

Speech-Evoked Cortical Potentials in Children

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Abstract

Event-related potentials (ERPs) were obtained to synthesized speech stimuli in 16 school-aged children (7-11 years) and compared to responses in 10 adults. P_1 , N_1 , and P_2 event-related potentials were elicited by the phoneme /ga/. The mismatch negativity (MMN) was elicited by variants of /da/ and /ga/, which differ in the onset frequency of the second and third formant transitions. In general, the well-defined N_1/P_2 complex characteristic of the adult response, was not found in children. Waves P_1 and N_1 had longer peak latencies in children than in adults. Wave P_2 amplitude was smaller in children than in adults. In contrast to the often poorly delineated earlier cortical potentials, the MMN was well defined in children. Significant MMNs were obtained in all subjects tested. MMN magnitude (peak amplitude and area) was significantly larger in the children. No significant differences were found in peak latency and duration of the MMN in children compared to the adult response. Another negative wave occurring at 400 msec was also observed in response to the deviant stimuli. This negative wave occurred at a similar latency in adults and children and was significantly larger and more robust in children. Results support the view that development of ERPs does not involve a hierarchical process with respect to latency. That is, earlier occurring waves do not necessarily mature before later occurring waves. The latencies of P_1 , N_1 , and P_2 and overall morphology of these waves may provide a measure of maturation of central pathways. The early development of the MMN, its apparent robustness in school-aged children, and its reflection of the processing of acoustic differences in speech stimuli suggest its possible use in the assessment of central auditory function.

Key Words: Auditory evoked potentials (AEP), children, development, event-related potentials, mismatch negativity (MMN), speech

Certain event-related auditory evoked potentials (ERPs or AEPs) can be elicited passively, without requiring the subject to perform a task. The well-studied auditory brainstem and middle latency responses are in this group, as are P_1 , N_1 , P_2 , and the mismatch negativity (MMN), which are the

focus of the present investigation. The MMN (Näätänen et al, 1978) permits an objective analysis of sensory processing and discrimination of stimulus differences in patients who cannot consistently attend to stimuli.

Like the auditory brainstem response, waves P_1 , N_1 , and P_2 are elicited by repetitive acoustic stimuli, but these later potentials are less dependent on stimulus rise time and frequency and can be elicited by complex stimuli such as speech (Wood et al, 1971; Picton and Stuss, 1984; Woods and Elmasian, 1986). Wave P_1 , occurring at about 50 msec in adults, is alternately considered a middle latency or late auditory evoked potential depending on the time window and repetition rate with which it is elicited (Kraus and McGee, 1992). The N_1 - P_2 complex, originally described by Davis (1939), is

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ubiquitous in adults. There is some question as to its developmental sequence in children (Courchesne, 1990). As the N_1 wave is thought to consist of at least 3 distinctively generated subcomponents (Näätänen and Picton, 1987), these components may have distinctive developmental time courses as well.

The MMN is elicited by an "oddball" sequence in which a deviant stimulus occurs within a series of homogenous, or standard, stimuli (Näätänen et al, 1978). Again, these stimuli can be acoustically complex. The MMN is thought to reflect a neuronal mismatch process occurring between the input from a deviant stimulus and a sensory-memory trace, which has stored the physical features of the repetitive standard stimulus (Sokolov, 1975; Näätänen, 1990). In addition, the MMN, unlike other cognitive potentials, decays rapidly when interstimulus intervals are longer than 2 seconds (Näätänen et al, 1987). These features have linked the MMN to "engram" or immediate short term memory processes (Näätänen et al, 1989a, b).

The MMN has been obtained in response to minimal changes in acoustic parameters such as frequency, intensity, location, and duration (Sams et al, 1985; Näätänen et al, 1987, 1989a, b; Kaukoranta et al, 1989; Paavilainen et al, 1989) and can occur when the difference between the standard and deviant stimuli is near psychophysical threshold (Sams et al, 1985a; Kraus et al, 1993a; Sharma et al, 1993). The MMN has been elicited by speech stimuli (Aaltonen et al, 1987; Sams et al, 1990; Kraus et al, 1992a, 1993a, b; Sharma et al, 1993). As a neurophysiologic index of processing of small acoustic differences, MMN may be used to investigate mechanisms underlying auditory sensory discrimination. Clinical applications including assessment of auditory learning problems can be envisioned.

Of theoretical interest is how the development of the event-related potentials P_1 , N_1 , P_2 , and MMN compare with each other. Although there is a general trend toward the development of early waves before later waves, it is unlikely that a strict hierarchical process with respect to latency underlies ERP development (Eggermont, 1989; Courchesne, 1990) or ERP generation in general (Picton et al, 1977; Satya-Murti et al, 1983; Kraus et al, 1993c). Rather, developmental changes are likely to involve an interactive process where some potentials have independent developmental time courses. The developmental time course of these potentials may

provide information about the underlying generating systems and sensory processes. More practically, if clinical applications are to be envisioned for these ERPs in children, it is necessary to know the characteristics of the normal response in this population.

In this study, P_1 , N_1 , P_2 , and MMN evoked potentials were compared in school-aged children and adults in response to speech stimuli differing in well-specified acoustic features. Additionally, a negativity at 400 msec was described. MMN data have been reported previously in part (Kraus et al, 1992a). Unique to this communication are additional subjects and the statistical analyses used to define the MMN in groups and in individuals. Evaluation of the MMN in individual subjects is essential to its clinical application.

METHOD

Subjects were 16 healthy children, 7–11 years old (8 males, 8 females) with normal hearing thresholds (< 15 dB HL) from 500 to 8000 Hz. Also reviewed here, for comparison with the child data, are data from 10 adults (17–29 years old; 4 males, 6 females) (Kraus et al, 1992a).

Stimuli

Synthesized (Klatt, 1980) speech stimuli (sampling rate of 10,000 Hz) were variants of the voiced stop consonants /da/ and /ga/. The stimuli were composed of five formants and differed in the onset frequency of the second and third formant transitions. The speech stimuli were chosen from a nine-item continuum in which the second and third formants were varied systematically from /da/ to /ga/ (Walley and Carrell, 1983). The $P(c)$ max scores, a measure of behavioral discrimination (Green and Swets, 1974), for stimuli 3 and 8 in the continuum were 78 percent for children and 98 percent for adults. These two stimuli, referred to as /da/ and /ga/, were used to elicit the ERPs in this study.

The starting frequencies of the second and third formants were 1685 Hz and 2625 Hz for the /da/ stimulus and 1647 Hz and 2187 Hz for the /ga/ stimulus. The center frequencies of the formants for the steady-state vowel /a/ were 720, 1240, 2500, 3600, and 4500 Hz for F_1 , F_2 , F_3 , F_4 , and F_5 , respectively. The F_2 and F_3 formant transitions were linear and 40 msec in duration. F_4 and F_5 were steady-state formants.

Stimulus duration was 90 msec. The amplitude of voicing was constant for 80 msec and fell linearly to 0 in the last 10 msec of the stimuli. The fundamental frequency began at 103 Hz, increased linearly to 125 Hz in 35 msec, and then decreased to 83 Hz in 55 msec. The peak amplitudes of both stimuli were within 0.5 dB of each other.

Files from the Klatt synthesizer were downloaded to a PC-based stimulus delivery system that outputs the signals through a 12-bit D/A converter. That system controlled time of delivery, the stimulus sequence, and the stimulus intensity. It also triggered a PC-based evoked potential averaging system and indicated whether the trial contained a standard or deviant stimulus.

Electrophysiologic Measures

To control for level of arousal, subjects were instructed to watch videotaped movies or cartoons, a practical procedure for school-aged children. The videotape audio levels were kept below 40 dB SPL. Speech stimuli were presented to the right ear at 75 dB SPL through insert earphones. The interstimulus interval was 1.5 sec. Evoked responses elicited by standard stimuli and those elicited by deviant stimuli were averaged separately. P_1 , N_1 , and P_2 were evident in both the ERP obtained to the standard as well as to the deviant stimuli; the MMN occurred only in response to the deviant stimuli in the oddball paradigm. An oddball paradigm was used in which a deviant stimulus /da/ (probability of occurrence = 15%) was presented in a series of standard stimuli /ga/ (probability of occurrence = 85%). The averaged responses were obtained in blocks of 25 deviant stimuli and approximately 140 standard stimuli. A total of eight such blocks were run for each subject. Stimuli were presented in a pseudorandom sequence with at least three standard stimuli separating presentations of deviant stimuli. Twenty standard stimuli preceded the occurrence of the first deviant stimulus. Responses to standard stimuli immediately following deviant stimuli were excluded from the standard stimulus average.

As a control, responses also were measured to eight blocks of 25 stimulus presentations of the /da/ stimulus in a "/da/-alone" condition. The MMN should occur only when the auditory system makes a discrimination between the standard and deviant stimuli. Therefore, the

ERP obtained to /da/ presented in the oddball paradigm should be different from the response to the same /da/ stimulus when it is presented alone (Sams et al, 1985b; Näätänen et al, 1989b; Kraus et al, 1993a, b).

The ERPs were recorded from Fz/A2 with the forehead as ground. Trials containing eye movements were excluded from the averages. Eye movements were monitored with a supraorbital electrode referenced to the contralateral mastoid or a bipolar electrode montage (supraorbital-to-lateral canthus). Prior to data collection, subjects were instructed to blink and move their eyes while amplifier settings were adjusted to ensure detection of eye movements (artifact level ± 163 – $245 \mu\text{V}$). Averaging was suspended automatically when the eye channel registered movement. The recording window included a 50-msec prestimulus period and 500 msec of poststimulus time. Evoked responses were analog bandpass filtered on-line from 0.1 to 100 Hz (12 dB/octave). Responses were digitally lowpass filtered with a Blackman filter at 40 Hz off-line.

Data Analysis

Individual Subject Data Analysis

For each subject, responses from a total of eight stimulus blocks of /ga/ and /da/ in the oddball paradigm and eight blocks of the /da/-alone condition were used in the analysis. An individual grand average of those eight blocks was computed. Thus the individual grand averages generally consisted of a total of 1,200 responses to the standard (/ga/) stimulus and 200 responses each to the deviant (/da/) and /da/-alone stimuli.

For each subject, ERPs were analyzed for the presence of components (P_1 , N_1 , and P_2), amplitude, and latency. P_1 , N_1 , and P_2 were considered present if amplitudes exceeded or were equal to $0.5 \mu\text{V}$. Amplitude and latency measurements of waves P_1 , N_1 , and P_2 were obtained from the response to the standard stimulus /ga/. Peak latencies were measured from the midpoint of each wave in the latency ranges of 25–100, 75–200, and 100–250 msec for P_1 , N_1 , and P_2 , respectively. Peak amplitudes of P_1 and N_1 were computed with respect to the prestimulus baseline. P_2 amplitude was measured from the preceding trough, because it is influenced by several "negativities" associated with latencies at N_1 and just following N_1 (Näätänen and Picton, 1987). Some of these negativities extend well into the latency of P_2 .

Wave P_2 inherently "rides" on those negativities and the apex of P_2 often lies below baseline. The most consistent measure of P_2 amplitude appears to be from the preceding trough to the P_2 peak. Waves P_1 , N_1 , and P_2 occurred at similar latencies in response to standard and deviant stimuli.

Since the MMN is, by definition, elicited only by the deviant stimulus, a difference wave was computed by subtracting the individual grand average response to the standard stimulus from the response to the deviant stimulus. Likewise, a difference wave was computed by subtracting the response to the /da/-alone stimulus from the response to the deviant (/da/) stimulus.

The morphologies of the standard, deviant, /da/-alone, and difference waveforms (deviant minus standard, deviant minus /da/-alone) were assessed relative to the previously described morphology of speech-evoked MMNs (Aaltonen et al, 1987; Sams et al, 1990; Kraus et al, 1993a). The MMN was identified visually as a relative negativity following the N_1 , within a latency range of 150–300 msec. Statistical tests were performed on the individual responses to ensure that the MMN identified visually was indeed a significant negative deflection as described previously (Kraus et al, 1993a, b). Using the subject's grand average waveforms, a latency (in msec) was determined for the onset, offset, and peak (point of maximum negativity) of the MMN. Using the contributing difference waves, t-tests were performed comparing the amplitudes of 5-msec periods flanking the three marked latencies (onset, peak, offset). Likewise, t-tests were performed on the individual deviant (oddball paradigm) and deviant minus /da/-alone difference waveforms comparing the amplitudes at the same three latency points identified above. An MMN was considered to be present for that individual if the amplitude of the peak was significantly different from the onset or offset amplitudes of the MMN in both the deviant minus standard and deviant minus /da/-alone conditions.

The magnitude and latency of the MMN was measured from each individual subject's difference wave. MMN duration was defined as the offset minus the onset latency for each subject. MMN magnitude was calculated (1) by measuring the peak-to-peak amplitude (minimum 0.5 μ V) from the preceding peak to the midpoint of the MMN (onset-to-peak) and from the midpoint to the end of the MMN waveform (peak-to-offset) and (2) by measuring the area of

the MMN waveform. To measure the area of the MMN, a line was drawn between the onset and offset of the MMN in the difference wave. The enclosed area of the difference waveform was measured in msec \times μ V.

The amplitude and latency of MM4 were also calculated from each individual subject's difference wave. MM4 latency was measured from the midpoint of the wave. MM4 amplitude was measured from that trough to the preceding peak.

Group Data Analysis

Grand averages were computed across subjects. T-tests were used to compare differences between children and adults for P_1 , N_1 , P_2 , and MMN. In addition, a grand average of the difference waveform (deviant minus standard) was calculated. A point-by-point t-test of the values of the contributing waveforms determined the latency duration over which the grand averages were significantly different from zero (i.e., a significant difference between the standard and deviant waveforms). A significant negativity (seen in the grand average difference wave) following the N_1 (seen in the grand average standard and deviant waveforms) was defined as the group MMN. A similar analysis was performed on the deviant (oddball paradigm) minus /da/-alone difference waveforms. By definition, the MMN occurs in response to the deviant stimulus only when it is presented in the oddball paradigm and not when the deviant stimulus is presented alone.

RESULTS

Grand average responses across subjects to the standard and deviant stimuli and the corresponding difference waves are shown in Figure 1 for children and adults. Waves P_1 , N_1 , and P_2 are evident in response to both standard and deviant stimuli. The MMN and MM4 are evident in the responses to the deviant stimuli. Latency and magnitude values of all waves and statistical comparisons between adults and children are listed in Table 1.

P_1 , N_1 , and P_2

Figure 2 shows representative responses to the standard stimulus /ga/. Responses from several children are compared to the adult response. While the MMN was adult-like in mor-

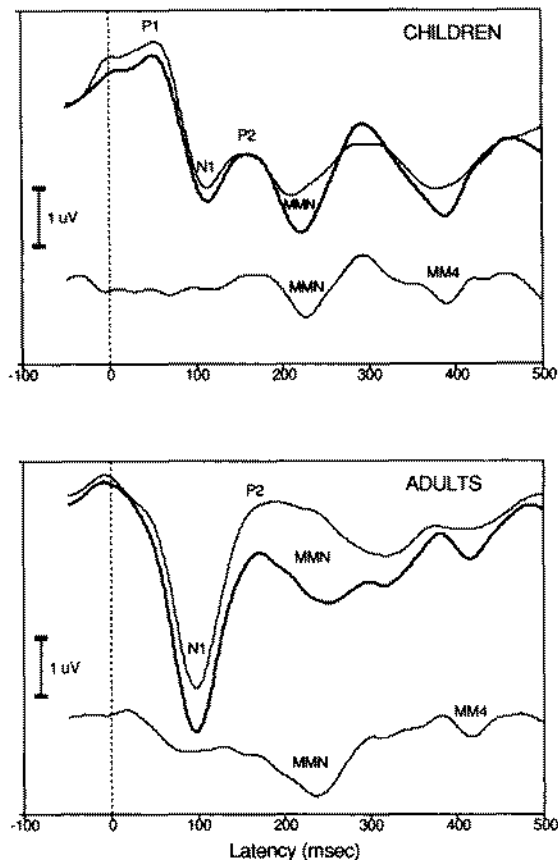


Figure 1 Grand average ERPs across subjects are shown for children (top) and adults (bottom). For each group, the light trace is the response to the standard stimulus /ga/ and the darker trace is the response to the deviant stimulus /da/. The bottom trace is the difference wave obtained by subtracting the ERP to the standard stimulus from the response to the deviant stimulus. Positive is up.

Table 1 Comparison of Latency and Amplitude Values for Children and Adults

	Children		Adults		Significant Difference
	Mean	SD	Mean	SD	
Latency					
P ₁	61	11	50	6	p < .05
N ₁	119	25	98	12	p < .05
P ₂	171	31	177	23	
MMN peak	220	41	231	31	
MMN onset	163	34	170	19	
MMN offset	277	33	280	30	
MM4	388	39	418	28	
Amplitude					
P ₁	1.5	1.1	1.1	1.1	
N ₁	-2.5	1.5	-2.6	1.1	
P ₂	2.5	1.0	4.3	1.7	p < .01
MMN onset/peak	3.3	2.2	1.2	0.6	
MMN offset/peak	3.3	1.8	1.3	0.5	p < .01
MMN area	280	169	153	67	p < .05
MM4	4.3	2.2	1.4	1.2	p < .0005

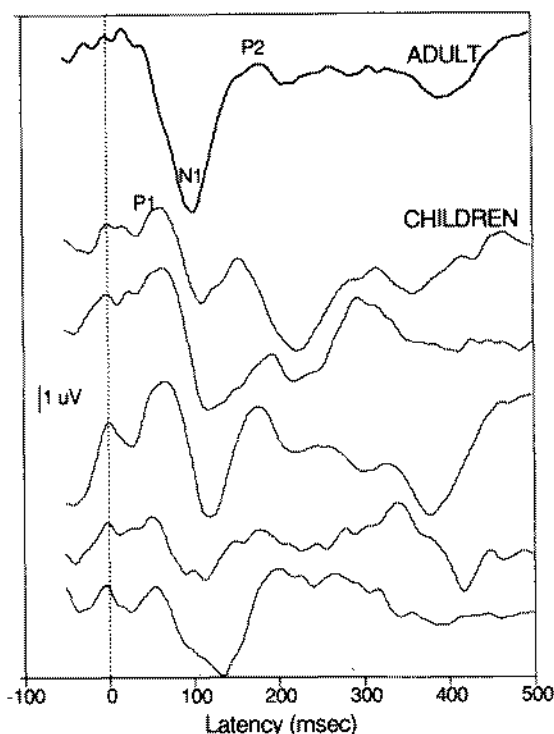


Figure 2 Representative responses to the standard stimulus /ga/ from several children are compared to the adult response. Note the large P₁ and the later and more variable latency of N₁ in the children in comparison to the adults.

phology, the earlier components differed in the children. Adults showed the classic, well-defined N₁-P₂ complex. In children, a large positive wave (P₁) tended to dominate the P₁/N₁ complex. N₁ peak latency was less well defined (consistent with greater latency variability), and P₂ was smaller in children, as compared to adults. Both P₁ and N₁ latency were significantly longer in children.

Overall, wave P₁ was present in 14 children and 6 adults tested. All subjects in this study exhibited waves N₁ and P₂. The latency of both P₁ and N₁ were significantly longer and more variable in children compared to adults as seen in the individual subject data in Figure 3. The P₂ latency was similar in adults and children. P₁ and N₁ amplitudes were similar in adults and children. The P₂ component was larger in adults.

Mismatch Negativity

There were no differences in the latency of the MMN in children, compared to adults. No significant differences were found in peak latency, onset, offset, or total duration. Peak la-

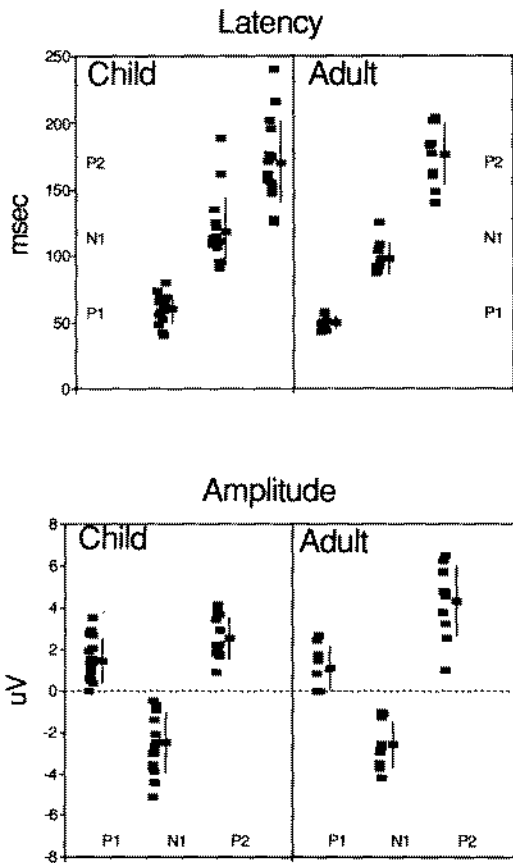


Figure 3 P₁, N₁, and P₂ latency and amplitudes. Top: Latency of waves P₁, N₁, and P₂ are shown for individual subjects and are compared in children and adults. Solid lines indicate ± 1 SD. Waves P₁ and N₁ occurred significantly later in children than in adults. Bottom: Amplitude of waves P₁, N₁, and P₂ are shown for individual adults and children. Solid lines indicate ± 1 SD. P₂ amplitude was greater in adults.

tency and duration measurements are shown in Figure 4 for each subject. Mean latency (± 1 SD) is depicted by vertical bars in the figure (top).

Individual amplitude values (onset-to-peak and peak-to-offset) for each individual child and adult are shown in Figure 5 (top). Mean amplitudes (± 1 SD) are also depicted by the vertical bars in the figure. Mean MMN area measures are shown for the children and adults at the bottom of the figure. Onset-to-peak amplitude was not significantly different in adults and children, although there was a trend for larger amplitudes in the children. Peak-to-offset amplitudes were significantly larger in the children, as compared to the adults. MMN area measures were also larger for the children than the adults. Using the criteria specified above, the MMN was present in each individual, in the deviant minus standard condition as well as in

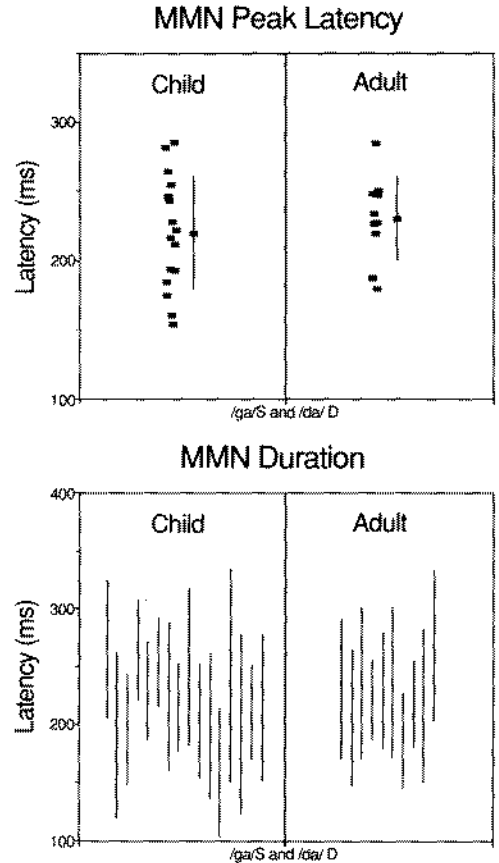


Figure 4 MMN peak latency (± 1 SD) and duration for individual adults and children. Solid lines (top) indicate ± 1 SD. There were no significant differences between adults and children. S and D indicate standard and deviant, respectively.

the corresponding control deviant /da/ minus /da/-alone condition (Table 2).

Figure 6 shows the grand average MMN difference wave for all 16 children and 10 adults. The cross-hatched area below the waveforms denotes the portion of the difference wave that is significantly different from zero. The analysis shows a region of significance in the latency range of the MMN, which occurs at 200 msec.

Another negative wave occurring at about 400 msec (MM4) was observed in the response to the deviant stimuli and was clearly present in the difference waves. That negative wave occurred at a similar latency in adults and children and was significantly larger and more robust in children.

DISCUSSION

From this cohort of school-aged children, it appears that various passively elicited event-related potentials develop at different

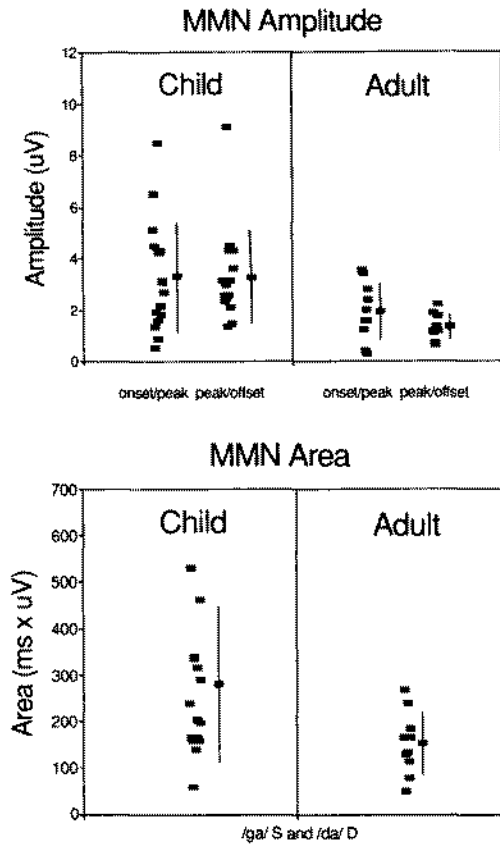


Figure 5 MMN magnitude (amplitude and area) for individual children and adults. Solid lines indicate ± 1 SD. Peak-to-offset and area measures were significantly larger in the children than in the adults. S and D indicate standard and deviant, respectively.

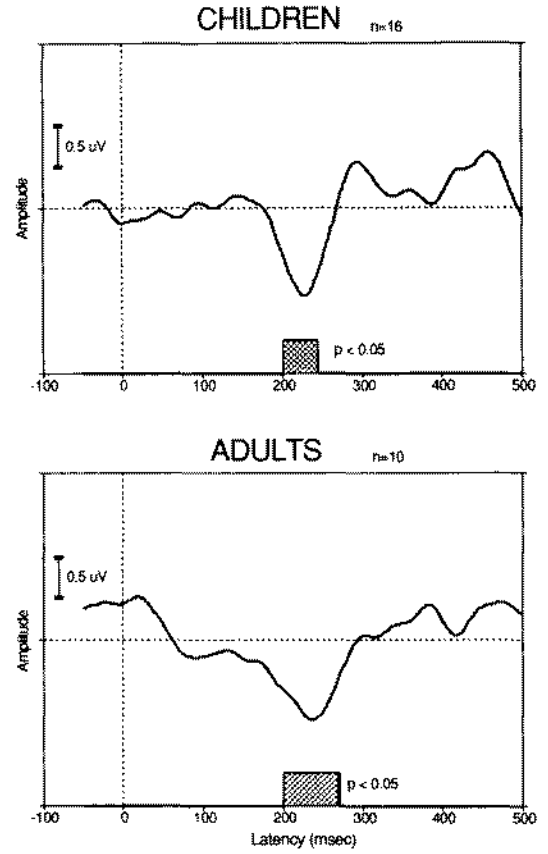


Figure 6 Grand average difference waves obtained by subtracting the response to the standard stimulus /ga/ from the response to the deviant stimulus /da/ in children (top) and in adults (bottom). The significant MMN region in children ($t > |1.75|$; $df = 15$; $p < 0.05$) and adults ($t > |1.83|$; $df = 9$; $p < 0.05$) are indicated by the cross-hatched areas.

Table 2 Analysis of MMN Significance in Individual Children

Subject	Deviant-Standard				Deviant Rare-Deviant Alone			
	Onset/Peak		Peak/Offset		Onset/Peak		Peak/Offset	
	Amp	t	Amp	t	Amp	t	Amp	t
AA	4.29	10.10 †	2.08	6.13 †	3.69	10.21 †	2.66	1.38
AK	2.65	3.59 †	3.60	3.80 †	3.18	9.61 †	4.19	4.88 †
CM	1.32	3.21 †	2.41	3.41 †	5.58	3.69 †	3.16	4.46 †
CS	0.55	0.72	2.57	2.70 †	2.91	3.96 †	3.93	2.81 †
DN	8.46	9.09 †	9.12	7.69 †	4.64	3.71 †	6.42	3.36 †
JA	3.06	2.91 †	1.32	1.34	2.96	1.87 *	1.53	0.83
JB	2.15	3.23 †	4.45	6.48 †	2.03	2.71 †	2.60	3.16 †
KS	5.10	8.53 †	2.50	3.54 †	4.36	7.10 †	7.82	5.72 †
LM	0.88	2.25 *	1.33	2.56 †	4.70	0.73	1.54	4.93 †
MB	1.79	4.14 †	3.16	3.28 †	2.00	1.70 *	3.52	2.88 †
MG	4.23	1.63	4.33	4.71 †	11.41	4.2 †	6.83	8.03 †
NP	1.95	3.06 †	2.95	4.99 †	3.00	4.09 †	3.84	2.01 *
RM	6.51	9.83 †	3.15	3.82 †	3.04	7.94 †	8.75	2.50 *
SG	4.46	7.74 †	2.30	3.43 †	4.31	7.74 †	3.86	4.59 †
SM	1.59	1.72 *	2.56	4.43 †	4.91	2.25 *	2.57	5.80 †
ZG	3.11	3.94 †	4.30	5.48 †	7.37	7.03 †	5.87	4.69 †

* $p < .05$; † $p < .01$.

rates. Development of speech-evoked P_1 , N_1 , and P_2 waves appears to continue beyond the ages of the children in the present study. The child MMN was similar to the adult response in both latency and morphology, indicating that this response is largely mature in school-aged children. The results support the view that developmental changes in late potentials do not involve changes in a single parameter, like a gradual latency or amplitude shift, as a function of age (Eggermont, 1989; Courchesne, 1990).

P_1

In children, a wave at the latency of P_1 often dominated the early portion of the ERP. The generating system underlying this wave in adults has been linked to the reticular activating system at the level of the thalamus (Buchwald et al, 1981; Erwin and Buchwald, 1986; Buchwald, 1989). The continuing development of P_1 into the second decade of life is consistent with the view that the reticular activating system or the areas it influences, (although clearly functional early in life) continue to myelinate during this time (Yakovlev and Lecours, 1967). The decrease in P_1 latency with age may reflect this continuing myelination process. The reticular formation is also thought to affect the preceding positive wave (P_a), which also continues to develop until preadolescence (Kraus et al, 1985, 1989, 1992b).

N_1 - P_2 Complex

N_1 was detected in all children 7 years of age and older in this sample, but N_1 latency occurred significantly later in children compared to adults. Although N_1 was identifiable, its peak was not clearly delineated and the significantly reduced amplitude of P_2 in children impeded the identification of the " N_1/P_2 complex" that is so characteristic of the adult response. Both Goodin et al (1978) and Martin et al (1988) report that N_1 and P_2 are unreliably obtained in 6-year-olds, but become easier to identify in older children. According to Courchesne (1990), it is not until preadolescence and puberty that the familiar auditory N_1/P_2 vertex potential begins to be clearly seen. Similarly, Csépe and colleagues (1992) reported P_1 , N_1 , and P_2 morphologies and latencies consistent with those reported here for school-aged children. In contrast, Johnson (1989) reported adult-like latencies and amplitudes for N_1 and P_2 in school-aged children.

Mismatch Negativity

The speech-evoked MMN was elicited to speech stimuli in all children tested. The MMN to speech stimuli appears to be a robust phenomenon occurring at adult latencies even in the youngest subjects tested. Consistent with other reports (Csépe, 1992), MMN magnitude was larger in children. This may be an indication that the MMN reflects processes that are particularly salient in children. Alternatively, the geometric orientation of the generating system may optimize MMN amplitude at Fz in children.

The P300 ERP (Sutton et al, 1965) consists of an inconsistently present, passively elicited component, P3a (Roth, 1973; Squires et al, 1975; Polich, 1989) as well as a task-related, P3b response (Donchin, 1979; Woods et al, 1980). The MMN has been shown to be distinct from P3a, particularly when standard and deviant stimulus differences are small (Sams et al, 1985a; Duncan and Kaye, 1987) and when the subject's attention is clearly diverted from the stimulus (Näätänen et al, 1989b). However in this study, acoustic differences between /da/ and /ga/ may have been distinct enough to allow the peak-to-offset portion of the MMN and P3a to overlap. If this is the case, our results are consistent with previous investigations reporting the existence of an early developing positive wave in this latency range. For example, a P300-like response to speech stimuli presented in the oddball paradigm is present at birth (Kurtzberg et al, 1986). Other studies have shown that from early childhood to young adulthood, there is a P3a-like positivity of about 300–360 msec that is elicited by auditory targets, auditory high-probability nontargets, and auditory novel, unexpected sounds (Courchesne, 1990). That P3a-like component does not change in latency from childhood to adulthood. Also, there is a component of similar latency, in an animal model, dependent upon the locus coeruleus (Pineda et al, 1988), that may be an analog of the P3a. In contrast, strong developmental differences between adults and children are reported for the task-related P3b component of the P300 response. Latency of P3b decreases systematically throughout childhood, reaching asymptote beyond puberty (Goodin et al, 1978; Finley et al, 1985; Polich et al, 1985; Martin et al, 1988; Johnson, 1989; Courchesne, 1990). The negativity at 400 msec seen in the response to deviant

stimuli is likely related to the Nc response described by Courchesne (1977, 1978), which is known to be larger in children and inconsistently obtained in adults.

Overall Comparisons

Waves P₁, N₁, and P₂ differed morphologically in children and adults. P₁ and N₁ latencies were longer and P₂ amplitude was smaller in the children. The MMN appears to reflect auditory processes that develop early in childhood. The latency and morphology of the MMN was similar in children and adults, and MMN and MM4 magnitude was greater in children. It should be noted that the number of subjects in this study was not large enough to fully characterize the developmental continuum of these ERPs from the ages of 7 to 11 years. Instead, this is a comparison of data from a group of children versus a group of adults. Characteristics of the developmental sequence would require a larger subject group.

The neurophysiologic processes underlying certain automatic responses may mature earlier than the processes underlying task-related responses (Shiffrin and Schneider, 1977; Näätänen and Picton, 1987). The MMN was elicited while the subjects attended to stimuli other than the test stimuli. There were no differences in the latency or morphology of the MMN in adults and children under those conditions. This contrasts with the P3b response, (which requires attention to the stimuli) and has longer latencies in children than adults (Goodin et al, 1978; Martin et al, 1988; Johnson, 1989). Nevertheless, the larger response in children may also reflect attentional effects to the deviant stimuli (Woldorff and Hillyard, 1991; Alho et al, 1992; Woods et al, 1992) which may be more pronounced in children.

The MMN is thought to originate in central auditory and nonauditory structures (Hari et al, 1984; Csépe et al, 1987; Kaukoranta et al, 1989; Giard et al, 1990). As an early developing response, it may become a useful tool for evaluating patients with auditory disorders affecting central pathways (Korpilahti et al, 1992; Kraus et al, 1993c). The response can be obtained in school-aged children in conjunction with viewing cartoons or a movie, which is a practical and effective way to maintain the cooperation of young children during a 1 to 2 hour test session. Additional information about the specific aspects of sensory processing reflected by these different ERPs are likely to be obtained through

a combination of electrophysiologic and behavioral studies.

In summary, P₁ tends to dominate the P₁/N₁ complex, N₁ peak latency is less well defined, and P₂ is smaller in children as compared to adults. Both P₁ and N₁ latency are significantly longer in children. Thus, the overall morphology and latencies of these waves may provide a clinically useful measure of the maturation of central pathways. In contrast to these earlier waves, the MMN is well developed in school-aged children. The early development of the MMN and its reflection of fine auditory discrimination make it a potentially important tool for the assessment of central auditory system function in children.

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