Journal of Autism and Developmental Disorders, Vol. 19, No. 2, 1989

Brainstem and Middle Latency Auditory Evoked Potentials in Autism and Developmental Language Disorder[1]

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Brainstem auditory evoked potentials (BAEP) and middle latency responses (MLR) were studied in 8 nonretarded subjects with infantile autism (mean age = 23.3, SD = 2.8), 8 subjects with receptive developmental language disorder (mean age = 16.3, SD = 1.4), and normal control subjects matched to each group for age, gender, and Performance IQ. Click stimuli were delivered monaurally to the left and the right ear and binaurally for both the BAEPs (70-dB HL, 7/sec) and the MLRs (60-dB HL, 13/sec). Amplitudes and latencies (Waves I to VI), interwave latencies (III–V, I– V, and I–III), and Wave I/V amplitude ratio of the BAEPs were determined for each group. For the MLR study, Wave Na, Pa, and Nb latencies, and Wave Na–Pa and Pa–Nb amplitudes were calculated. There were no consistent differences in

[1]The authors thank Jamie Costello who facilitated the use of the evoked potentials equipment at the Speech, Hearing and Neurosensory Center, Children's Hospital, San Diego. The research reported was supported, in part, by National Institute of Mental Health grant 1-R01-MH36840 and NINCDS grant 5-R01-NS19855 awarded to Eric Courchesne.

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0162-3257/89/0600-0255$06.00/0

the BAEP and MLR characteristics of the control and the experimental groups. These results suggest that the abnormal cognitive processes indexed by the cognitive and attention-related event-related potential components in infantile autism and receptive developmental language disorder are not due to abnormal sensory processing in the brainstem and in areas central to the brainstem whose activity generates the BAEPs and MLRs.

INTRODUCTION

Subjects with infantile autism and receptive developmental language disorder (RDLD) share important similarities and differences. Both appear early in life, involve language impairments, and present abnormalities in such areas as auditory sensory and temporal integration, and auditory verbal abstraction. However, the severe social and behavioral abnormalities that accompany autism are lacking in RDLD (Rutter, 1978). Rutter, in attempting to characterize the behavioral and verbal similarities and differences between autistic and RDLD subjects, concluded that the quality and nature of the language impairments were quite different in the two groups. He also emphasized the necessity for further elucidation of the "link" as well as the differences between the two disorders.

Event-related potentials (ERPs) are physiological markers of different stages of processing from sensory registration, to cognitive, and finally to motor. Therefore, they appear to be valuable tools for investigating the electrophysiological characteristics of both autistic and RDLD subjects. A global classification of the ERPs is the distinction between sensory or "exogenous" and cognitive or "endogenous" components. The exogenous ERPs reflect the integrity and organization of the sensory pathways, whereas the characteristics of the endogenous ERPs are determined by psychological factors rather than by the physical properties of the stimulus events (Figure 1).

In a recent electrophysiological investigation of nonretarded adolescents with infantile autism and RDLD, we have shown that auditory Nc and P3b, two endogenous ERP components associated with attention and memory processes, differed in these two clinical groups (Courchesne, Lincoln, Yeung-Courchesne, Elmasian, & Grillon, 1989). Nc is recorded from over the frontal cortex, is among the first endogenous ERP components to appear during development, and is known to be elicited by attention-getting stimuli (Courchesne, Elmasian, & Yeung-Courchesne, 1987). Auditory Nc and P3b were smaller than normal in the autistic group, whereas auditory P3b was abnormally enlarged in the RDLD group. Given these findings, it is of interest to determine whether or not the early processing of auditory sensory information is abnormal in autistic and dysphasic subjects.

Two classes of auditory exogenous potentials are the brainstem auditory evoked responses (BAEP) and the middle latency responses (MLR). The

BAEPs have been useful to both audiologists and neurologists for testing high-frequency acoustic responses in the auditory periphery and the brainstem. The BAEPs consist of six components (labeled by Roman numerals I though VI) which appear in the first 10 msec after stimulus onset. Studies of the analogous potentials in animals (Buchwald & Huang, 1975; Caird, Sontheimer, & Klinke, 1985; Legatt, Arezzo, & Vaughan, 1986), and recordings

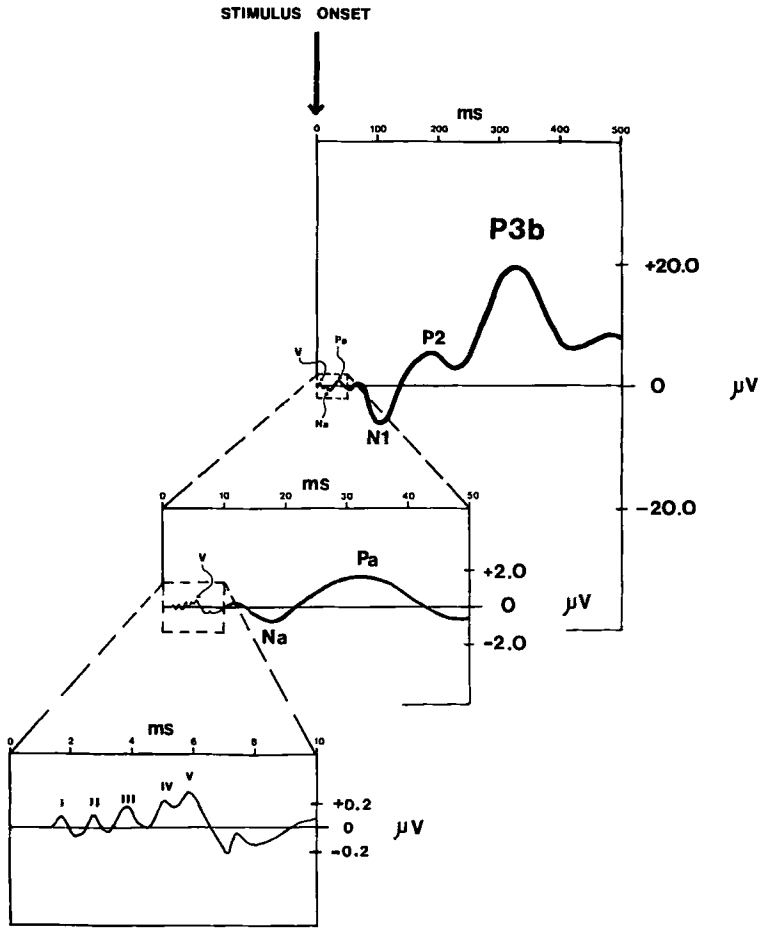

Fig. 1. Idealized illustration of an ERP response elicited by a 70-dB normal-hearing-level sound—a click. The ERP is a continuous series of components occurring at various latencies after stimulus onset and having various amplitudes and durations. Shorter latency components—Wave I, II, III, IV, and Na and Pa—are classic examples of exogenous (or sensory) components. The longer latency component P3b is a classic example of endogenous (or cognitive) component. From Courchesne (1987).

from human patients with neurological lesions (Starr & Hamilton, 1976; Stockard & Rossiter, 1977) indicate that these waves probably arise from structures in the classical auditory pathways, that is, from the VIII cranial nerve to the medial geniculate body and acoustic radiations.

The MLRs are a series of potentials occurring between 10 and 80 msec poststimulus. They share a common time domain with myogenic responses and, following their discovery (Geisler, Frishkopf, & Rosenblith, 1958), many years passed before it was demonstrated that MLRs were unaltered by neuromuscular blocking agents (Rickard, Clark, McMahon, & Dewhurst, 1973). Major components of the MLRs are Na, Pa, and Nb. The origins of the MLRs are not as well documented as the BAEPs, and the locations of their exact generators have been controversial. However, there are some indications that the MLRs represent activity in the thalamus, the thalamic radiations, and the auditory cortex (Cohen, 1982; Hammond, Bruni, & Wilder, 1980; Kraus, Ozdamar, Hier, & Stein, 1982; Picton, Hillyard, Krausz, & Galambos, 1974; Woods, Clayworth, & Knight, 1985). Since these anatomical areas underly the ability to comprehend speech, it has been thought that the MLRs could be affected in patients with impairments of speech perception (Kraus, Smith, Reed, Stein, & Cartee, 1985; Mason & Mellor, 1984). Moreover, since the MLRs, unlike the BAEPs, are evoked by low- as well as high-frequency tones, they may have considerable utility in evaluating cochlear function across the frequency spectrum.

Factors such as age, gender, body temperature, and mental retardation are now known to affect BAEP characteristics. Earlier BAEP studies in autistic subjects did not take these factors into account. When most of these factors were controlled and when subjects with neurological disorders other than autism were excluded, the BAEPs were found to be normal in the majority of subjects with autism (Courchesne, Yeung-Courchesne, Hicks, & Lincoln, 1985; Rumsey, Grimes, Pikus, Duara, & Ismond, 1984; Tanguay, Edward, Buchwald, Schwafel, & Allen, 1982). There has not yet been a MLR study in autistic subjects.

There has been little BAEP or MLR investigation in subjects with developmental language disorders. Mason and Mellor (1984) did not find any abnormality in a group of children that included those with expressive language disorders as well as those with both receptive and expressive language disorders. However, the use of subjects with a mixture of diagnoses does not allow for any conclusion to be drawn about the functioning of the auditory pathway in subjects with receptive language disorders. Moreover, the recording was carried out with a mastoid reference and the authors raised the possibility that their recordings may have been distorted by postauricular muscle artifacts and myogenic activity.

In the present study, BAEPs and MLRs were recorded in nonretarded autistic and RDLD subject groups whose cognitive and attention-related ERP

components were shown to be abnormal in previous experiments (Ciesielski, Courchesne, & Elmasian, in press; Courchesne, Kilman, Galambos, & Lincoln, 1984; Courchesne et al., 1989). The goal of this work was to test the integrity of the sensory pathway associated with the processing of auditory information in these two groups. In order to address this goal, our design took into account several methodological points: (a) As in our previous BAEP study (Courchesne et al., 1985), subject matching was based on gender, age, and Performance IQ; (b) since our subjects were cooperative, no sedative drug was given; (c) body temperature, which is known to affect BAEP characteristics, was recorded in each subject; and (d) in order to avoid the contamination of the MLRs by temporal cortical activity (Wolpaw & Wood, 1982) or postauricular responses and myogenic activity (Erwin & Buchwald, 1986a), we used a noncephalic reference.

METHOD

Subjects

Eight nonretarded male autistic subjects (a subset of the 14 autistic subjects used in Courchesne et al., 1985),[3] 8 RDLD subjects (6 male, 2 female), and 10 control subjects (8 male, 2 female) participated in this experiment. From this group of 10 control subjects, two groups of 8 subjects each were drawn in order to match the autistic and RDLD subjects for gender, age, and Performance IQ on a pair basis. There is therefore some overlap between the control groups. The mean ages for the autistic subjects and their controls were 23.3 years (SD = 2.8) and 20.1 years (SD = 2.3), respectively. The mean ages for the RDLD subjects and their controls were 16.3 years (SD = 1.4) and 18.5 years (SD = 2.3), respectively. With one exception, the subjects did not have a history of long-term medication use. One RDLD subject had seizure disorder controlled by medication (Dilantin).

Control subjects were interviewed by a psychologist and were excluded from the study if there was a history of drug abuse, neurological disorders, or learning disabilities. None were taking medications at the time of testing. Control subjects were also administered the WISC-R or the WAIS-R in order to match them to the autistic and RDLD subjects for PIQ.

Each autistic and RDLD subject was separately diagnosed by two psychologists. The diagnoses were obtained via face-to-face interviews, review of medical records, and interview with parents. Both diagnosticians had to agree that infantile autism or RDLD was the primary and only Axis I diagnosis present in each individual.

[3]The autistic subjects were tested on BAEPs again to test BAEP normality on sessions in which MLRs were recorded.

The autistic subjects had to meet the full diagnostic criteria for infantile autism described in the DSM-III (American Psychiatric Association, 1980).[4] None of the subjects had other forms of neurological or physical disorder. The mean PIQ and VIQ of the autistic group was 97.4 (*SD* 13.5) and 88.1 (*SD* 18.9), respectively. For the normal control group, it was 105 (*SD* 10.5) and 109.5 (*SD* 11.7), respectively. Further descriptions of the autistic group can be found in Courchesne et al. (1984) and Ciesielski et al. (in press).

RDLD subjects had to (a) meet the DSM-III (see Footnote 4) criteria for receptive developmental language disorder not attributable to oral-motor dyspraxia or dysarthria, (b) have a documented history of early onset of abnormal language development (i.e., little or no expressive speech by 4 years of age), and (c) have evidence of continuous language impairment up to the present time. This latter point included current language test scores that fell outside the normal range (see below) and a consistent history of and current enrollment in special education classes for the learning- and language-impaired. After being identified, a RDLD person was included in this study if he or she had a Wechsler Performance IQ > 70 and a Wechsler Verbal IQ one standard deviation below the Performance IQ. The mean PIQ and VIQ of the RDLD group was 104.1 (*SD* 9.7) and 78.2 (*SD* 5.9), respectively. For the normal control group, it was 106.2 (*SD* 7.1) and 106.5 (*SD* 6.9), respectively.

Each autistic subject was administered the Test of Adolescent Language Development (Hammill, Brown, Larsen, & Wiederholt, 1980). The mean Adolescent Language Quotient for the group was 75.12 (*SD* 22.45). The RDLD subjects were administered the Peabody Picture Vocabulary Test-Revised (Dunn & Dunn, 1981), the auditory attention span for unrelated words, oral commissions, and auditory attention span for related syllables subtest from the subtests from the Detroit Test of Learning Aptitude (Baker & Leland, 1959), and the Processing subtests from the Clinical Evaluation of Language Aptitude (CELF; Semel-Mintz & Wii, 1982). The mean Peabody standard score was 74.5 (*SD* 6.99). The difference between chronological age and language age scores for the CELF and the Detroit were calculated for each RDLD subject. The mean difference on the CELF was -5.28 years (*SD* 3.01) and -6.07 years (*SD* 1.50) on the Detroit.

Recording and Stimulus Types

The stimuli were delivered through earphones, and evoked potentials were recorded using standard Ag/AgCl EEG electrodes. The electrode im-

[4]All subject selection occurred prior to DSM-III-R.

pedance was less than 5 Kohm. For both the BAEPs and the MLRs, the stimuli were presented monaurally to the right and left ear, and binaurally. Each condition was presented twice for reliability purposes.

Brainstem Auditory Evoked Potentials (BAEP). Seventy-dB HL rarefaction clicks were used. The clicks were 0.1 msec in duration and were delivered at a rate of 7/sec. The electrodes were placed at Cz, and over the left (A1) and the right (A2) mastoids according to the 10–20 system. The Fpz electrode was the earth electrode. Two recording channels were used: Cz referenced to A1 and Cz referenced to A2. Half-amplitude bandpass settings were 150 and 3,000 Hz. The recording window was 15 msec and each BAEP was the average of 2,000 stimuli.

Middle Latency Responses (MLR). Sixty-dB HL clicks with a duration of 0.1 msec were delivered at a rate of 13/sec. The electrodes were placed at Fpz, Fz, Cz, A1, and A2. The reference electrode was a noncephalic balanced electrode (Stephenson & Gibbs, 1951). For the monaural right ear and binaural stimulations, the earth electrode was A1. For the monaural left stimulation, the earth electrode was A2. Half-amplitude bandpass settings were 15 and 1,500 Hz. The recording window was 70 msec. Each MLR was the average of 4,096 stimuli.

Procedure

At the begining of the recording session, the psychoacoustic threshold for click stimuli for each subject was determined and body temperature was measured orally. There was no evidence of hearing impairment in any subject and each subject's body temperature was within the normal range. Each subject was seated in a reclining chair in a quiet room during the ERP recording. They were instructed to close their eyes and to remain quiet while clicks were being presented. The BAEPs were always recorded before the MLRs. Muscle or eye artifacts were automatically rejected from the recording using preestablished criteria. Each average was recorded on a floppy disk for further analysis.

Data Analysis

All peak latencies were relative to click onset. The amplitudes of Waves I, II, III, IV, V, and VI of the BAEPs were defined as the amplitude from their peaks to the first negative peak following each wave. For each subject, the interpeak latencies of Wave III–V, Wave I–V, and Wave I–III, and the amplitude ratio Wave I/Wave V were also calculated. For the MLRs, Na–Pa and Pa–Nb amplitudes were measured. Since visual inspection and measurement of the BAEPs and MLRs indicated that the initial and the replica-

tion averages were very similar, the data across replications were combined. Each patient group was compared to its own control group separately in the analyses.

RESULTS

Brainstem Auditory Evoked Potentials

The BAEP for monaural left and right ear stimulation and for binaural stimulation in a representative subject for each group are presented in Figure 2. Waves I to VI were identifiable in all the subjects in all the conditions. The means and standard deviations for Waves I, III, and V are presented in Table I. For the monaural stimulation, a series of two-way ANOVAs with repeated measures with group and ear of stimulation as the two factors were performed on the latency and amplitude measures of all the waves (Waves I to V), on the interpeak latencies (Waves I–V, III–V, and I–III), and on the amplitude ratio measures (Wave I/V). For the binaural stimulation, a

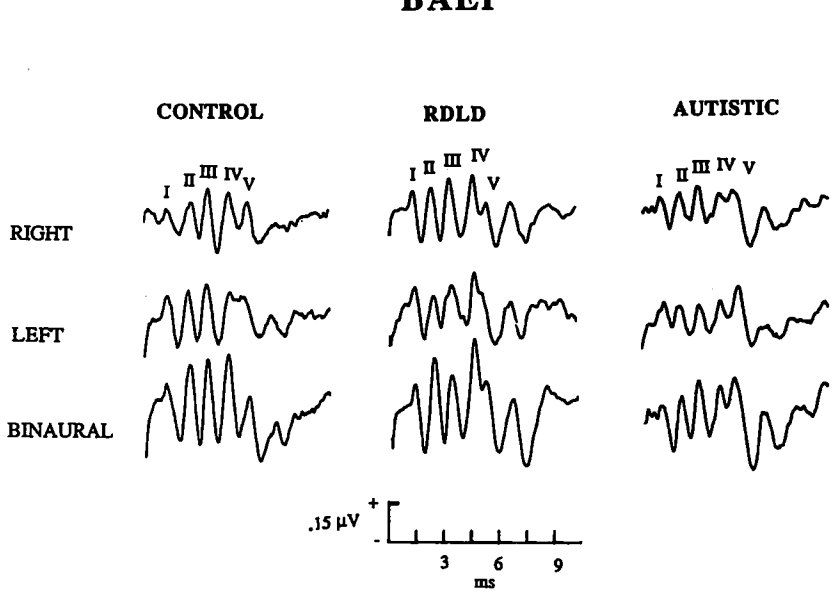

Fig. 2. BAEPs in a representative subject from each group for monaural left and right and binaural stimulation (Cz electrode, right mastoid reference).

Table I. Means and Standard Deviations for the BAEP Study in the Autistic and RDLD Groups and Their Respective Normal Control Groups

	Wave latency (msec)			Interpeak latency (msec)			Wave amplitude (μvolt)			
Group	I	III	V	I–III	III–V	I–V	I	III	V	I/V
Control										
Left	1.58	3.73	5.71	1.99	2.01	4.01	0.33	0.28	0.31	1.19
	(0.10)	(0.09)	(0.21)	(0.11)	(0.16)	(0.16)	(0.06)	(0.11)	(0.06)	(0.09)
Right	1.61	3.70	5.72	2.04	2.02	4.06	0.26	0.30	0.31	1.54
	(0.23)	(0.11)	(0.24)	(0.07)	(0.19)	(0.15)	(0.09)	(0.12)	(0.09)	(0.95)
Binaural	1.55	3.63	5.74	2.00	1.99	4.04	0.43	0.39	0.51	1.71
	(0.08)	(0.25)	(0.24)	(0.09)	(0.20)	(0.15)	(0.14)	(0.19)	(0.08)	(1.35)
Autistic										
Left	1.65	3.64	5.66	2.15	2.02	4.18	0.30	0.27	0.32	1.39
	(0.11)	(0.09)	(0.11)	(0.13)	(0.09)	(0.19)	(0.14)	(0.07)	(0.14)	(1.03)
Right	1.59	3.63	5.65	2.15	2.01	4.10	0.28	0.29	0.36	1.66
	(0.03)	(0.11)	(0.13)	(0.19)	(0.13)	(0.19)	(0.10)	(0.10)	(0.11)	(1.47)
Binaural	1.60	3.60	5.62	2.07	2.06	4.18	0.33	0.39	0.57	1.92
	(0.12)	(0.08)	(0.10)	(0.21)	(0.31)	(0.20)	(0.11)	(0.09)	(0.16)	(1.08)
Control										
Left	1.58	3.65	5.64	2.14	1.98	4.13	0.34	0.28	0.34	0.98
	(0.09)	(0.18)	(0.24)	(0.14)	(0.11)	(0.22)	(0.08)	(0.09)	(0.11)	(0.51)
Right	1.62	3.65	5.64	2.07	1.99	4.04	0.30	0.29	0.35	1.40
	(0.25)	(0.20)	(0.28)	(0.19)	(0.21)	(0.20)	(0.14)	(0.10)	(0.09)	(0.83)
Binaural	1.55	3.59	5.69	2.04	2.09	4.13	0.48	0.39	0.57	1.41
	(0.07)	(0.24)	(0.29)	(0.15)	(0.29)	(0.24)	(0.09)	(0.18)	(0.18)	(0.67)
RDLD										
Left	1.71	3.79	5.74	2.09	1.96	4.04	0.30	0.26	0.36	1.27
	(0.12)	(0.19)	(0.31)	(0.18)	(0.17)	(0.33)	(0.08)	(0.10)	(0.14)	(0.64)
Right	1.65	3.70	5.69	2.06	2.00	4.06	0.32	0.37	0.32	1.17
	(0.11)	(0.11)	(0.20)	(0.14)	(0.15)	(0.21)	(0.11)	(0.18)	(0.16)	(0.58)
Binaural	1.63	3.71	5.71	2.07	2.00	4.07	0.39	0.43	0.49	1.32
	(0.10)	(0.11)	(0.29)	(0.11)	(0.20)	(0.28)	(0.13)	(0.24)	(0.18)	(0.64)

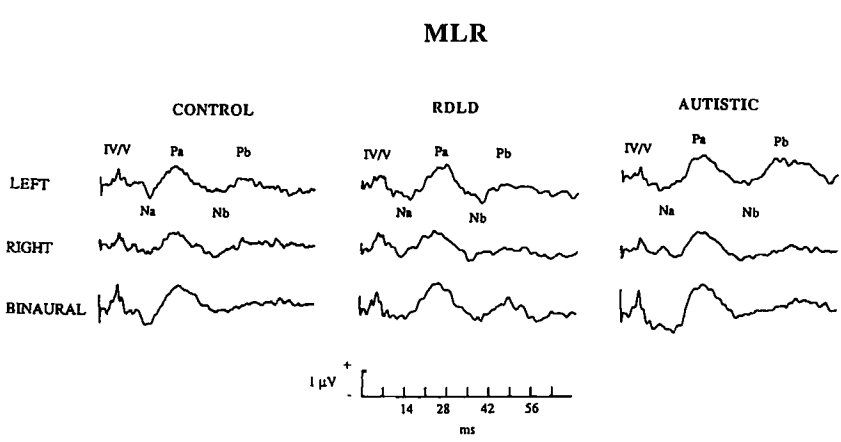

Fig. 3. MLR in a representative subject from each group for monaural left and right and binaural stimulation (Fz electrode, noncephalic reference).

series of two-way ANOVAs with group and side of recording as the two factors were performed on all the BAEP measures. None of the group effects or group interaction effects were significant.

Middle Latency Responses

The MLRs for a representative subject in each group are illustrated in Figure 3. Table II shows group means and *SD*s for the amplitude and latency measures recorded at Cz. There was no difference between the MLRs of the autistic and RDLD group and their respective control groups. This result was confirmed by a series of three-way ANOVAs with group, electrode site, and stimulus condition as the three factors. None of the group comparisons were significant.

DISCUSSION

In this study, BAEP and MLR measures were normal in both the RDLD and the autistic groups. The present study, therefore, replicates our earlier finding with 8 of the 14 autistic subjects tested previously (Courchesne et al., 1985) and are in agreement with studies that found minor or no differences between the BAEP of the autistic and control subjects (Ornitz, Olson, & Walter, 1980; Rumsey et al., 1984; Tanguay et al., 1982).

Because MLRs are believed to originate from the thalamus, the thalamic radiations, and the auditory cortex (Erwin & Buchwald, 1986a, 1986b;

Table II. Means and Standard Deviations for the MLR Study

Group and Stimulus type	Wave amplitude (μ volt)		Wave latency (msec)		
	Na–Pa	Pa–Nb	Na	Pa	Nb
Control					
Left	0.91	0.89	16.28	27.58	42.23
	(0.36)	(0.22)	(1.90)	(2.63)	(3.45)
Right	0.82	0.79	16.72	27.81	43.02
	(0.22)	(0.36)	(2.54)	(3.02)	(1.63)
Binaural	1.28	0.95	16.95	27.58	43.12
	(0.41)	(0.24)	(0.73)	(2.63)	(4.47)
Autistic					
Left	0.95	1.04	15.13	28.35	42.32
	(0.23)	(0.25)	(3.13)	(3.13)	(3.23)
Right	0.88	0.97	17.75	28.36	39.56
	(0.26)	(0.31)	(2.43)	(2.24)	(3.90)
Binaural	1.28	0.99	17.70	26.98	43.72
	(0.49)	(0.40)	(2.12)	(3.17)	(4.65)
Control					
Left	0.70	0.74	15.32	28.41	42.83
	(0.17)	(0.21)	(1.90)	(2.70)	(2.73)
Right	0.89	0.65	15.86	27.27	40.88
	(0.35)	(0.38)	(2.36)	(2.36)	(3.53)
Binaural	1.28	0.86	16.47	28.43	43.40
	(0.32)	(0.19)	(0.70)	(1.76)	(4.82)
RDLD					
Left	0.76	0.81	16.80	28.81	44.27
	(0.28)	(0.35)	(1.74)	(3.30)	(4.10)
Right	0.87	0.74	16.69	28.65	41.62
	(0.25)	(0.41)	(2.23)	(3.10)	(5.06)
Binaural	1.25	0.86	16.61	28.13	42.03
	(0.37)	(0.53)	(1.86)	(5.19)	(3.08)

Kraus et al., 1982, 1985; Woods et al., 1985), it had been thought that they might be sensitive to impairments in language functions. Both the autistic and the RDLD subjects we tested had language impairments, and yet both groups had normal MLRs. There have been many MLR studies with patients presenting with diverse language and speech impairments. Kraus et al. (1982) found that Na and Pa were within normal limits in patients who had cortical lesions of the left and the right hemispheres and had a variety of expressive and receptive aphasias. They concluded that Pa does not appear to be an electrical sign of language processing. Parving, Salomon, Elberling, Larsen, and Lassen (1980) reported normal MLRs in a patient with auditory agnosia, but Ozdamar, Kraus, and Curry (1982) found an absence of the Pa component in a subject with cortical deafness. Mason and Meller (1984) did not find any abnormality in MLRs recorded at the vertex site in children with mixed dysphasia or motor speech disorder due to congenital suprabulbar pa-

resis or developmental verbal dyspraxia. In their motor speech disorder group, they attributed larger MLR amplitudes in the temporal electrode to myogenic activity. Kraus et al. (1985) recorded MLRs in different patient groups including 10-year-old children with diagnosed communicative disorders (speech and language delays or learning disabilities). They did not find any difference in the detectability of the Na and Pa components in the language-impaired children as compared to the normal controls and concluded that absent or abnormal MLR could not be considered a sign of auditory pathway dysfunction.

These studies suggest that subjects with speech and language impairment often do not present with abnormal MLRs and question the validity of the MLR as a test of speech or language dysfunction. However, MLR research is still in its infancy and, unlike the BAEPs, little is known about their determinants. It is possible that the type of MLR studies conducted to date have not targeted the most relevant stimulation or recording parameters. Therefore, studies of the functional (binaural interaction, intensity-amplitude and intensity-latency functions, refractory properties) and the topographical characteristics of the MLRs in homogeneous groups of patients with specific audiological, speech, and language disorders, and with relevant neurological lesions are needed before any definitive conclusion can be drawn.

We did not find any difference in the group evaluation of BAEPs and MLRs. However, the possibility remains that specific BAEP or MLR abnormalities may be found in some subgroups of autistic or RDLD individuals. Some authors have reported BAEP abnormalities in some of their autistic subjects (Fein, Skoff, & Mirsky, 1981; Gillberg, Rosenhall, & Johansson, 1983; Rumsey et al., 1984; Taylor, Rosenblatt, & Linschoten, 1982) and Gillberg et al. (1983) suggested that there may be a subgroup of autistic subjects with abnormal BAEPs. However, large numbers of BAEP measures from individual "patients" have often been compared to "normative" data from small control groups (typically about 10 subjects). This technique could be criticized for two reasons. First, this technique increases the possibility of making Type I errors due to the very high number of comparisons for each individual. In our study, this would have led to 39 and 45 comparisons per subject for the BAEPs and the MLRs, respectively. Second, the sample of the control groups is usually too small to qualify the data as representative of "the normal population" (see American Electroencephalographic Society, 1984). Normative data should be collected from a much larger population and tolerance limits should be used. In studies of individual BAEP data, the fact that both increased and decreased peak and interpeak latencies have been reported (Garreau, Tanguay, Roux, & Lelord, 1984; Rumsey et al., 1984; Tanguay et al., 1982) supports our contention that the techniques used for identification of individual abnormalities are inadequate.

In conclusion, the BAEPs and MLRs were normal in both the nonretarded autistic and the nonretarded RDLD subjects. Pathology in the brain structures that generate the BAEP or MLR is, therefore, not necessary for autism or RDLD to develop. Abnormalities in other brainstem, cerebellar, thalamic, or auditory cortex structures, however, may still be possible. The autistic and RDLD subjects tested in this study have abnormalities in their late cognitive ERP components (Ciesielski et al., in press; Courchesne et al., 1984, 1989). Therefore, in autism and RDLD, the abnormal cognitive processes indexed by the cognitive and attention-related ERP components are not due to abnormal sensory processing by the neural structures whose activity generates the BAEPs and MLRs.

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