Speech-Evoked Neurophysiologic Responses in Children with Learning Problems: Development and Behavioral Correlates of Perception

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Objectives: **To evaluate the maturational progression of speech-evoked P1/N1/N2 cortical responses over the life span, determine whether responses are distinctive in clinical populations experiencing learning problems and elucidate the functional significance of these responses.**

Design: **The P1/N1/N2 complex was measured in 150 normal subjects (5 to 78 yr) and 86 subjects with learning problems (LP) (8 to 15 yr) to a synthetic CV syllable. Analyses included description and comparison of the developmental time course in both groups and evaluation of the relationship between P1/N1/N2 and children's performance on speech discrimination tasks and standardized learning measures.**

Results: **Findings revealed significant changes in waveform morphology, latency and amplitude as a function of age. Maturational patterns in the group of children with learning problems did not differ from the normal group. P1/N1/N2 parameters were significantly correlated with standardized tests of Spelling, Auditory Processing and Listening Comprehension in the LP group. Moreover, there was a predictive relationship between Auditory Processing and N2 latency.**

Conclusions: **The P1/N1/N2 complex changes throughout life from school-age to old age. The developmental sequence throughout the school-age years is similar in normal and LP children. Thus, differences in the rate of P1/N1/N2 latency and amplitude development do not appear to be distinctive in these two populations. The relationship between P1/N1/N2 parameters and standardized measures of learning (particularly between Auditory Processing and N2 latency) provides new information about the role of these responses in hearing and highlights the potential value in characterizing auditory processing deficits.**

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Auditory function is a complex process that develops and changes throughout life. Auditory skills related to basic perception of frequency, intensity and time develop early, reaching maturity by 5 yr of age (Collins & Gescheider, 1989; Olsho, 1985; Trehub, Schneider, & Henderson, 1995; Werner, 1996; Wightman, Allen, Dolan, Kistler, & Jamieson, 1989). In contrast, development of more advanced auditory behavior related to aspects of speech perception and listening in noise progresses into the school-age years (Elliott, 1979). Normal auditory development provides a solid foundation for the acquisition of more complex processes such as speech and language and in turn, academic skills such as reading and written language.

Many school-age children have difficulty demonstrating basic proficiency in these academic areas, and are eventually diagnosed with learning and/or attention problems. There is growing evidence to suggest that in some children the root cause of these learning problems may lie in auditory perceptual deficits specifically related to the processing of complex signals such as speech (Elliott & Hammer, 1988; Kraus, McGee, Carrell, Zecker, Nicol, & Koch, 1996; Nittrouer, 1999). To date, the exact nature of this deficit remains controversial. Some researchers claim that these children demonstrate temporal processing deficits (Tallal, 1980; Tallal et al., 1996). Others contend that deficits arise from impaired phonologic coding (Studdert-Kennedy & Mody, 1995). Another hypothesis purports that maturational delays in the acquisition of advanced auditory processes may be a contributing factor. Although these issues are far from resolved, even less is known about the neurophysiologic processes underlying these auditory perceptual deficits.

Recent studies have begun to elucidate the biologic bases of speech perception deficits. For example, individuals with reading problems have been shown to differ from normal readers in neural recovery time in response to rapidly presented stimuli (Nagarajan, Mahncke, Salz, Tallal, Roberts, & Merzenich, 1999). In another study, children with learning problems showed a significant reduction in a passively elicited cortical response (mismatch negativity), which reflects discrimination of acoustic elements (Kraus et al., 1996). Decrements in the magnitude of this speech-evoked response were related to impaired behavioral discrimination of certain

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speech contrasts. These contrasts reflect fine-grained onset frequency differences and are particularly susceptible to perceptual confusion in an impaired system (Bradlow et al., 1999; Elliott, Hammer, & Scholl, 1989; Godfrey, Syrdal-Lasky, Millay, & Knox, 1981; Reed, 1989; Tallal & Piercy, 1974; Tallal & Stark, 1981).

What is not known is whether differences between normally developing children and children with learning problems may be seen using other evoked potentials that reflect different and more elementary levels of sensory encoding. One approach to this issue is examining the speech-evoked P1/N1/N2 response complex. This waveform complex has been described for decades (Davis, 1939). It is characterized as a series of positive and negative waves that are robust and easily identifiable in adults. Adult waveform morphology, response to stimulus parameters and subject state are well described (Näätänen & Picton, 1987). Yet, despite the long history of discussion, many central questions related to the maturational progression of these evoked responses over the life span, their functional significance and whether they are distinctive in clinical populations experiencing perceptual or learning difficulties remain unanswered. The goals of this study are to shed light on these three issues.

Development of Neurophysiologic Responses

P1 • P1 is a dominant waveform in school-age children that can be reliably recorded using a variety of stimuli. Past research has shown gradual changes in waveform measures as an effect of age. For instance, P1 latency to brief click trains displayed an exponential decrease as age increased from 6 to 19 yr in 14 normal-hearing children (Ponton, Don, Eggermont, Waring, & Masuda, 1996). This finding was confirmed in a subsequent study using 143 normal children from 5 to 20 yr (Ponton, Eggermont, Kwong, & Don, 2000). Decreases in P1 latency (Kraus, McGee, Carrell, Sharma, Micco, & Nicol, 1993; Sharma, Kraus, McGee & Nicol, 1997) and amplitude during school-age years (Sharma et al., 1997) have also been shown in response to the speech stimulus /ba/.

N1 • Numerous studies using adult subjects have consistently portrayed the features of the auditory N1 response and have remarked about its robust nature. The adult N1 response generally occurs at 100 msec after stimulation and has been traditionally labeled N1b over midline (Giard, Perrin, Echallier, Thévenet, Froment, & Pernier, 1994; McCallum & Curry, 1980; Näätänen & Picton, 1987). In contrast, labeling this response in children has not been clear-cut. Most notably, comparisons are complicated by qualitative age-related differences in waveform morphology. For example, investigators have reported an unreliable N1 response in young children between ages 5 and 7 yr (Goodin, Squires, Henderson, & Starr, 1978; Martin, Barajas, Fernandez, & Torres, 1988) that becomes progressively consistent as age increases to 9 yr (Ponton et al., 2000) or adolescence (Courchesne, 1990; Sharma et al., 1997 [labeled N1a in this study]).

Reports also vary with respect to the degree of developmental changes seen in N1. Goodin et al. (1978) indicated that N1 latency did not differ between a group of children (7 to 15 yr) and a group of adults (16 to 76 yr) to binaurally presented tone bursts. The stability of the auditory N1 response was also supported by findings from Johnson (1989). In contrast, Martin et al. (1988) described a small nonsignificant decrease in N1 latency from 6 to 23 yr in response to binaural tone pips. Still others found significant decreases in N1 latency with stable amplitudes to both nonspeech (Polich, Ladish, & Burns, 1990; Ponton et al., 2000; Tonnquist-Uhlén, Borg, & Spens, 1995) and speech stimuli (Kraus et al., 1993; Sharma et al., 1997) across the school-age years.

P2 • Developmental changes reported for the P2 response elicited by simple stimuli have generally been minimal. Some researchers have shown that P2 latency increases with age (Goodin et al., 1978; Iragui, Kutas, Mitchiner, & Hillyard, 1993), whereas others have reported no maturational changes in the P2 response (Barrett, Neshige, & Shibasaki, 1987). Still others have reported very different patterns of P2 response change as a function of age depending on electrode location. Recently, Ponton et al. (2000) reported that small increases in P2 latency to brief click trains were evident at central electrodes locations (C3, C4, and CZ) and minute decreases in P2 latency were seen over the Fz electrode. Anderer, Semlitsch, and Saletu (1996) also reported inconsistencies across electrode sites stating that that P2 latency increased to standard tones in anterior locations, but not at posterior sites.

N2 • Few studies have quantified the developmental patterns of the late negativity peaking at 200 to 250 msec that dominates the response complex in young school-age children. The latency of this response is considerably later than the adult N1 (vertex N1b response) and some researchers have stated that it may reflect different components (Csepe, Dieckmann, Hoke, & Ross, 1992; Sharma et al., 1997). New findings have suggested that the N1 response in children may actually correlate with the adult N2 response (Ponton et al., 2000). Thus, in this study, the late prominent negativity seen in children was labeled "N2." To nonspeech stimuli, N2 latency has

been described as exhibiting a positive correlation with age from 6 to 15 yr (Martin et al., 1988), 6 to 20 yr (Ponton et al., 2000) and 20 to 89 yr (Anderer et al., 1996). N2 amplitude increases in early childhood and then decreases from 11 to 20 yr (Ponton et al., 2000). A different pattern of development has emerged in response to speech. Sharma et al. (1997) reported that this late negativity, which the authors termed N1b, showed a significant decrease in latency with age and no amplitude effects.

Overall, reports describing the development of P1 are consistent. In contrast, the characterization of N1, P2 and N2 development varies and is difficult to decipher. Inconsistencies in these studies can be attributed to the lack of a well-defined N1 response in children, differences in experimental protocols (stimuli and task), small numbers of subjects and disparate age ranges.

Clinical Populations

Even less is known about P1/N1/P2/N2 development in children with learning and attention problems. In a study examining children with attention difficulties, Satterfield, Schell, and Backs (1987) reported that click-evoked P1 latency decreased significantly and N1 latency, P1 amplitude, P2 amplitude, P1/N1 amplitude and P2/N2 amplitude did not change significantly with age in 34 normal subjects and nine delinquent hyperactive children. In contrast, the pattern of development in 25 nondelinquent hyperactive children was less consistent. Another report suggested that N1 latency decreased in 20 normal school-age subjects as a function of age, but did not in 20 severely language-impaired children (Tonnquist-Uhlén, Borg, Persson, & Spens, 1996). Based on these limited data, it is unclear whether children with learning and attention difficulties differ from normal children in their P1/N1/P2/N2 response characteristics. Moreover, both of these studies used nonspeech stimuli. The lack of a distinct divergent pattern of development in children with learning and attention problems may be related to the choice of stimuli. Of interest here was the neurophysiologic representation of a complex, ecologically significant stimulus, particularly one that is perceptually vulnerable to disruption.

The focus of this investigation was to study the auditory complex P1/N1/N2 evoked by speech stimuli in a large group of normal school age children and a group of children with learning and attention problems. Specifically, this included 1) a description of the development of the response characteristics of P1, N1 and N2; 2) a comparison of the P1/N1/N2 complex between normal children and children with learning problems; and 3) an evaluation of the relationship of the P1/N1/N2 complex to auditory perception, including fine-grained speech-sound discrimination, and standardized measures of learning, auditory processing and listening comprehension.

METHODS

Subjects

Subjects were 216 school-age children, 10 young adults, and 10 senior adults. The normal group (WNL [within normal limits]) included 1) children between the ages of 5 and 15 yr (N = 130, 70 female, 60 male) with no history of learning or attention problems based on a detailed parent questionnaire; moreover, children displayed scores within normal limits (including no discrepancy between ability and achievement) on all standardized tests of learning and academic achievement described below; 2) young adults between the ages of 19 and 27 yr (N = 10, all female); and 3) senior adults between the ages of 55 and 78 yr (N = 10, all female). Young and senior adult data were obtained from Bellis, Nicol, and Kraus (2000). The group with learning problems (LP) included children between the ages of 8 and 15 yr (N = 86, 21 female and 65 male) previously diagnosed as having a learning disability (LD; N = 35, 12 female and 23 male), attention deficit disorder (ADD; N = 30, four female and 26 male) or both (ADLD; N = 21, five female and 16 male). Juvenile subjects demonstrated normal intelligence measured by the Brief Cognitive Scale (Cognitive Ability [IQ] measure) of Woodcock-Johnson Psychoeducational Battery (scores >85). All participants including children, young adults and senior adults demonstrated normal hearing sensitivity (<25 dB HL) from 500 to 4000 Hz.

Differences in the gender and age distribution in the two groups of children (WNL and LP) should be noted. In the LP group, unequal male/female representation was attributed to the male-dominant nature of the disorders. The elevated age range in the LP group was a result of the age of diagnosis. Typically, a diagnosis of learning impairments is made in the school-age years after academic difficulties arise. Finally, the senior adults in the normal group were female to control for normal peripheral hearing sensitivity.

Neurophysiologic Experiment

Stimuli • /Ga/ was a five-formant syllable of 100 msec total duration. The stimulus was synthesized using a KLATT (1980) software serial synthesizer. The fundamental frequency began at 103 Hz, increased linearly to 125 Hz in 35 msec and then

decreased to 80 Hz in 55 msec. Voicing amplitude was constant for 80 msec and fell linearly to 0 in the last 10 msec of the stimulus. F1 and F2 had starting frequencies of 220 Hz and 1700 Hz and then transitioned linearly to 720 Hz and 1240 Hz in 40 msec. F3, F4 and F5 were steady state frequencies of 2500 Hz, 3600 Hz and 4500 Hz, respectively. This specific stimulus was chosen because it has been previously shown that children with learning problems may have difficulty perceiving stop consonants in general (reviewed in Bradlow et al., 1999) and this stimulus specifically (Kraus et al., 1996).

Electrophysiologic Measures • Electrophysiologic responses were obtained using Cz active, a forehead ground and a nasal reference electrode. Eye blinks and movements were monitored using a bipolar electrode montage (supraorbital-to-lateral canthus) and were off-line rejected at ($+100\mu V$/$-100\mu V$) (Berg & Davies, 1988; Picton, van Roon, Armilio, Berg, Ille, & Scherg, 2000). The recording window included a 90 msec prestimulus and a 500 msec poststimulus period, with sampling rates of 1000 points/sec. Electrophysiologic responses were off-line analog bandpass filtered from 0.1 to 100 Hz (12 dB/octave roll-off). Approximately 2500 responses were averaged for each individual. These methodologic techniques were identically applied to the young and senior adult data.

During the electrophysiologic acquisition period, subjects sat comfortably in a reclining chair within an electrically shielded, sound-attenuated booth and viewed a videotaped movie of their choice. Videotape audio levels were set below 40 dB SPL (A-weighted). Experimental speech stimuli were presented to the right ear through an ER-3 insert earphone at a level of 75 dB SPL. Stimuli were presented at a rate of 1.7 per second with an onset-to-onset interval of 590 msec. All subjects were instructed to ignore the sounds and to sit as quietly as possible. None of the subjects experienced difficulty complying with this instruction. The duration of the test session was approximately 1½ hr.

Data Analysis • For each subject, individual grand average waveforms were computed. Latency (P1, N1, N2) and amplitude (P1 relative to baseline [P1], N1 relative to baseline [N1], N2 relative to baseline [N2], P1 to N1 [P1/N1], P1 to N2 [P1/N2]) measures of auditory evoked potentials were visually identified for all subjects by three experienced testers. P1 was identified as the relative positivity occurring within the range of approximately 50 to 100 msec. N1 was considered the earlier negativity between 110 to 160 msec seen in all of the adults and in some children. The large negativity that dominated the waveform complex in most of the children and some young adults within the range of 175 to 275 msec was labeled N2 for this study.

It should be noted that examination of the P2 obligatory response was excluded from this study because the majority of the subjects across age groups did not exhibit a reliable P2 response. This may be explained by short-term habituation created by the fast stimulus presentation rate and by the type of stimulus used here. Research has shown that the rapid presentation of auditory stimuli (with inter-stimulus intervals of 2 sec or less) results in a marked reduction of N1-P2 amplitude (Davis, Mast, Yoshie & Zerlin, 1966). Moreover, others have stated that the degree of short-term habituation may be stimulus-specific. For instance, Woods and Elmasian (1986) demonstrated that the amplitude of the P2 response elicited by tones and complex tones was larger than for vowels and consonant-vowel-consonant stimulus tokens. Therefore, both of these methodologic factors may explain the apparent absence of the P2 response across subjects in this study.

Fine-Grained Discrimination

A continuum of synthetic speech syllables (/da/-/ga/) was used to assess the speech perception abilities of children in this study. These specific stimuli were chosen because it has been previously shown that children with learning problems may have difficulty perceiving these speech sounds (Kraus et al., 1996). The experiment was conducted in an electrically shielded, sound-attenuated booth. Stimuli were presented binaurally through Sennheiser HD 540 earphones at 75 dB SPL. Using an adaptive tracking algorithm (Parameter Estimation Sequential Tracking) (Taylor & Creelman, 1967) with a four-interval AX discrimination task, just noticeable differences were obtained. Ideal examples of the syllables served as endpoints of the continua and values of the synthesis parameters were interpolated linearly to generate the intermediate stimuli (Carrell, Bradlow, Nicol, Koch, & Kraus, 1999; Kraus et al., 1996; Walley & Carrell, 1983). The continuum (/da/ to /ga/) reflected a change in the third formant (F3) onset frequency. F3 changed from 2580 (anchor stimulus) to 2180 Hz in 40 steps of 10 Hz each.

Each stimulus presentation trial block consisted of two stimulus pairs. One pair contained two of the same stimuli (/da-da/) and the other pair contained the same stimulus and a different stimulus (/da-ga/). The subjects were required to indicate whether the stimuli in the first or second pair were different. Following a correct response, stimuli in the different pair comprised tokens on the continuum that were closer together in F3 onset frequency. Conversely, an incorrect response yielded a subsequent stimulus

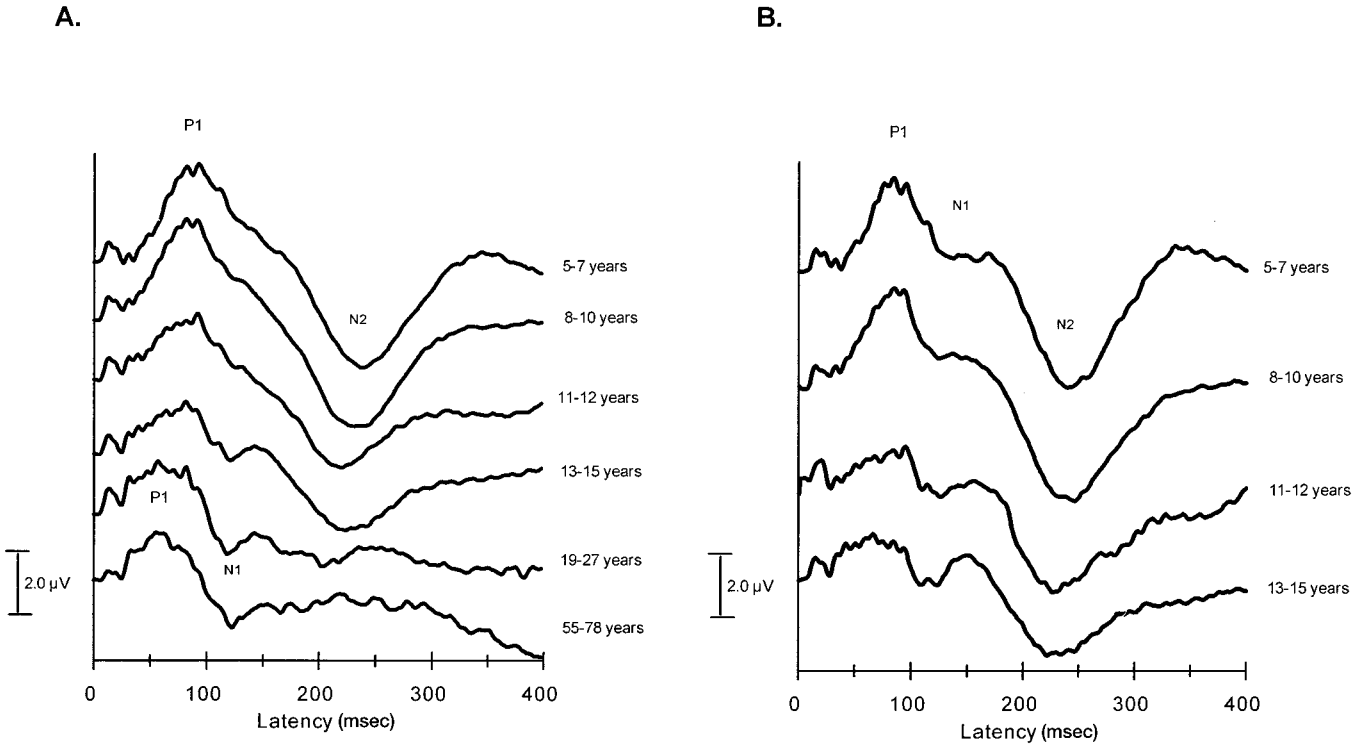

Figure 1. (A) Grand average P1/N1/N2 responses elicited by /ga/ for normal subjects in six age groups (Cz electrode location). The top trace is the response recorded in children between 5 and 7 yr. The bottom trace is the response recorded in senior adults between 55 and 78 yr. Intermediate age ranges also are plotted. (B) Grand average P1/N1/N2 responses elicited by /ga/ for normal children in four age groups (5 to 7, 8 to 10, 11 to 12, 13 to 15 yr) at a Cz electrode location. Only those children with a measurable N1 response are included.

pair that was more disparate in F3 onset frequency (more discriminable). The same/different stimulus pair presentation order was randomized within a test trial. Feedback was provided immediately after each test trial. The average test session was completed in 1 hr. A detailed description of stimuli, behavioral test procedures and data from 134 normal children can be found in Kraus, Koch, McGee, and Nicol (1999).

Learning and Academic Achievement

Portions of the Woodcock-Johnson Psychoeducational Battery-Revised (W-J PEB-R, 1989) were administered to assess performance on auditory and visual processing skills in the children. These subtests included: Memory for Words, Incomplete Words, Sound Blending, Sound Patterns, Listening Comprehension, Auditory Processing (composite score based on Incomplete Words, Sound Blending and Sound Patterns subtests), Cross-Out (a measure of visual processing speed), and Brief Cognitive Scale (cognitive ability [IQ] measure, W-J PEB, 1977). The Wide Range Achievement Test (Wilkinson, 1993) was used to assess single word Reading and Spelling abilities. All standardized learning measures produced standardized scores that were age-normed.

RESULTS

Normal P1/N1/N2 Development

Specifically examined were whether latency and amplitude of P1, N1 and N2 change with age in the normal population and whether boys and girls develop at different rates. Figure 1a illustrates grand average responses of the normal population across six age ranges (5 to 7, 8 to 10, 11 to 12, 13 to 15, 19 to 27, and 55 to 78 yr). Visual inspection of the P1/N1/N2 complex shows significant change in waveform morphology across age. Changes can be described in two ways. First, there appears to be a maturational progression in the presence/absence of response components. As detailed below, P1 was present in all groups, N1 was consistently identified in the adult groups, and N2 was present in the children. Second, latency and amplitude changed with age. In Table 1, normative data on mean and standard deviations of P1, N1, and N2 latency and

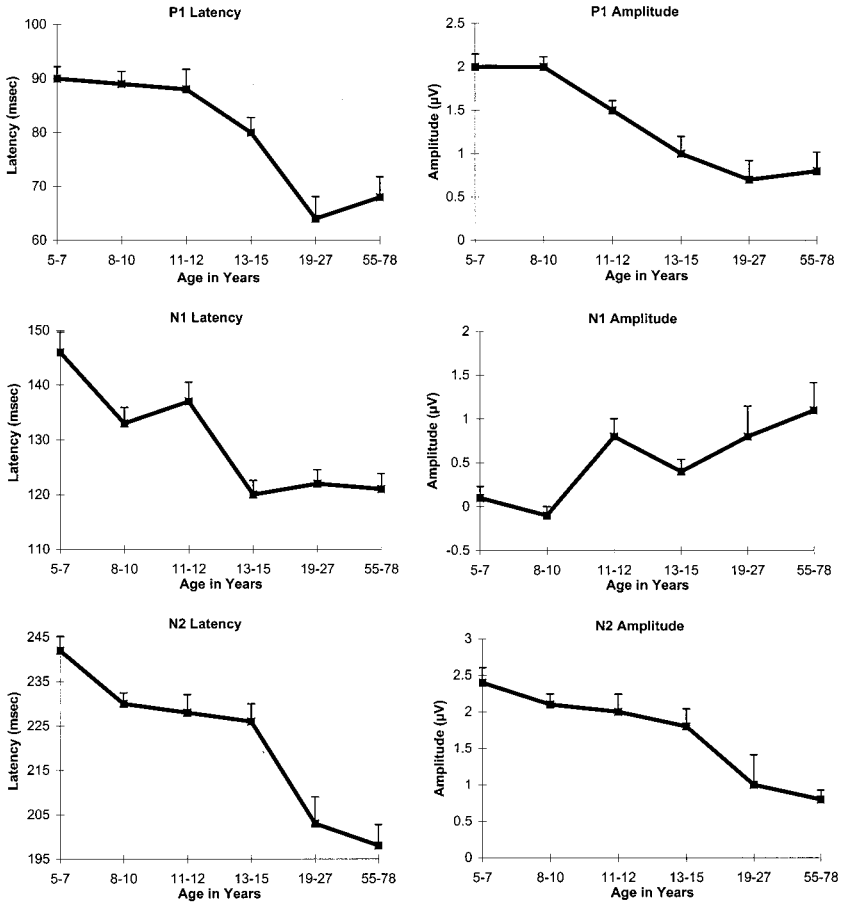

Figure 2. Mean latencies and amplitude (with standard errors) for P1, N1, and N2 are plotted across the entire age span studied.

amplitudes for each age group are provided. The number of subjects used in the calculation of these tabled values can be found in the description of the 1-way analysis of variance (ANOVA) results detailed below. In Figure 2, the means and standard errors for each response component are plotted. This figure provides a visual representation of data presented in Table 1.

P1 • P1 was present and easily identifiable in each age group. In the youngest children (5 to 7 yr), P1 appeared as a large positivity occurring at approximately 90 msec with an amplitude of 2 μV. As age increased from 5 to 78 yr, both latency and amplitude significantly decreased to 68 msec and 1.3 μV, respectively (age by latency, r = −0.41, p = 0.001; age by baseline amplitude r = −0.33, p = 0.001). Significant changes in P1 latency and amplitude also occurred at more restricted age ranges (5 to 15 yr; age by latency, r = −0.20, p = 0.019; age by baseline amplitude, r = −0.45, p = 0.001 and 8 to 15 yr; age by latency, r = −0.21, p = 0.030; age by baseline amplitude r = −0.49, p = 0.001).

To further understand developmental changes, 1-way ANOVAs were performed across six age groups (5 to 7 [N = 29], 8 to 10 [N = 47], 11 to 12 [N = 29], 13 to 15 [N = 25], 19 to 27 [N = 10], and 55 to 78 [N = 10] yr). The analysis revealed a significant age effect for latency [F(5,144) = 7.55, p = 0.001] and amplitude [F(5,144) = 9.55, p = 0.001]. Follow-up measures using the Scheffé test showed

TABLE 1. Means and standard deviations (in parentheses) for latency and amplitude of response components in normal subjects across the age span.

Age in Years	P1 Latency	P1/Baseline Amplitude	N1 Latency	N1/Baseline Amplitude	N2 Latency	N2/Baseline Amplitude	P1 to N1 Amplitude	P1 to N2 Amplitude
5–7	90 (13)	2.0 (0.8)	146 (20)	0.1 (0.7)	242 (17)	2.4 (0.9)	2.1 (0.6)	4.4 (1.1)
8–10	89 (17)	2.0 (0.8)	133 (22)	−0.1 (0.8)	230 (17)	2.1 (0.9)	2.0 (0.1)	4.2 (1.1)
11–12	88 (20)	1.5 (0.7)	137 (20)	0.8 (1.1)	228 (22)	2.0 (1.3)	1.8 (1.3)	3.4 (1.0)
13–15	80 (15)	1.0 (1.0)	120 (13)	0.4 (0.5)	226 (20)	1.8 (1.2)	1.3 (0.8)	2.7 (1.1)
19–27	64 (13)	0.8 (0.5)	122 (9)	0.8 (0.9)	203 (21)	1.0 (1.1)	1.7 (0.7)	2.2 (0.9)
55–78	68 (13)	1.3 (1.1)	121 (9)	1.1 (1.0)	198 (7)	0.8 (0.2)	2.5 (1.1)	1.4 (0.2)

TABLE 2. Normal development; significant post hoc (Scheffé) analyses are shown ($p < 0.05$*, $p < 0.01$). The data represented in this table are symmetric about the diagonal. Axes indicate age in years.**

	P1 Latency					
	55–78	19–27	13–15	11–12	8–10	5–7
5–7	**	**	*			
8–10	**	**	*			
11–12	**	**				
13–15	*	**				
19–27						
55–78						

	P1 Amplitude					
	55–78	19–27	13–15	11–12	8–10	5–7
5–7	**	**	**	**		
8–10	**	**	**	**		
11–12		*	*			
13–15						
19–27						
55–78						

	N1 Latency					
	55–78	19–27	13–15	11–12	8–10	5–7
5–7	**	**	**		*	
8–10			**			
11–12	*	*	**			
13–15						
19–27						
55–78						

	N1 Amplitude					
	55–78	19–27	13–15	11–12	8–10	5–7
5–7	**	*		*		
8–10	**	**		**		
11–12						
13–15	*					
19–27						
55–78						

	N2 Latency				
	19–27	13–15	11–12	8–10	5–7
5–7	**	**	**	**	
8–10	**				
11–12	**				
13–15	**				
19–27					

that P1 latency did not differ significantly between age groups comprising the young school-age years (5 to 7, 8 to 10, 11 to 12 yr). Yet, the P1 latency values of the 13 to 15, 19 to 27, and 55 to 78-yr-olds were significantly shorter than 5 to 7 and 8 to 10-yr-olds. P1 latency was similar between the 19 to 27 and 55 to 78 yr age groups. P1 amplitude was not significantly different between 5 to 7 and 8 to 10-yr-olds, yet began to decrease as a function of age. For instance, P1 amplitude measured in 5 to 7 and 8 to 10-yr-olds was significantly larger than the response seen in 11 to 12, 13 to 15, 19 to 27, and 55 to 78-yr-olds. There was no difference in P1 amplitude in across teen and adult years (13 to 15, 19 to 27, and 55 to 78 yr). In Table 2, results from these analyses indicating only significant age-group differences are shown. The test results are symmetric about the diagonal.

N1 • In contrast to P1, N1 was not present in all groups. The presence of N1 was variable in children, but consistently seen in the young and senior adults. There was a maturational progression in the appearance of N1 during the school-age years. This statement is based on a measured increase in N1 presence from 45%, 50%, 55% and 60% of subjects in the age ranges of 5 to 7 yr, 8 to 10 yr, 11 to 12 yr, and 13 to 15 yr, respectively. N1 was measured in 100% of adults.

Once present, N1 underwent developmental changes in latency and amplitude shown in Figure 1b. For all analyses, only individuals exhibiting an N1 response were included. N1 first appeared as a small indentation on the negative slope from P1 to N2 at approximately 146 msec with an amplitude of 0.1 μV. As age increased from 5 to 78 yr, N1 latency decreased to 121 msec and amplitude increased to 1.1 μV until the waveform resembled the classically defined adult response (age by latency, $r = -0.26$, $p = 0.014$; age by baseline amplitude, $r = 0.33$, $p = 0.001$). Significant Pearson r correlation coefficients can also be seen at more restricted age ranges (5 to 15 yr; age by latency, $r = -0.38$, $p = 0.001$; age by baseline amplitude, $r = 0.29$, $p = 0.021$ and 8 to 15 yr; age by baseline amplitude, $r = 0.31$, $p = 0.024$).

One-way ANOVAs were performed across six age groups (5 to 7 [$N = 14$], 8 to 10 [$N = 21$], 11 to 12 [$N = 15$], 13 to 15 [$N = 15$], 19 to 27 [$N = 10$], and 55 to 78 [$N = 10$] yr) for N1 latency and N1/baseline amplitude. The analysis revealed significant age effects for latency [$F(5,79) = 5.48$, $p = 0.001$] and amplitude [$F(5,79) = 4.87$, $p = 0.001$]. Post hoc measures (Scheffé) indicated that the greatest degree of change for latency and amplitude occurred in the early school age years between 5 and 12 yr. N1 latency appeared adult-like by 13 to 15 yr. There were no latency differences between the 13 to

15- yr-old group, young and senior adults. Instead, N1 amplitude increased from age 5 to well into the teenage years. N1 amplitude was stable by adulthood.

N2 • N2 was also not present in all groups. The response was consistently seen in school-age children, but was variable in adults. In fact, a maturational pattern of N2 disappearance occurred from young to senior adulthood. For instance, N2 was measurable in 95% of children, 80% of young adults and 20% of senior adults.

In young children, N2 was seen as a large, dominant negativity at approximately 242 msec with an amplitude of 2.4 μV. As age increased, N2 latency and amplitude decreased to 198 msec and 0.8 μV, respectively ($r = -0.41$, $p = 0.001$), until it was virtually absent in the senior adult group. At more restricted age ranges different periods of maturation emerged. For instance, Pearson r correlation coefficients for the age range, 5 to 15 yr, revealed that N2 latency ($r = -0.30$, $p = 0.001$) significantly correlated with age. Yet, between 8 and 15 yr, N2 latency and amplitude did not (age by latency, $r = -0.10$, $p = 0.286$; age by baseline amplitude, $r = -0.11$, $p = 0.270$).

One-way ANOVAs were performed across five age ranges (5 to 7 [$N = 27$], 8 to 10 [$N = 47$], 11 to 12 [$N = 25$], 13 to 15 [$N = 24$], and 19 to 27 [$N = 8$] yr). Because most of the senior adults did not have a measurable N2 response, this group was not included in the analysis. A significant age effect for latency [$F(4,126) = 6.78$, $p = 0.001$] was shown. No significant age effects were seen for N2 amplitude [$F(4,126) = 2.15$, $p = 0.078$]. Post hoc analyses (Scheffé) revealed that N2 latency significantly decreased between the ages of 5 and 10 yr, was stable between 10 and 15 yr and then decreased again during young adulthood.

Gender • To examine maturational changes related to gender, P1/N1/N2 latency and amplitude were compared between 52 normal boys and girls age-matched to within 1 yr. No significant group differences were found for any AEP components (Latency [P1: $t(51) = 0.87$, $p = 0.385$; N1: $t(24) = 0.18$, $p = 0.852$; N2: $t(51) = 0.001$, $p = 0.999$]; Amplitude [P1: $t(51) = 1.0$, $p = 0.319$; N1: $t(24) = 0.14$, $p = 0.885$; N2: $t(51) = 1.04$, $p = 0.303$; P1N1: $t(24) = 0.04$, $p = 0.966$; P1N2: $t(51) = 3.9$, $p = 0.160$]). A Bonferroni correction was applied to this set of analyses. Thus, the same normative data can be used for school-age boys and girls.

In summary, age-related changes were observed in the morphology, amplitude, latency and detectability of all P1/N1/N2 components.

LP Development

Specifically examined were whether latency and amplitude of P1, N1 and N2 change with age in the population of children with learning problems and whether boys and girls develop at different rates. Moreover, comparisons of AEP components were made between the WNL and LP groups.

Maturational patterns for the group of children with learning problem (LP) as a whole were similar to the normal group (WNL) for the 8 to 15 yr age range. For instance, significant relationships between age and P1 latency, N1 latency, P1/baseline amplitude and P1/N2 amplitude were present. Correlations between age and N2 latency, N1/baseline amplitude, N2/baseline amplitude and P1/N1 amplitude all failed to reach significance. Pearson r correlation coefficients differed from the normal group for N1/baseline amplitude only. That is, the normal group showed a significant increase in N1/baseline amplitude within this age range, whereas the LP group did not. In Table 3, age by latency and amplitude Pearson r correlation coefficients and significance values are shown.

To examine developmental patterns for each diagnostic category, the LP group was further divided into three diagnostic categories. These included: children with learning disabilities (LD), children with attention deficit disorder (ADD), and those with a comorbid diagnosis, attention deficit disorder and learning disabilities (ADLD). Pearson r correlation coefficients yielded some significant findings for some of the groups again shown in Table 3. For instance, learning-disabled children demonstrated a significant decrease in P1 latency, N1 latency, N2/baseline amplitude and P1/N2 amplitude with increasing age. Children with attention deficit disorder exhibited significant decreases in N1 latency and no significant changes in amplitude. Finally, children with a comorbid diagnosis exhibited a significant decrease in P1/baseline and P1/N2 amplitude as age increased.

Gender • To examine maturational changes related to gender, P1/N1/N2 latency and amplitude, were compared between 21 boys and girls with learning problems age-matched to within 1 yr. No significant group differences were found for any P1/N1/N2 component (Latency [P1: $t(20) = 0.30$, $p = 0.762$; N1: $t(10) = 0.11$, $p = 0.910$; N2: $t(20) = 0.85$, $p = 0.404$]; Amplitude [P1: $t(20) = 0.69$, $p = 0.493$; N1: $t(10) = 0.29$, $p = 0.771$; N2: $t(20) = 0.933$, $p = 0.361$; P1N1: $t(10) = 0.19$, $p = 0.849$; P1N2: $t(20) = 0.33$, $p = 0.740$]). A Bonferroni correction was applied to this set of analyses.

TABLE 3. Age by latency and age by amplitude Pearson r correlation coefficients ($p < 0.05$*, $p < 0.01$).**

		Latency			Amplitude				
		P1	N1	N2	P1	N1	N2	P1 to N1	P1 to N2
WNL	n	150	85	133	150	85	133	85	133
(5–65 yr)	r	−0.34**	−0.26**	−0.34**	−0.33**	0.34**	−0.23**	−0.01	−0.41**
WNL	n	101	51	96	101	51	96	51	96
(8–15 yr)	r	−0.21*	−0.27*	−0.1	−0.5**	0.31*	−0.11	−0.15	−0.44**
WNL	n	130	65	123	130	65	128	65	123
(5–15 yr)	r	−0.2**	−0.38**	−0.26**	−0.45**	0.29*	−0.19*	−0.19	−0.46**
LP	n	86	49	82	86	49	82	49	82
(8–15 yr)	r	−0.43**	−0.34**	−0.09	−0.27**	0.16	−0.15	−0.13	−0.27**
LD	n	35	17	34	35	17	34	17	34
(8–15 yr)	r	−0.54**	−0.52*	0.06	−0.20	−0.27	−0.35*	−0.44	−0.41**
ADD	n	31	21	29	31	21	29	21	29
(8–15 yr)	r	−0.33	−0.45*	−0.10	−0.2	0.18	0.06	0.08	−0.05
ADDLD	n	21	13	19	21	13	19	13	19
(8–15 yr)	r	−0.41	−0.14	−0.36	−0.52**	0.13	−0.22	−0.49	−0.54**

WNL = within normal limits; LP = learning problems.

Developmental Comparisons

To evaluate developmental differences in the P1 and N2 response components between the WNL and LP group, latency and amplitude measures were compared using a 2 (groups) × 3 (age levels) ANOVA. Each sample (WNL and LP) was divided into three age-matched groups (A: 8 to 10 yr, $N = 32$, B: 11 to 12 yr, $N = 27$, C: 13 to 15 yr, $N = 17$). Separate 2 (groups) × 3 (age levels) ANOVA statistical procedures were performed for each evoked potential response component (P1 and N2).

Analyses revealed significant age effects only for P1 latency [$F(2,135) = 3.19$, $p = 0.043$] and P1/baseline amplitude [$F(2,135) = 8.19$, $p = 0.001$]. In addition, nonsignificant group effects and interactions for P1 and N2 were seen. In Figure 3, results of these analyses are shown. Post hoc analyses using paired *t*-tests with a Bonferroni correction revealed that P1 latency differed between the age groups, A and B [$t(116) = 2.28$, $p = 0.040$], B and C [$t(86) = 2.72$, $p = 0.007$], and A and C [$t(96) = 5.09$, $p = 0.001$]. P1/baseline amplitude differed between the age groups, A and B [$t(116) = 2.61$, $p = 0.010$], B and C [$t(86) = 2.87$, $p = 0.005$], and A and C [$t(96) = 4.96$, $p = 0.001$].

Group comparisons for the N1 response were made using a subset of children from the above-mentioned ANOVA. Because the N1 response is inconsistent in young children, only those with a measurable N1 response were used. Latency and amplitude were compared between 25 WNL and LP children using an age-matched *t*-test. There were no group differences for any N1 response measure (Latency: $t = 1.69$, $p = 0.103$; Amplitude: $t = 0.852$, $p = 0.402$).

Examining subgroups of LP children with age-matched paired *t*-tests revealed no P1/N1/N2 complex differences between: WNL and LP ($n_{pairs} = 76$), WNL and ADD ($n_{pairs} = 28$), WNL and LD ($n_{pairs} = 31$), WNL and ADD/LD ($n_{pairs} = 21$), WNL and a subset of LP children who were classified as reading disabled based on a 15 point difference between scores on the Brief Cognitive Scale (IQ) and Reading ($n_{pairs} = 19$), and ADD and LD ($n_{pairs} = 25$). A Bonferroni correction was applied to this set of analyses.

In summary, children with learning problems showed similar maturational changes in P1/N1/N2 components as compared with normal children. There were no significant differences in the developmental time course among the diagnostic categories including, LD, ADD, ADD/LD and reading disabled. Moreover, similar findings were evident in male and female children.

Functional Significance of P1/N1/N2

First, it was of interest to determine whether the P1/N1/N2 complex had a relationship to basic fine-grained speech discrimination and/or to cognitively influenced measures of auditory processing. Second, analyses were performed to evaluate how well a

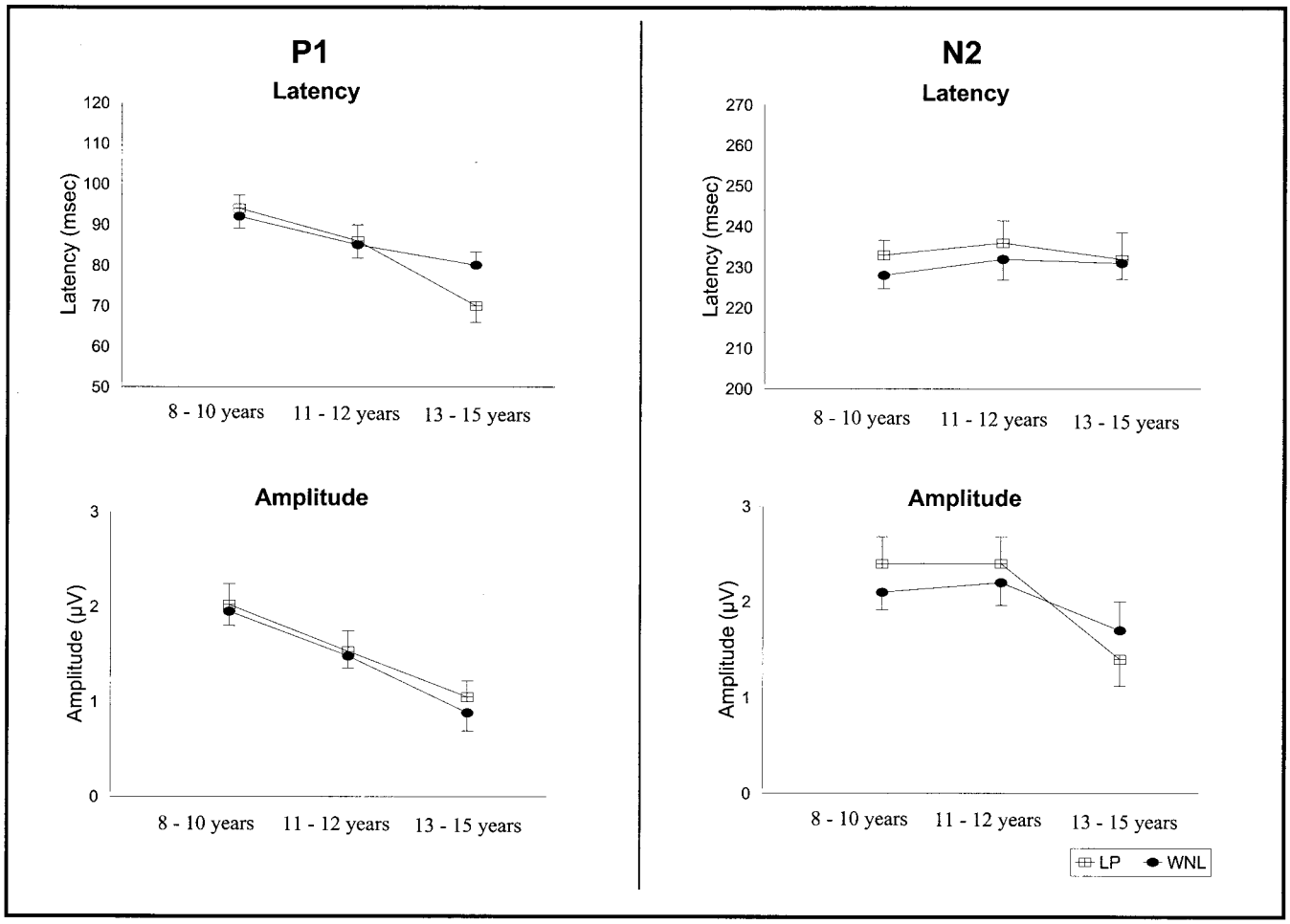

Figure 3. The P1 and N2 response components, latency and amplitude, were compared between the normal group and the learning problems (LP) group using a 2 (group) × 3 (age level) analysis of variance. Each sample was divided into three age groups (8 to 10, 11 to 12, and 13 to 15 yr). No significant differences or interactions were found across the response components from the two samples. Mean and standard errors for P1 latency/amplitude and N2 latency/amplitude are shown. WNL = within normal limits; LP = learning problems.

subject's performance on these subtests would predict basic latency ("early" or "late") and amplitude ("small" or "large") characteristics of the P1/N1/N2 potentials.

Fine-Grained Discrimination • The relationship between P1/N1/N2 and behavioral discrimination along a (/da/-/ga/) continuum was first examined using Pearson r correlation coefficients. P1/N1/N2 measures did not correlate significantly with performance on behavioral discrimination for the WNL (N = 123), the LP (N = 77) or both groups, WNL and LP, combined (N = 200).

The P1/N1/N2 response components were also compared between 20 good and poor perceivers on fine-grained speech discrimination measures using an age-matched t-test. The group of good perceivers was constrained to normal subjects who scored 1 SD better than the mean just noticeable difference score for the /da/ to /ga/ continuum. The group of poor perceivers consisted of LP subjects who scored 1 SD less than the mean just noticeable difference score. Normal mean scores were obtained from Kraus et al. (1999). Both groups were further restricted to children who demonstrated mastery of the Parameter Estimation Sequential Tracking procedure on a /ba/ to /wa/ continuum used in other studies. This ensured that all children could perform the task adequately. Statistical analyses indicated no significant group differences in evoked response components between good and poor perceivers (Latency [P1: t (19) = 1.11, p = 0.276; N1: t (12) = 0.59, p = 0.558; N2: t (18) = 0.18, p = 0.860]; Amplitude [P1: t (19) = 0.44, p = 0.660; N1: t (12) = 0.33, p = 0.744; N2: t (18) = 0.05, p = 0.956; P1N1: t (12) = 0.53, p = 0.596; P1N2: t (18) = 0.90, p = 0.375]). A Bonferroni correction was applied to this set of analyses. Thus there appears to be no relationship between P1/N1/N2 and fine-grained speech-sound perception.

TABLE 4. Significant Pearson r correlation coefficients ($p < 0.05$*, $p < 0.01$).**

		Latency			Amplitude				
		P1	N1	N2	P1	N1	N2	P1/N1	P1/N2
WNL	*N* =	96	46	89	96	46	89	46	89
Test									
Brief Cognitive Scale						+0.46**	+0.20*	+0.39**	
Cross Out				−0.23*		−0.35*			
Incomplete Words				−0.21*					

		Latency			Amplitude				
		P1	N1	N2	P1	N1	N2	P1/N1	P1/N2
LP	*N* =	67	37	63	67	37	63	37	63
Test									
Brief Cognitive Scale		−0.27*							
Incomplete Words					−0.23*				−0.28**
Listening Comprehension						−0.37*	−0.32**	−0.42**	
Auditory Processing								−0.33*	
Spelling		−0.29**							

Measures of Learning and Academic Achievement • The relationship between P1/N1/N2 and measures of learning were first examined using Pearson r correlation coefficients. Because the N1 response is inconsistent in young children, only those with a measurable N1 response were used in this analysis. In the normal group, Incomplete Words and Cross-Out were significantly correlated with N2 latency. Correlations were also seen in the group of children with learning problems. Incomplete Words, Listening Comprehension, and Auditory Processing were all significantly correlated with P1/N1/N2 amplitude measures. All significant correlations were negative; that is, high scores were associated with low amplitude evoked responses. Also for the LP group, Spelling correlated significantly with P1 latency. Finally, evoked potential parameters correlated with the Brief Cognitive scale in both groups. In Table 4, only significant Pearson r correlation coefficients are detailed. It is interesting to note that different learning measures were significant in the normal group versus the LP group. Moreover, most of the correlations in the LP group were negative (69 out of 80), whereas correlations were both positive (48 out of 80) and negative (32 out of 80) in the normal group, irrespective of whether the correlations reached statistical significance or not.

Another way of assessing relationships between P1/N1/N2 and learning measures was to determine whether correct classification of child's P1/N1/N2 response components (Latency ["early" or "late"]/ Amplitude ["small" or "large"]) could be predicted at a rate better than chance by performance on the standardized measures of learning. The median values for P1, N1 and N2 latency and P1/N2 amplitude were determined for all subjects (WNL and LP combined) between the ages of 8 to 15 yr. The large group was then divided into two smaller groups based on the median value of each particular evoked response component being evaluated. Thus one group contained subjects with measures less than the median value and the other group consisted of subjects with measures greater than the median value regardless of diagnostic category.

A discriminant function analysis (Tabachnick & Fidell, 1996) revealed that performance scores on the five auditory based learning measures (predictor variables: Incomplete Words, Sound Blending, Sound Patterns, Listening Comprehension and Auditory Processing) were predictors of N2 latency group affiliation (median = 230 msec, <230 msec = "early," >230 msec = "late") [$F(5,131) = 2.66$, $p = 0.020$]. Results indicated that children with an earlier N2 latency demonstrated higher scores on these learning measures. Based on the use of all five of these tests, children were correctly classified 65% of the time, a value significantly greater than chance (Binomial test, $Z = 3.6$, $p < 0.001$). Individually, each of the predictor variables was also significant in predicting N2 latency group affiliation (Auditory Processing [$F(1,131) = 8.5$, $p = 0.004$], Listening Comprehension [$F(1,131) = 7.2$, $p = 0.008$], Sound Patterns [$F(1,131) = 3.90$, $p = 0.050$], Sound Blending [$F(1,131) = 4.2$, $p = 0.041$], and Incomplete Words [$F(1,131) = 6.22$, $p = 0.014$]). The strongest single indicator of group affiliation was the child's score on Auditory Processing followed by Listening Comprehension, Sound Patterns, Sound Blending and Incomplete Words in descending order. Based on the single best predictor, Auditory Processing,

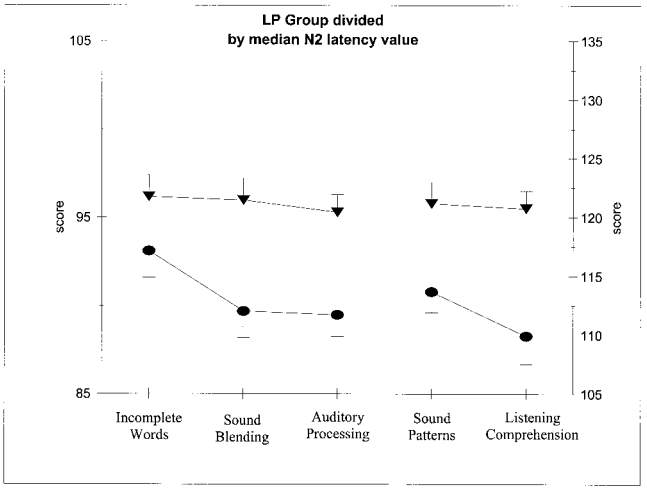

Figure 4. In the group of children with learning problems (LP), a discriminant analysis revealed that a child's scores on five auditory-based learning measures (Incomplete Words, Sound Blending, Sound Patterns, Listening Comprehension, and Auditory Processing) were a reliable predictor of N2 latency group affiliation. The two N2 latency groups were labeled "early" (N2 latency less than 230 msec), represented by filled triangles, and "late" (N2 latency greater than 230 msec), represented by filled circles. Findings indicated that LP children in the "early" group displayed significantly higher auditory-based learning scores compared with those children in the "late" group.

children would be correctly classified 60% (Binomial test, $Z = 2.5$, $p < 0.01$) of the time.

To design a test battery that would have the highest rate of correct N2 latency classification, eight additional discriminant function analyses were performed using various combinations of the five learning measures mentioned above. Results indicated that the best classification results for a two-test battery were obtained with Auditory Processing and Listening Comprehension ([$F(2,131) = 5.57$, $p = 0.005$], 64% classified, Binomial test, $Z = 3.4$, $p < 0.001$), a three-test battery was Auditory Processing, Listening Comprehension and Sound Patterns ([$F(3,131) = 3.91$, $p = 0.010$)], 66% classified, Binomial test, $Z = 3.8$, $p < 0.001$), and a four-test battery was Auditory Processing, Listening Comprehension, Sound Patterns and Incomplete Words ([$F(4,131) = 3.25$, $p = 0.014$], 67% classified, Binomial test, $Z = 4.1$, $p < 0.001$).

Similar Discriminant Function analyses were performed for the WNL and LP groups, separately. Results indicated that the five auditory-based learning measures were strongly predictive of N2 latency group for the LP subjects alone [$F(5,55) = 3.07$, $p = 0.016$], but not for the WNL subjects [$F(5,69) = 1.56$, $p = 0.182$]. In Figure 4, the relationship between N2 latency and auditory-based learning measures is shown for the LP children. Yet, in both groups, the single measure of Auditory Processing predicted N2 latency group affiliation (WNL: F [1,69] = 5.38, $p = 0.023$; LP: F [1,55] = 6.60, $p = 0.013$). P1 latency, N1 latency or P1/N2 amplitude could not be predicted from performance on any of the learning measures for WNL, LP or the combined groups.

DISCUSSION

In summary, the development of the auditory P1/N1/N2 complex continued through the school-age years into adulthood. Patterns were similar between children with learning and attention problems and normal healthy children. Moreover, findings elucidated the relationship between the P1/N1/N2 auditory complex evoked by speech stimuli and auditory function, learning and cognition. First, performance on five auditory subtests of standardized measures of academic achievement showed a predictive relationship with N2 latency for all subjects (in a combined group) and a single standardized measure of Auditory Processing reflected this same relationship for normal and LP children. Second, P1/N1/N2 parameters correlated with Cognitive Ability (IQ) measures in both groups. In the LP group, EP parameters also correlated with performance on standardized tests of Spelling, Auditory Processing and Listening Comprehension. Third, these measures did not correspond with performance on fine-grained speech discrimination tasks.

Normal Development

In general, the most notable developmental changes occurred in the overall waveform morphology and latency/amplitude measures of each P1, N1, and N2. First, P1 latency and amplitude demonstrated significant differences between school-age (5 to 7, 8 to 10 yr) and older years (13 to 15, 19 to 27, 55 to 78 yr). Beyond young adulthood, there were no significant developmental changes. Accordingly, it may be concluded that P1 is mature by approximately age 20. These findings are consistent with data published by Ponton et al. (1996, 2000) and Sharma et al. (1997). Second, N1 latency significantly decreased and became larger in amplitude (more negative) during the school-age years. It reached the adult value around 13 to 15 yr in a manner similar to that reported by others (Martin et al., 1988; Ponton et al., 2000; Sharma et al., 1997; Tonnquist-Uhlén et al., 1995). Third, N2 latency and amplitude decreased in early childhood and again in adulthood until it became essentially absent in seniors. N2 latency remained stable during the school-age years. Systematic decreases in N1b latency (analogous to N2) between the ages of 6 and 15 yr to

the speech stimulus /ba/ have been reported by Sharma et al. (1997).

Developmental changes in the underlying generating systems of these neurophysiologic responses may be responsible for the latency/amplitude changes and patterns of waveform emergence seen in childhood. First, age-related decreases in the latency of P1, N1 and N2 may be explained by simultaneous increases in myelination as well as improvements in synapse efficacy (Courchesne, 1990; Eggermont, 1988; Kraus et al., 1993; Pearce, Crowell, Tokioka, & Pacheco, 1989). Second, changes in intra and inter hemispheric connections, thought to contribute to the generation of the N1 response, may correspond to the emergence of N1 during the school age years (Mäkelä & McEvoy, 1996). Neuroanatomical data suggest that interhemispheric commisural fibers continue myelinating throughout the second decade of life and association fibers continue into the third decade (Courchesne, 1990). Finally, marked reduction in N2 amplitude in subjects over 55 yr may be linked to years of sensory experience that may streamline or improve the efficiency of neural transmission to such a degree that the involvement of additional higher level processes are no longer needed.

Gender • In this study, we did not find any differences between male and female children in the P1/N1/N2 auditory complex. Neuroanatomical studies have shown that the planum temporale in females is smaller than in males (Kulynych, Vlada, Jones, & Weinberger, 1994). Neurophysiologically, Tonnquist-Uhlén et al. (1995) demonstrated that boys (8 to 16 yr) displayed a nonsignificant tendency for larger N1 amplitudes than females. Meanwhile, Martin et al. (1988) reported no significant gender differences in N1 latency at any age group studied (6 to 7, 9 to 10, 12 to 14, 19 to 23 yr) and no significant amplitude differences between male and female children.

LP Development

The results from this study indicated no differences in the development of P1/N1/N2 between normal children and children with learning problems. Thus, differences in the rate of P1/N1/N2 latency and amplitude development do not appear to be distinctive in these two populations. This was the case for the LP group as a whole as well as for children comprising various diagnostic categories (LD, ADD, ADD/LD, and reading-disabled).

Relationship to Auditory Function, Learning, and Cognition

Numerous studies have thoroughly described the characteristic features, effects of stimulus manipulation, scalp topography and underlying generators associated with this waveform complex. Yet, to our knowledge, this is the first study to provide information regarding the functional significance. For instance, analyses showed that there was a relationship between tests of auditory function (Auditory Processing, Incomplete Words, Sound Blending, Sound Patterns, and Listening Comprehension) and N2 latency. Specifically, school-age children with higher scores on these auditory-based learning measures displayed earlier N2 latencies. These results were independent of age because the standardized learning scores are age-corrected and the N2 latency failed to show any developmental changes across 8 to 15 yr. Additionally in both groups, a child's score on the Auditory Processing subtest was highly predictive of N2 latency group affiliation ("early" or "late"). This finding highlights the potential value of the speech-evoked N2 response for identifying auditory processing deficits. Used in conjunction with other neurophysiologic and/or behavioral measures, viable clinical applications can be envisioned. Other factors supporting the utility of this response include the simplicity of data collection, small time requirements and the robust and easily identifiable nature of N2 in children.

P1/N1/N2 parameters were also significantly correlated with tests of Spelling, Auditory Processing and Listening Comprehension in the LP group. Moreover, these waveforms were related to performance on the Brief Cognitive Scale in both groups. Nevertheless, these significant correlations must be viewed cautiously. For instance, some significance can be achieved by simply performing a large number of correlations. Here, we performed 80 correlations for each diagnostic group (4 out of 80 would be expected by chance). Moreover, given the large number of subjects in this study, significance may be attained despite small correlations. Thus, although a number of the correlations were statistically significant, some of them may be spurious and most accounted for modest proportions of variance. In summary, these overall differences in the relationship between the P1/N1/N2 parameters and behavioral measures between the LP and normals are intriguing and need to be further investigated.

Second, the present findings indicate that there does not appear to be a relationship between P1/N1/N2 and fine-grained speech discrimination. Instead, the mismatch negativity evoked potential has been correlated with this behavioral auditory measure in similar populations (Kraus et al., 1996). Moreover, the present data show that P1/N1/N2 has a systematic developmental time course, whereas MMN area and duration are mature by school-age (Kraus et al., 1999). In addition, the generating

sources underlying the N1 response are different from the MMN (King, McGee, Rubel, Nicol, & Kraus, 1995; Kraus, McGee, Carrell, King, Littman, & Nicol, 1994; Näätänen & Picton, 1987; Sams, Kaukoranta, Hämäläinen, & Näätänen, 1991; Scherg, Vajsar, & Picton, 1989). Taken together, the evidence suggests that the P1/N1/N2 and MMN responses reflect different aspects of auditory function. Whereas the MMN reflects fine-grained discrimination and echoic memory processes (Näätänen, Pääviläinen, Alho, Reinikainen, & Sams, 1989), it appears that the P1/N1/N2 reflects developmentally sensitive, basic neural encoding of sound in the thalamo-cortical segment of the auditory pathway. The auditory information provided by the neurophysiologic processes underlying P1/N1/N2 may serve a wide-range of auditory perceptual functions ultimately leading to listening comprehension.

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