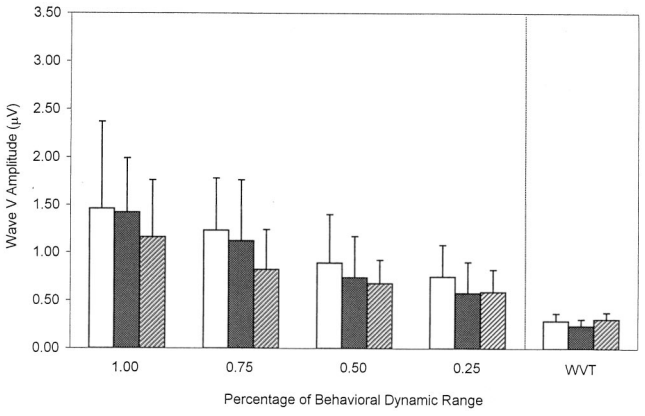

Figure 2. Amplitude of the EABR Wave V response averaged across subjects and plotted for each tested electrode (1 open bar, 4 filled bar, 7 hatched bar) at the stimulus levels that represent 100%, 75%, 50%, and 25% of the BDR. WVT was not always less than 25% of the BDR for individual subjects. Error bars represent one standard deviation from the mean.

Absolute Latency Measures • Mean latency data for Na and Pa are shown in Table 4. Absolute latencies for both Na and Pa increased slightly as stimulus level decreased from 100% of the BDR to threshold. For Na and Pa, respectively, the average latency increase was 0.74 and 1.33 msec for Electrode 1, 1.19 and 0.60 msec for Electrode 4, and 1.41 and 0.26 msec for Electrode 7.

For the latency of Na, there were significant effects for electrode ($F = 5.178$, $df = 2,12$, $p = 0.024$) but not for stimulus level ($F = 3.770$, $df = 2,12$, $p = 0.054$). Post hoc comparisons for electrode site were significant for only Electrodes 4 versus 7 ($F = 6.539$, $df = 1,6$, $p = 0.043$). There were no significant interactions between electrode site and stimulus level for Na latency. For Pa latency, there were no

TABLE 3. Means and standard deviations for EABR Wave II and Wave III amplitudes across electrodes and stimulus levels.

	Wave II Amplitude											
	Electrode 1			Electrode 4			Electrode 7			All Electrodes		
Percent of BDR	Mean	SD	*n*	Mean	SD	*n*	Mean	SD	*n*	Mean	SD	*n**
100	0.95	0.41	5	1.05	0.49	3	0.78	0.35	2	1.00	0.41	10
75	0.64	0.48	5	0.79	0.18	3	0.47	0.06	2	0.65	0.35	10
50	0.73	0.19	3	0.39	0.08	3				0.56	0.23	6
25	0.32	0.23	2							0.32	0.23	2
	Wave III Amplitude											
	Electrode 1			Electrode 4			Electrode 7			All Electrodes		
Percent of BDR	Mean	SD	*n*	Mean	SD	*n*	Mean	SD	*n*	Mean	SD	*n**
100	1.06	0.74	9	0.86	0.60	8	0.52	0.39	7	0.82	0.65	24
75	0.88	0.47	8	0.99	0.60	7	0.40	0.33	7	0.76	0.52	22
50	0.72	0.47	6	0.77	0.24	4	0.46	0.37	3	0.73	0.35	13
25	0.42	0.23	5	0.43	0.22	2				0.42	0.21	7

*Amplitude values are expressed in μV. BDR = behavioral dynamic range; n** value represents total electrodes across subjects.

significant effects for either electrode site or stimulus level.

Amplitude Measures • The Na-Pa amplitude was determined by measuring the trough-to-peak amplitude between the negative Na trough and the positive peak of Pa. In Figure 5, the mean Na-Pa amplitude data by electrode site and stimulus level are illustrated. At 100% of the BDR, Electrode 1 showed the largest amplitude, followed by Electrodes 4 and 7, but these were not statistically significant differences.

The Na-Pa amplitude decreased with a decrease in stimulus level. There were significant effects for stimulus level ($F = 10.991$, $df = 2,12$, $p = 0.002$). Post hoc comparisons for stimulus level were significant for 100% of the BDR versus the Na-PaT ($F = 19.189$, $df = 1,6$, $p = 0.005$), and 75% of the BDR and the Na-PaT ($F = 15.388$, $df = 1,6$, $p = 0.008$). There were no significant interactions between electrode

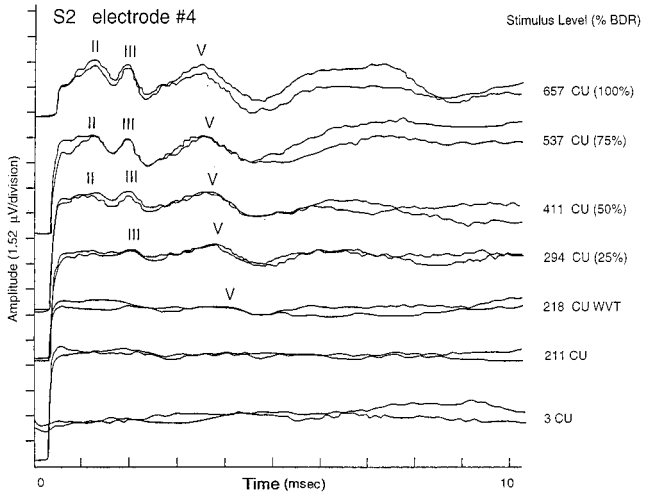

Figure 3. EABR waveforms recorded from Subject 2 on Electrode 4 are displayed. Two recordings are shown for each stimulus current level ranging from 100% of the BDR to a control run at 3 CU. WVT was identified at 218 CU.

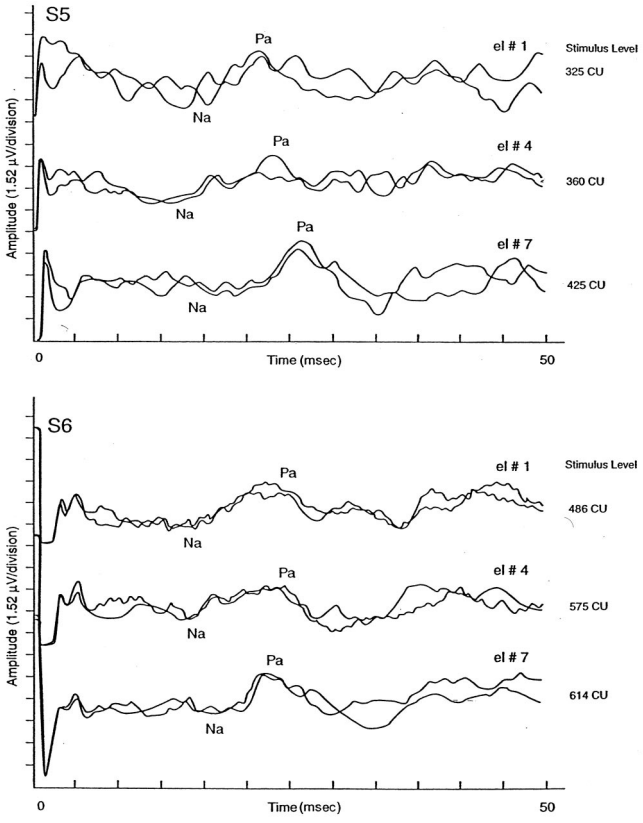

Figure 4. EAMLR tracings (Na-Pa) obtained from two subjects at the stimulus level that corresponded to 100% of the BDR. Recordings from Electrodes 1, 4, and 7 are displayed for Subject 5 (upper panel) and Subject 6 (lower panel).

TABLE 4. Means and standard deviations for EAMLR Na and Pa absolute latencies across electrodes and stimulus levels

	Na Absolute Latency											
	Electrode 1			Electrode 4			Electrode 7			All Electrodes		
Percent of BDR	Mean	SD	*n*	Mean	SD	*n*	Mean	SD	*n*	Mean	SD	*n**
100	15.78	1.47	8	15.24	1.56	8	15.20	1.38	7	15.41	1.47	23
75	15.23	1.32	8	15.56	1.26	8	15.81	0.91	7	15.53	1.16	23
50	15.68	1.24	6	16.09	1.04	8	16.70	0.85	5	16.16	1.04	19
25	16.79	1.61	5	16.28	2.33	4	16.52	1.11	3	16.53	1.68	12
NaT	16.52	1.35	8	16.43	1.25	8	16.61	1.07	7	16.52	1.22	23
	Pa Absolute Latency											
	Electrode 1			Electrode 4			Electrode 7			All Electrodes		
Percent of BDR	Mean	SD	*n*	Mean	SD	*n*	Mean	SD	*n*	Mean	SD	*n**
100	25.76	0.74	8	26.86	1.91	8	26.92	1.68	7	26.37	1.77	23
75	26.66	0.47	8	27.44	1.88	8	27.17	1.68	7	27.09	1.78	23
50	27.54	0.47	6	27.28	2.26	8	27.53	2.10	5	27.45	2.05	19
25	28.17	0.23	5	27.25	2.25	4	28.45	1.65	3	27.96	2.00	12
PaT	27.09	1.77	8	27.04	2.25	8	27.18	2.34	7	27.10	2.12	23

*Latency values are expressed in msec. BDR = behavioral dynamic range; Values at NaT = latency of Na at the threshold of the Na-Pa complex; Values at PaT = latency of Pa at the threshold of the Na-Pa complex; n** value represents total electrodes across subjects. The percentage of the behavioral dynamic range at which threshold occurred for the Na-Pa complex was not always less than 25% of the BDR for individual subjects.

site and stimulus level for the amplitude of the Na-Pa complex.

Threshold Measures • Stimulation of Electrode 1 resulted in the lowest Na-PaT, followed by Electrode 4, and then Electrode 7. These differences in threshold across electrodes were statistically significant ($F = 8.623$, $df = 2,12$, $p = 0.005$). Post hoc comparisons were significant for Electrodes 1 and 4 ($F = 11.195$, $df = 1,6$, $p = 0.015$) and Electrodes 1 and 7 ($F = 12.650$, $df = 1,6$, $p = 0.012$), but not for Electrodes 4 and 7.

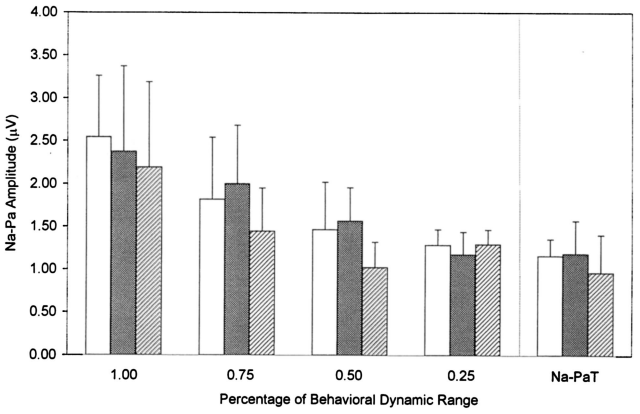

Figure 5. Amplitude of the Na-Pa complex averaged across subjects and plotted for Electrodes 1 (open bar), 4 (filled bar), and 7 (hatched bar) at the stimulus levels that represent 100%, 75%, 50%, and 25% of the BDR. Error bars represent one standard deviation from the mean.

ELAR

Electrical N1-P2 cortical responses of the ELAR were recorded for at least two electrodes from 9 of 11 subjects. As with the EABR and EAMLR, two subjects had no measurable responses. One subject, who had no EAMLRs, had measurable N1-P2 responses for Electrode 1 only. The same subject who had no EAMLR response present for Electrode 7 did not produce an ELAR on that electrode.

Morphology • Figure 6 displays N1-P2 responses at 100% of the BDR for two subjects for Electrodes 1, 4, and 7. The consistency across subjects and across electrodes for the N1-P2 waveform was similar to that observed for the Na-Pa complex. The effects of decreasing stimulus level were less orderly than those noted for the EABR, and more similar to those observed with the EAMLR. Components N1 and P2 were present in all subjects on Electrode 1 at 100% and 75% of the BDR, with fewer components recorded at 50% and 25% on all three electrodes. The fewest responses were present at 25% of the BDR for Electrode 7.

Absolute Latency Measures • The mean N1 and P2 absolute latency values and one standard deviation from the mean for all subjects, and the average values across electrodes are shown in Table 5. The mean latency across electrodes at 100% of the BDR was 86.50 msec for N1 and 181.02 msec for P2. The latencies for N1 and P2 increased with decreased stimulus level from 100% of the BDR to the thresh-

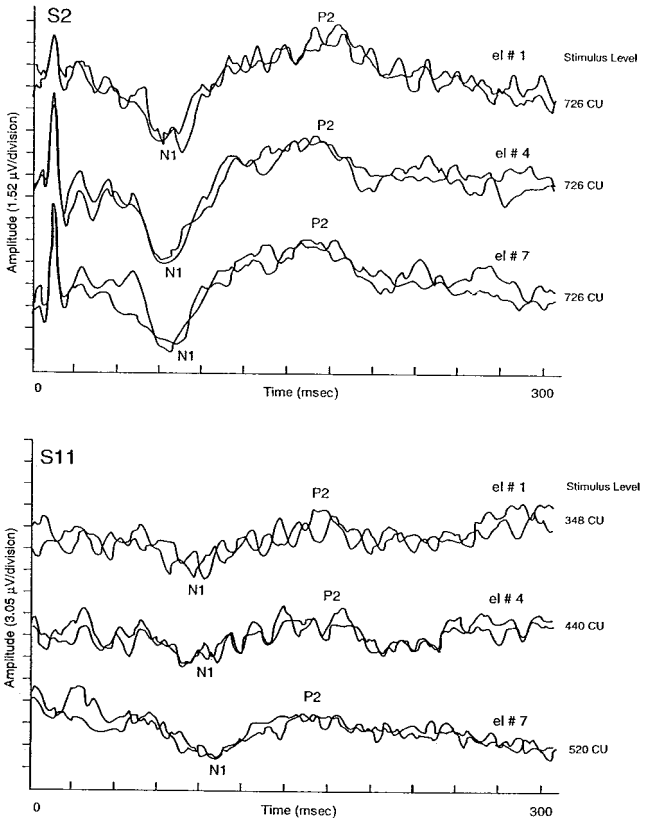

Figure 6. ELAR tracings (N1-P2) obtained from two subjects at the stimulus level that corresponded to 100% of the BDR. Recordings are displayed for stimulation of Electrodes 1, 4, and 7 for Subject 2 (upper panel) and Subject 11 (lower panel).

old of the N1-P2 complex. N1 increased across the BDR by 3.90 msec for Electrode 1, 5.41 msec for Electrode 4, and 3.73 msec for Electrode 7, while P2 increased 7.82 msec, 8.78 msec, and 9.07 msec for Electrodes 1, 4, and 7, respectively.

For both the N1 and P2 latencies, there were no significant main effects for electrode but there were significant effects for stimulus level (for N1, $F = 7.701$, $df = 2,12$, $p = 0.007$; for P2, $F = 62.764$, $df = 2,12$, $p < 0.001$). For N1 latency, post hoc comparisons for stimulus level were significant for 100% versus 75% of the BDR ($F = 16.240$, $df = 1,6$, $p = 0.007$) and 100% of the BDR versus Na-PaT ($F = 10.325$, $df = 1,6$, $p = 0.018$). For P2 latency, post hoc tests for stimulus level were significant for all comparisons ($p < 0.01$). There were no significant interactions between electrode site and stimulus level for either N1 or P2 latency.

Amplitude Measures • The N1-P2 amplitude was determined by measuring the trough-to-peak amplitude between the negative N1 trough and the positive P2 peak. The mean amplitude at 100% of the BDR for Electrodes 1, 4 and 7 was 5.65 μV, 4.78 μV, and 3.85 μV, respectively. Figure 7 illustrates the mean N1-P2 amplitude data for subjects by electrode site and stimulus level. The N1-P2 amplitude on each electrode decreased with decrease in stimulus level. Consistent with the EABR and EAMLR results, the N1-P2 amplitude tended to be smallest for Electrode 7 compared with Electrode 1 or 4, but differences were not significant statistically.

TABLE 5. Means and standard deviations for ELAR N1 and P2 absolute latencies across electrodes and stimulus levels

	N1 Absolute Latency											
	Electrode 1			Electrode 4			Electrode 7			All Electrodes		
Percent of BDR	Mean	SD	*n*	Mean	SD	*n*	Mean	SD	*n*	Mean	SD	*n**
100	87.31	11.51	9	86.85	15.14	8	85.04	13.10	7	86.50	12.71	24
75	87.54	12.31	9	87.85	15.20	8	86.69	16.48	7	87.40	13.06	24
50	87.30	10.68	7	87.74	12.54	7	86.93	13.45	5	87.35	11.55	19
25	88.75	12.12	6	88.66	13.16	7	74.70	11.46	2	86.83	12.66	15
N1T	91.21	15.24	9	92.26	15.19	8	88.77	11.75	7	90.85	13.75	24
	P2 Absolute Latency											
	Electrode 1			Electrode 4			Electrode 7			All Electrodes		
Percent of BDR	Mean	SD	n	Mean	SD	n	Mean	SD	n	Mean	SD	n*
100	182.49	12.97	9	182.76	12.49	8	177.14	12.52	7	181.02	12.38	24
75	184.34	12.22	9	184.15	12.92	8	181.36	13.35	7	183.41	12.29	24
50	183.37	13.92	7	185.16	14.11	7	184.52	11.88	5	185.16	12.96	19
25	185.90	14.61	6	187.52	15.95	7	172.50	14.85	2	184.89	15.07	15
P2T	190.31	11.14	9	191.54	14.80	8	186.21	11.59	7	189.52	12.33	24

*Latency values are expressed in msec. BDR = behavioral dynamic range; Values at N1T = latency of N1 at the threshold of the N1-P2 complex; Values at P2T = latency of P2 at the threshold of the N1-P2 complex; n** value represents total electrodes across subjects. The percentage of the behavioral dynamic range at which threshold occurred for the N1-P2 complex was not always less than 25% of the BDR for individual subjects.

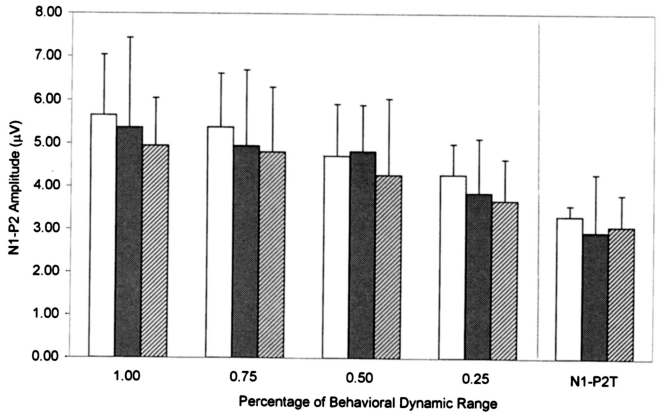

Figure 7. Amplitude of the N1-P2 complex averaged across subjects and plotted for Electrodes 1 (open bar), 4 (filled bar), and 7 (hatched bar) at the stimulus levels that represent 100%, 75%, 50%, and 25% of the BDR. Error bars represent one standard deviation from the mean.

There were significant effects for stimulus level ($F = 28.687$, $df = 2,12$, $p < 0.001$). Post hoc comparisons for stimulus level were significant for 100% of the BDR versus N1-P2T ($F = 26.379$, $df = 1,6$, $p = 0.002$), and 75% of the BDR versus N1-P2T ($F = 51.296$, $df = 1,6$, $p < 0.001$). There were no significant interactions between electrode site and stimulus level for the amplitude of the N1-P2 complex.

The amplitude of N1-P2 was plotted as a function of stimulus current level for individual subjects and electrodes. For each subject, an increase in current level generally resulted in an increase in amplitude, as observed for the other evoked potentials under study. There were five subjects and eight tested electrodes that showed evidence of amplitude saturation. One subject, S8, had little amplitude growth for N1-P2, which also was observed for the EABR.

Threshold Measures • Stimulation of Electrode 1 had the lowest mean threshold, and Electrode 7 had the highest. The differences in threshold across electrodes were statistically significant ($F = 10.871$, $df = 2,14$, $p = 0.001$). Post hoc testing indicates thresholds for Electrodes 1 and 7 ($F = 22.219$, $df = 1,7$, $p = 0.002$) and Electrodes 4 and 7 ($F = 8.806$, $df = 1,7$, $p = 0.025$) were significantly different.

Comparison of Wave V, Na-Pa, and N1-P2 Across Subjects

The latencies of Wave V, Na, Pa, N1 and P2 were included in analyses for each electrode at 100% of the BDR to determine whether effects of latency were consistent across the evoked potential responses under study. The analyses were repeated for 75% of the BDR and the evoked potential threshold stimulus level. Of the latency comparisons, only those for N1 and P2 were significantly correlated ($r = 0.87$ to 0.96). A similar analysis was performed for the measures of amplitude and threshold for all evoked potentials, and significant relations for the waveform components were not indicated for this subject sample.

DISCUSSION

A primary goal of this study was to record the EABR, the EAMLR, and the ELAR and to characterize the responses with regard to the effects of electrode location and stimulus current level. To achieve this goal, evoked potentials were elicited on three intra-cochlear electrodes along the implanted array, and responses were recorded at five stimulus levels that corresponded to proportions of the BDR. The evoked potential thresholds for the EABR, EAMLR, and ELAR were also obtained for each subject on each electrode.

Presence of the EABR, EAMLR, and ELAR

For the EABR, Wave V was the most robust, was present on more electrodes, and was identified at more stimulus presentation levels than Waves II and III. Wave I could not be identified due to stimulus artifact. Past studies (Brown, Abbas, & Gantz, 1990) and present studies with newer implant technology (Abbas et al., 1998; Franck & Norton, 2001) have demonstrated that the electrical whole nerve action potential (or wave I of the EABR) can be recorded with innovative recording paradigms that overcome the effects of stimulus artifact. For the EAMLR, the Pa component was the most robust, and Na and Pa were present for all subjects at high proportions of the BDR. For the ELAR, wave P2 was the most robust, and N1 and P2 were present at 100% and 75% of the BDR for most subjects and most electrodes.

Effects of Electrode Site and Stimulus Level on the EABR, EAMLR, and ELAR

For the main effect of electrode site, Wave V latency was significantly longer for Electrode 7 (basal) compared with either Electrode 1 (apical) or Electrode 4 (mid). Some studies have reported significant latency differences across electrodes for subjects where basal electrodes have longer latencies than apical electrodes (Abbas & Brown, 1991; Shallop et al., 1990). Other studies have reported no significant differences (Abbas & Brown, 1988; van den Honert & Stypulkowski, 1986). Wave V latency increased as stimulus current level decreased. This also occurs with acoustic stimulation, although with electrical stimulation, the latency changes are

smaller with corresponding changes in electrical stimulus current. There are no published reports for comparison of latency characteristics across electrodes with subjects who have received the Clarion implant.

Wave V amplitudes were largest for Electrode 1, second largest for Electrode 4, and smallest for Electrode 7, but these differences were not statistically significant. The amplitude values suggest that direct electrical stimulation tends to produce a Wave V that is larger in amplitude than that produced with acoustic stimuli, a finding noted in other reports of electrically evoked potentials (Abbas, 1993; van den Honert & Stypulkowski, 1986) and attributed to increased synchrony of auditory nerve fiber responses for electrical stimulation.

The WVT for each electrode was significantly different. The lowest WVT was for Electrode 1 and the highest for Electrode 7. Threshold measures are influenced by the proximity of the implanted electrode array to spiral ganglion cells. It may be that higher thresholds for the most basal electrode are a result of greater distance from neural elements compared with more apical electrodes, which are located further along the scala tympani.

For the EAMLR, there were fewer significant effects related to latency compared with the EABR. The Na component latency was significantly different for Electrodes 4 and 7 only, and for Pa, there were no significant latency differences for electrode site. Although Na and Pa latencies increased slightly with decrease in stimulus current level, the differences were not statistically significant. This finding regarding EAMLR and EABR latencies with respect to stimulus intensity parallels another report that used acoustic stimuli for recordings from normal-hearing subjects, in which the ABR was found to be highly stimulus intensity dependent but the AMLR was not (Özdamar & Kraus, 1983).

There are few published studies that discuss the EAMLR parameters, including latency, when recorded from intracochlear electrodes. A number of studies have reported findings of the EAMLR with transtympanic stimulation. Acoustic and electrical MLRs were compared for six subjects (Kileny et al.,1989) where electrical Pa latencies were shorter than acoustic Pa latencies (average electrical Pa latency of 31 msec, acoustic Pa latency 35 msec). In the present study, the average Pa latency for intracochlear stimulation across electrodes (26.37 msec at 100% of the BDR) was also earlier than typical Pa latencies elicited with acoustic stimuli.

The amplitude of the Na-Pa complex was largest for Electrode 1, and smallest for Electrode 7, but these differences were not statistically significant. The main effect for stimulus level on the amplitude of the response was significant for the EAMLR, but not at all stimulus levels, as it was for the EABR. This finding is similar to comparisons reported by Özdamar and Kraus (1983) between the acoustic ABR and AMLR recorded from the same subjects in which the Wave V response continued to increase in amplitude with increase in stimulus intensity while the Pa response showed no further increase in amplitude beyond 50 to 60 dB HL. Electrical Pa amplitudes have been shown to be larger (mean of 1.01 μV) than acoustic Pa amplitudes (mean of 0.70 μV) in the same subjects (Kileny et al.,1989). This contrast may result from enhanced neural synchrony with electrical stimulation.

Similar to the EABR in the present study, the electrical threshold for the Na-Pa complex (Na-PaT) was lower for Electrode 1, highest for Electrode 7, and differences by electrode site were generally significant. There are no other studies of the EAMLR components with electrical stimulation that report the evoked potential thresholds by electrode site.

For the ELAR, neither the latency of N1 nor the latency of P2 showed statistically significant differences between Electrodes 1, 4, and 7. The average latency across electrodes at 100% of the BDR was 86 msec for N1 and 181 msec for P2, which may be slightly earlier than the acoustic counterpart for each component, depending on the comparison study's subject sample and stimulus characteristics. In a study by Ponton and Don (1995), peak latencies (N1 and the MMN difference wave) for Nucleus 22 cochlear implant subjects were earlier (10 to 20 msec) than the responses for normal-hearing subjects. For computer-generated speech stimuli delivered through a loudspeaker in the sound field, a study of the P300 response indicated that N1 and P2 latencies were not significantly different between nine Nucleus implant subjects and nine normal-hearing control subjects (Micco et al., 1995). Click stimuli delivered directly to the implanted electrodes, as in the present study and that of Ponton and Don (1995), result in earlier ELAR latencies than those elicited with speech stimuli delivered through a loudspeaker in the sound field, as in the Micco et al. (1995) study.

There were significant differences found for the main effect of stimulus level for the latency of both N1 and P2. Previous studies tended to elicit the ELAR at suprathreshold stimulus levels; therefore, there are no published reports of the changes in latency for N1 or P2 in cochlear implant users with stimulus levels in the lower portion of the BDR, or at the threshold of the evoked response. With acoustic studies, latency changes with intensity for N1 and

P2 vary, depending on whether tonal, click or speech stimuli have been used to evoke the response.

As seen with the EAMLR, the amplitude of the N1-P2 complex of the ELAR was largest for Electrode 1 and smallest for Electrode 7, but statistical tests indicate no significant effects for electrode site. In the Ponton and Don (1995) study, the MMN was larger and more frequently identified for the apical electrode pair than for the basal pair. In the current study, there were also significant main effects for stimulus level at 100% or 75% of the BDR versus the threshold. Until now, there are no published reports of the changes in amplitude for N1 or P2 with differing stimulus levels in cochlear implant users. Acoustical studies indicate that amplitude increases with increase in stimulus intensity, but that the largest increase occurs within the first 20 to 30 dB above the auditory threshold, followed by increases in amplitude that are more gradual with increase in stimulus intensity, and for some subjects at high intensities, amplitude measures can reach a point of saturation (Buchsbaum, 1976; Davis & Zerlin, 1966; Näätänen & Picton, 1987; Picton, Woods, Baribeau-Braun, & Healey, 1977; Rapin, Schimmel, Tourk, Krasnegor, & Pollak, 1966). In the present study, there were 10 instances of apparent amplitude saturation for the ELAR (average amplitude decrease compared with the maximum amplitude of 0.92 μV, and a range of 0.14 μV to 2.51 μV) compared with five occurrences for the EAMLR (average amplitude decrease of 0.54 μV, and a range of 0.15 μV to 1.01 μV), and four occurrences for the EABR that were of very small magnitude (average amplitude decrease of 0.05 μV, and a range of 0.03 μV to 0.09 μV).

The pattern of lowest evoked potential threshold for Electrode 1, second lowest for Electrode 4, and highest for Electrode 7 held true for the ELAR as seen for the EABR and the EAMLR. For the ELAR, there were statistically significant differences for the threshold comparisons of Electrodes 1 and 7, and 4 and 7. There are no other published reports for late auditory response components with electrical stimulation and implant users that report threshold differences across electrode site.

Interpeak Intervals

In this sample of subjects, interpeak intervals for the EABR wave components occurred at shorter intervals than those that are typical for the acoustic ABR (i.e., 1 msec between each positive peak from Wave I to Wave V). As early as 1985, Gardi reported reduced interpeak latency values (0.75 msec to 0.80 msec). EABR interpeak latencies in the present study were 0.79 msec (II-III), 1.62 msec (III-V) and 2.41 msec (II-V). In other publications that have compared acoustic and electrical evoked potentials at higher levels of the auditory system, such as the middle and late latency responses, the electrical components often occur earlier than the acoustic components resulting in shorter latencies, as described previously. Enhanced neural synchrony with electrical stimulation may be related to compressed interpeak intervals throughout the auditory pathway. The site of initial electrical stimulation in the periphery bypasses the cochlear transduction processes and thus accounts for some of the small decreases in the latency of peaks. This small reduction, however, can not account for the larger differences observed in peak latencies for more centrally evoked potentials, which may be related to faster conduction time throughout the auditory system with electrical stimulation.

Recording Artifact

Several studies have indicated that signal contamination can occur when recording electrically evoked potentials that are sources of stimulus or muscle artifact (Abbas & Brown, 1991; van den Honert & Stypulkowski, 1986). Stimulus artifact interferes with the electrical recording of Wave I of the EABR. The spread of electrical stimulation can result in activation of nonauditory tissue or nerve fibers, such as muscle or vestibular endings, particularly at high stimulus current levels, and may result in large amplitude responses that are atypical. In this study, unusually large amplitude responses were observed at 100% of the BDR for one subject for EABR Wave V (Subject 6) and one subject for the Na-Pa complex (Subject 10) that were well outside the amplitude range for the respective response based on the other subjects, and were probably associated with nonauditory stimulation. Subject 1 had a large potential that occurred at 7 to 8 msec during the EAMLR recording at 100% of the BDR on Electrodes 1 (stimulus level 679 CU), 4 (stimulus level 751 CU), and 7 (stimulus level 635 CU), which then disappeared at stimulus levels that represented 75% of the BDR for each electrode. This potential was several milliseconds earlier than the latencies of Na and Pa, and did not interfere with the measures of the later Na and Pa components. Subject 2 also demonstrated a large triphasic potential between 5 msec and 9 msec at 100% of the BDR for Electrodes 1 (stimulus level 702 CU), 4 (stimulus level 679 CU), and 7 (stimulus level 679 CU), that was most likely stimulation of nonauditory fibers. Electrically evoked auditory potentials that are not similar to acoustically evoked auditory potentials in waveform morphology or that have unusu-

ally large amplitudes should be interpreted cautiously.

In conclusion, this study summarizes the response parameters of the EABR (Waves II, III, V), EAMLR (Na-Pa complex), and ELAR (N1-P2 complex) obtained from 11 Clarion cochlear implant users. Results of electrophysiologic recordings from three intra-cochlear electrodes and five stimulus current levels that corresponded to proportions of the BDR are presented. It is possible that other functional relationships would be evident if additional subjects, electrode sites, and stimulus levels were included. There are no published studies that discuss the latency, amplitude, and threshold values for EABR, EAMLR, and ELAR across the electrical dynamic range that have been elicited from the same experimental subjects. Electrically evoked auditory responses can provide a reliable and objective assessment of auditory function in cochlear implant recipients. As the need for objective measures with cochlear implant users increases, it is critical to understand how these electrical potentials behave when stimulus parameters are systematically varied. Data from this study may serve as a normative reference for expected latency, amplitude and threshold values for the recording of brainstem and cortical electrically evoked auditory potentials. Responses recorded from cochlear implant users show many similar patterns, yet important distinctions, compared with auditory potentials elicited with acoustic signals.

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REFERENCES

Abbas, P. J. (1993). Electrophysiology. In R. S. Tyler (Ed.), *Cochlear Implants: Audiological Foundations*. San Diego: Singular Publishing Group.

Abbas, P. J., & Brown, C. J. (1988). Electrically evoked brainstem potentials in cochlear implant patients with multi-electrode stimulation. *Hearing Research*, *36*, 153–162.

Abbas, P. J., & Brown, C. J. (1991). Electrically evoked auditory brainstem response: Growth of response with current level. *Hearing Research*, *51*, 123–138.

Abbas, P. J., Brown, C. J., Shallop, J. K., Firszt, J. B., Hughes, M. L., Hong, S. H., & Staller, S. J. (1998). Summary of results using the Nucleus CI24M implant to record the electrically evoked compound action potential (EAP). *Ear and Hearing*, *20*, 45–59.

Brown, C. J., Abbas, P. J., Fryauf-Bertschy, H., Kelsay, D., & Gantz, B. J. (1994). Intraoperative and postoperative electrically evoked auditory brainstem responses in Nucleus cochlear implant users: Implications for the fitting process. *Ear and Hearing*, *15*, 177–183.

Brown, C. J., Abbas, P. J., & Gantz, B. J. (1990). Electrically evoked whole-nerve action potentials I. Data from Symbion cochlear implant users. *Journal of the Acoustical Society of America*, *88*, 1385–1391.

Brown, C. J., Hughes, M. L., Lopez, S. M., & Abbas, P. J. (1999). Relationship between EABR thresholds and levels used to program the Clarion speech processor. *Annals of Otology, Rhinology, and Laryngology* (Suppl. 177), *108*, 4–2, 50–57.

Burton, N. J., Miller, J. M., & Kileny, P. R. (1989). Middle latency responses: I. Electrical and acoustic stimulation. *Archives of Otolaryngology*, *115*, 59–62.

Buchsbaum, M. (1976). Self-regulation of stimulus intensity: Augmenting/reducing and the average evoked response. In G. E. Schwartz & D. Shapiro (Eds.), *Consciousness and Self-Regulation* (pp. 101–135). New York: Plenum Publishing Corp.

Davis, H., & Zerlin, S. (1966). Acoustic relations of the human vertex potential. *Journal of the Acoustical Society of America*, *39*, 109–116.

Firszt, J. B., Chambers, R. D., & Kraus, N. (2002). Neurophysiology of cochlear implant users II: Comparison among speech perception, dynamic range and physiological measures. *Ear and Hearing*, *23*, 516–531.

Firszt, J. B., Rotz, L. A., Chambers, R. D., & Novak, M. A. (1999). Electrically evoked potentials recorded in adult and pediatric Clarion implant users. *Annals of Otology, Rhinology, and Laryngology* (Suppl. 177), *108*, 4–2, 58–63.

Franck, K. H., & Norton, S. J. (2001). Estimation of psychophysical levels using the electrically evoked compound action potential measured with the neural response telemetry capabilities of Cochlear Corporation's CI24M device. *Ear and Hearing, 22*, 289–299.

Gardi, J. N. (1985). Human brainstem and middle latency responses to electrical stimulation: Preliminary observations. In R. A. Schindler & M. M. Merzenich (Eds.), *Cochlear Implants* (pp. 351–363). New York: Raven Press.

Groenen, P., Snik, A., & van den Broek, P. (1997). Electrically evoked middle latency responses versus perception abilities in cochlear implant users. *Audiology*, *36*, 83–97.

Hodges, A. V., Ruth, R. A., Lambert, P. R., & Balkany, T. J. (1994). Electric auditory brain stem responses in Nucleus multichannel cochlear implant users. *Archives of Otolaryngology-Head and Neck Surgery*, *120*, 1093–1099.

Jyung, R. W., Miller, J. M., & Cannon, S. C. (1989). Evaluation of eighth nerve integrity by the electrically evoked middle latency response. *Otolaryngology Head and Neck Surgery*, *101*, 670–682.

Kaga, K., Kodera, K., Hirota, E., & Tsuzuka, T. (1991). P300 response to tones and speech sounds after cochlear implant: A case report. *Laryngoscope*, *101*, 905–907.

Kileny, P. R., & Kemink, J. L. (1987). Electrically evoked middle latency potentials in cochlear implant candidates. *Archives of Otolaryngology Head and Neck Surgery*, *113*, 1072–1077.

Kileny, P. R., Kemink, J. L., & Miller, J. M. (1989). An intrasubject comparison of electrical and acoustic middle latency responses. *American Journal of Otology*, *10*, 23–27.

Kraus, N., Micco, A. G., Koch, D. B., McGee, T., Carrell, T., Sharma, A., Wiet, R. J., & Weingarten, C. Z. (1993). The mismatch negativity cortical evoked potential elicited by speech in cochlear-implant users. *Hearing Research*, *65*, 118–124.

Makhdoum, M. J., Groenen, P., Snik, A., & van den Broek, P. (1997). Intra-and interindividual correlations between auditory evoked potentials and speech perception in cochlear implant users. *Scandinavian Audiology*, *22*, 1–8.

Mason, S. M., Sheppard, S., Garnham, C. W., Lutman, M. E., O'Donoghue, G. M., & Gibbin, K. P. (1993). Application of intraoperative recordings of electrically evoked ABRs in a paediatric cochlear implant programme. In F. B. Deguine (Ed.), *Cochlear Implants: New Perspectives* (pp. 136–141). Basel: Karger.

Micco, A. G., Kraus, N., Koch, D. B., McGee, T. J., Carrell, T. D., Sharma, A., Nicol, T., & Wiet, R. J. (1995). Speech-evoked cognitive P300 potentials in cochlear implant recipients. *American Journal of Otology*, *16*, 514–520.

Miyamoto, R. T. (1986). Electrically evoked potentials in cochlear implant subjects. *Laryngoscope*, *96*, 178–185.

Näätänen, R., & Picton, T. (1987). The N1 wave of the human electric and magnetic response to sound: A review and an analysis of the component structure. *Psychophysiology*, *24*, 375–425.

Oviatt, D. L., & Kileny, P. R. (1991). Auditory event-related potentials elicited from cochlear implant recipients and hearing subjects. *American Journal of Audiology*, *1*, 48–55.

Özdamar, O., & Kraus, N. (1983). Auditory middle latency responses in humans. *Audiology*, *22*, 34–49.

Picton, T. W., Hillyard, S. A., Krausz, H. I., & Galambos, R. (1974). Human auditory evoked potentials. I. Evaluation of components. *Electroencephalography and Clinical Neurophysiology*, *36*, 179–190.

Picton, T. W., Woods, D. L., Baribeau-Braun, J., & Healey, T. M. G. (1977). Evoked potential audiometry. *The Journal Of Otolaryngology*, *6*, 90–119.

Ponton, C. W., & Don, M. (1995). The mismatch negativity in cochlear implant users. *Ear and Hearing*, *16*, 130–146.

Rapin, I., Schimmel, H., Tourk, L. M., Krasnegor, N. A., & Pollak, C. (1966). Evoked responses to clicks and tones of varying intensity in waking adults. *Electroencephalography and Clinical Neurophysiology*, *21*, 335–45234.4.

Shallop, J. K., Beiter, A. L., Goin, D. W., & Mischke, R. E. (1990). Electrically evoked auditory brainstem responses (EABR) and middle latency responses (EMLR) obtained from patients with the Nucleus Multichannel cochlear implant. *Ear and Hearing*, *11*, 5–15.

Shallop, J. K., Van Dyke, L., Goin, D. W., & Mischke, R. E. (1991). Prediction of behavioral threshold and comfort values for Nucleus 22-channel implant patients from electrical auditory brainstem response test results. *Annals of Otology, Rhinology, and Laryngology*, *100*, 896–898.

Simmons, F. B., & Smith, L. (1983). Estimating nerve survival by electrical ABR. *Annals of New York Academy of Science*, *405*, 422–423.

Starr, A., & Brackmann, D. E. (1979). Brainstem potentials evoked by electrical stimulation of the cochlea in human subjects. *Annals of Otology, Rhinology and Laryngology*, *88*, 550–556.

van den Honert, C., & Stypulkowski, P. H. (1986). Characterization of the electrically evoked auditory brainstem response (ABR) in cats and humans. *Hearing Research*, *21*, 109–126.

Walsh, S. N., & Leake-Jones, P. (1982). Chronic electrical stimulation of auditory nerve in cat: physiological and histological results. *Hearing Research*, *7*, 281–304.

REFERENCE NOTE

1 Firszt, J. B., Gaggl, W., Wackym, P. A., & Reeder, R. M. (2000) Electrical evoked potentials recorded with the Med El cochlear implant device. Paper presented at the US Investigator Meeting, Stans, Austria, November.