

# Developmental changes in P1 and N1 central auditory responses elicited by consonant-vowel syllables

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## Abstract

Normal maturation and functioning of the central auditory system affects the development of speech perception and oral language capabilities. This study examined maturation of central auditory pathways as reflected by age-related changes in the P1/N1 components of the auditory evoked potential (AEP). A synthesized consonant-vowel syllable (ba) was used to elicit cortical AEPs in 86 normal children ranging in age from 6 to 15 years and ten normal adults. Distinct age-related changes were observed in the morphology of the AEP waveform. The adult response consists of a prominent negativity (N1) at about 100 ms, preceded by a smaller P1 component at about 50 ms. In contrast, the child response is characterized by a large P1 response at about 100 ms. This wave decreases significantly in latency and amplitude up to about 20 years of age. In children, P1 is followed by a broad negativity at about 200 ms which we term N1b. Many subjects (especially older children) also show an earlier negativity (N1a). Both N1a and N1b latencies decrease significantly with age. Amplitudes of N1a and N1b do not show significant age-related changes. All children have the N1b; however, the frequency of occurrence of N1a increases with age. Data indicate that the child P1 develops systematically into the adult response; however, the relationship of N1a and N1b to the adult N1 is unclear. These results indicate that maturational changes in the central auditory system are complex and extend well into the second decade of life. © 1997 Elsevier Science Ireland Ltd.

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## 1. Introduction

Development of the peripheral auditory system (ear and auditory brain-stem) is complete in early childhood (Eggermont, 1989). In contrast, central auditory pathways of the human brain exhibit progressive anatomical and physiologic changes through early adulthood (Kraus et al., 1985; Courchesne, 1990; Huttenlocher, 1979). This maturation is likely to have an impact on speech and oral language skills, which are primarily acquired through the auditory modality. Auditory evoked potentials (AEPs) reflect maturation of the human brain through changes in their

latency, amplitude and morphology (Eggermont, 1989; Courchesne, 1990). In this study, we used AEPs elicited by consonant-vowel syllables to examine normal maturation of the central auditory areas. Understanding the normal patterns of maturation of AEPs evoked by speech sounds may aid in the development of electrophysiologic techniques for diagnosing abnormal central auditory maturation coincident with speech, language and learning impairments. This study is part of a larger effort to examine the neurophysiologic substrate of auditory information processing abilities in normal and language-learning impaired children using auditory evoked responses (Kraus et al., 1996).

The 'late' or 'cortical' AEP consists of a series of positive and negative peaks (P1/N1 complex) occurring between 80 and 150 ms after stimulus onset. P1 and N1 components primarily reflect sensory encoding of auditory stimulus attributes and precede more endogenous components such as N2

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and P3 which are concerned with attention and cognition. P1 and N1 are generated by multiple temporally overlapping subcortical and cortical sources (Chen and Buchwald, 1986; Näätänen and Picton, 1987). These components are passively elicited in that the subject is not required to perform a task and is asked simply to remain alert. Since they are not influenced by behavioral and performance related demands these evoked responses provide a reliable objective measure of cortical auditory function in children.

While these late components of the AEP have been extensively studied in adults, little is known about the emergence and development of P1/N1 in childhood. The P1/N1 complex is a robust and ubiquitous component of the adult response. In contrast, its characteristics (and even presence) are ambiguous in children. Some studies have reported that the latency of N1 decreases as age increases up to 16 years (Tonnquist-Uhlen et al., 1995) and up to 20 years (Johnson, 1989), and N1 amplitude increases up to 15 years of age (Martin et al., 1988). Conversely, other studies have reported little or no age-related changes in N1 latency (Ohlrich et al., 1978; Martin et al., 1988) and amplitude (Fuchigami et al., 1993). Recent studies have also suggested that the component analogous to the adult N1 may not emerge until 8 or 10 years of age (Csepe, 1995; Ponton et al., 1996). These inconsistent findings may be in part due to the differences in age ranges, stimuli and number of subjects used in the above studies. Data on the maturation of P1 are more sparse. In a recent investigation, Ponton et al. (1996) reported that P1 decreases in latency and reaches adult values at age 19 years. It is clear that large population studies across wide age ranges are required to describe maturation of P1 and N1 responses.

AEPs evoked by consonant-vowel syllables provide an opportunity to assess auditory pathways engaged in the acoustic analysis of speech. Our goal is to define the neurophysiologic responses to these speech sounds because the representation of such sounds is undoubtedly important for speech and language development. This study examines age-related changes in P1 and N1 AEPs in normal school-age children and adults. Results of this study will provide information on normal development of central auditory pathways.

## 2. Methods

### 2.1. Subjects

Subjects were 86 healthy normal children (6–15 years) and 10 young adults (21–27 years). Children were judged normal based on their scores on standardized tests of learning and academic achievement (Woodcock-Johnson Psychoeducational Battery and Wide Range Achievement Test) and their histories. All subjects had normal hearing thresholds and no history of speech or language disorders.

The number of subjects and the mean age in each range are shown in Table 1.

### 2.2. Stimuli

A synthesized consonant-vowel syllable (ba) was used to elicit evoked responses. The 5 formant consonant-vowel stimulus was generated using the Klatt (1980) speech synthesizer. The starting frequencies of the first and second formants (i.e. F1 and F2) were 234 Hz and 616 Hz, respectively. The center frequencies for the formants for the vowel (a) were 769 Hz, 1232 Hz, 2862 Hz, 3600 Hz and 4500 Hz for F1, F2, F3, F4 and F5, respectively. F1 and F2 formant transitions were linear and 40 ms in duration. F3, F4 and F5 were steady-state formants. Stimulus duration was 90 ms. The amplitude of voicing was constant for 80 ms and fell linearly to 0 in the last 10 ms of the stimuli. The fundamental frequency began at 103 Hz, increased linearly to 125 Hz in 35 ms and then decreased to 80 Hz in 55 ms.

### 2.3. Electrophysiologic recording

Evoked responses were collected using Fz as the active electrode. The right earlobe served as the reference electrode with forehead as ground. Eye movements were monitored with a bipolar electrode montage (supraorbital to lateral canthus). Prior to data collection subjects were asked to blink and move their eyes while amplifier settings were adjusted to ensure detection of eye movements. Averaging was automatically suspended when eye movements were detected. The recording window included a 100 ms pre-stimulus period and 500 ms post-stimulus time. Evoked responses were analog filtered online from 0.1 to 100 Hz. Averaged responses were elicited in blocks of approximately 140 stimuli. Ten blocks (i.e. approximately 1400 stimuli) were collected per subject. During the testing, subjects watched videotaped movies and were instructed to ignore the test stimuli. Stimuli were presented to the right ear using insert earphones at a level of 75 dB SPL and onset-to-offset interstimulus interval of 510 ms. The left ear was unoccluded and videotape audio levels were kept below 40 dB SPL-(A).

### 2.4. Data analysis

For each subject, an individual grand average waveform was computed by averaging the ten records. The P1 was identified visually as a relative positivity in the 50–150 ms region. N1b was identified visually as a relative negativity (following P1) occurring within the 150–250 ms region. Some subjects showed an earlier negativity (N1a) occurring in the 90–160 ms range. For each subject, the peak latency, peak amplitude (relative to the baseline) of AEP components were marked. Pearson's (*r*) correlation coefficients and linear regressions were computed to determine the relationship between AEP components and age.

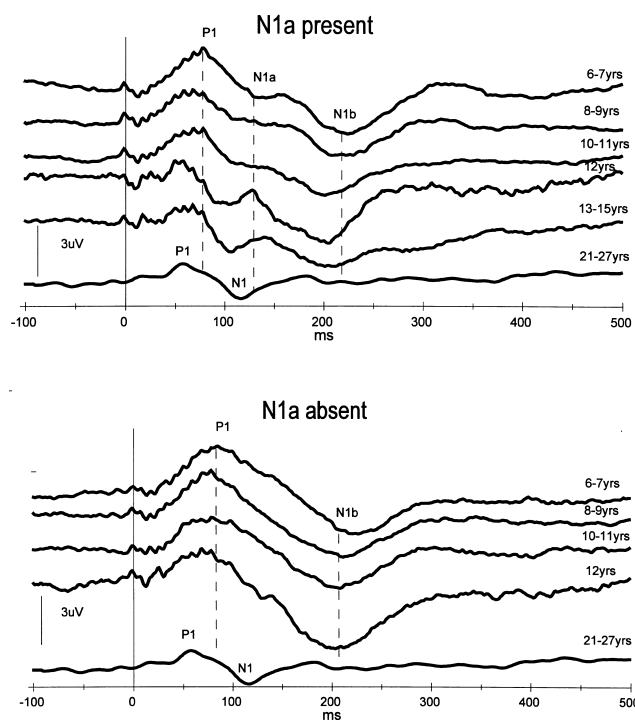


Fig. 1. Top panel: group grand average waveforms for subjects (aged 6–12 years) in whom the N1a component was present. Bottom panel: group grand average waveforms for subjects (aged 6–12 years) in whom the N1a component was absent. Note: N1a was present in all 13–15 year old subjects. Group grand average waveforms for adults are shown at the bottom in both panels.

Group grand average waveforms were computed by averaging waveforms for all subjects in a restricted age range (e.g. 6–7 years, 8–9 years, etc.)

### 3. Results

#### 3.1. Morphology

Distinct age-related changes are seen in the AEP waveform morphology. The adult response consists of a prominent negativity (N1) at about 100 ms, preceded by a small positive (P1) response at about 50 ms. In contrast, the child response is dominated by a large P1 response. In children the P1 is followed by a broad negativity at about 200 ms, which we term N1b. Many subjects also show an earlier negativity (N1a). While all children have an N1b, the frequency of occurrence of N1a appears to increase with age (61% of 6–7 year olds, 63% of 8–9 year olds, 69% of 10–12 year olds and 100% of 13–15 year olds have N1a). Grand average waves for subjects with and without N1a are shown in Fig. 1.

#### 3.2. Latency and amplitude

Latency and amplitude values for AEP components are

shown in Tables 2 and 3. Latency of all three components (i.e. P1, N1a and N1b) show significant negative correlations with age (Fig. 2). Amplitude of P1 shows a significant age-related decrease. N1a and N1b amplitudes do not show a significant correlation with age. Linear regression lines were computed to fit the latency data for P1, N1a and N1b and the P1 amplitude data. Values for correlation coefficients, slopes and intercepts of the regression lines are shown in Table 4. For latency data (ages 6–15 years) slopes of each pair of regression lines (i.e. P1 vs. N1a, N1a vs. N1b, and P1 vs. N1b) were compared using a *t* test (Glantz, 1992). Results revealed that the slopes of the regression lines for P1, N1a and N1b latency were not significantly different ( $P > 0.05$ ) from each other.

### 4. Discussion

Our results show that the AEP waveform changes in a complex manner with age. In our youngest age group (6–7 years), the waveform is dominated by a large positivity (P1) followed by a broad negativity (N1b). A similar morphology has been reported for children as young as 2–4 years of age (Courchesne, 1990). The child P1 decreases systematically in latency and amplitude to reach adult values at around age 20 years. These findings are consistent with recent data from Ponton et al. (1996) showing that adult P1 latencies are reached around age 19 years.

An important finding of this study is that an earlier negativity (N1a) occurs as early as 6 years of age in some children. The frequency of occurrence of the N1a component increases with age, and it is consistently elicited around age 13 years. These data are consistent with Ponton et al. (1996) who report emergence of a component (similar to our N1a) around 8–10 years which they consider to be a precursor to the adult N1 response.

Latencies of both N1a and N1b decrease systematically up to age 15 years. Since we do not have data for a 15–19 year age group, the relationship of N1a and N1b to the adult N1 is unclear at the present time. For example, N1a latency appears to have reached adult values by age 13–15 years. On the other hand, N1b latency would have to continue to

Table 1

Number of subjects and the mean age in each age range

Number of subjects	Age range (years)	Mean age (years)
12	6–6.9	6.5
11	7–7.9	7.4
19	8–8.9	8.5
19	8–8.9	8.5
8	9–9.9	9.6
11	10–10.9	10.5
13	11–11.9	11.5
5	12–12.9	12.4
7	13–14.9	14.11
10	20–27 (adults)	22.6



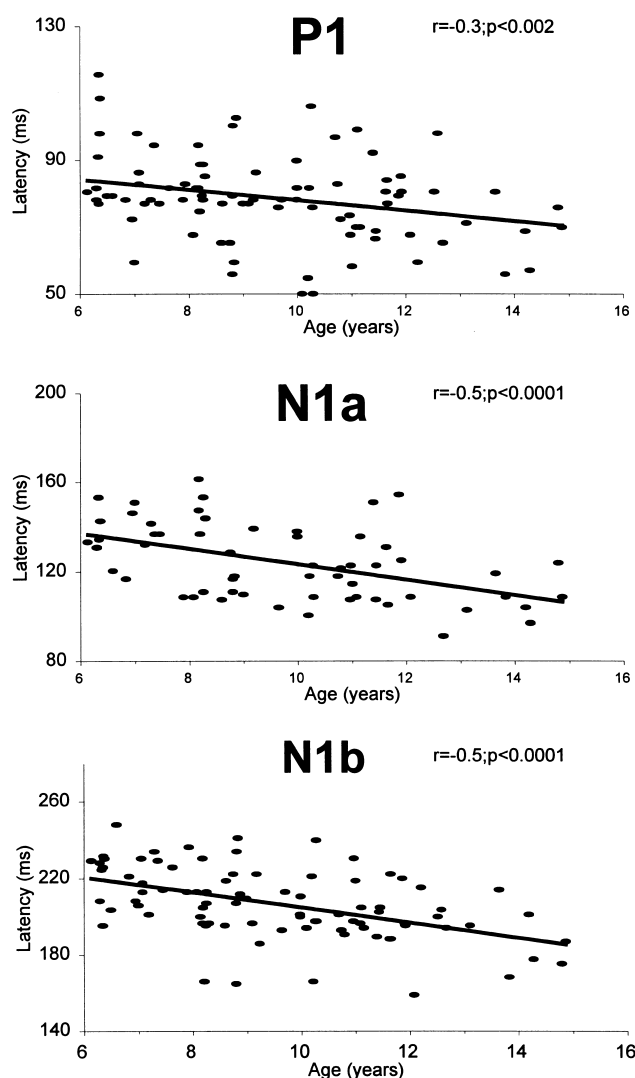


Fig. 2. Latency versus age functions for P1, N1a and N1b components for children aged 6–15 years.

Table 4

Correlation and linear regression values for P1, N1a and N1b responses

	<i>n</i>	Pearson's ( <i>r</i> )	<i>P</i> value	Slope	Intercept
<b>Peak latency</b>					
P1 (6–17 years)	86	– 0.32	0.002	– 1.85 ms/year	96
P1 (6–27 years)	96	– 0.50	0.0001	– 1.55 ms/year	94
N1a (6–15 years)	58	– 0.5	0.0001	– 3.45 ms/year	158
N1b (6–15 years)	86	– 0.5	0.0001	– 3.96 ms/year	244
<b>Baseline to peak amplitude</b>					
P1 (6–15 years)	86	– 0.31	0.003	– 0.16 $\mu$ V/year	3.5
P1 (6–25 years)	96	– 0.35	0.0001	– 0.09 $\mu$ V/year	2.9
N1a (6–15 years)	58	– 0.19	NS		
N1b (6–15 years)	86	– 0.03	NS		
<b>Peak to peak amplitude</b>					
P1-N1a (6–15 years)	58	– 0.08	NS		
P1-N1b (6–15 years)	86	– 0.18	NS		

appear that the general trend of P1/N1 maturation is comparable for speech and non-speech stimuli.

The linear maturation functions for P1, N1a and N1b in the 6–15 age groups showed modest slopes (i.e. latency decrease on the order of 1.9, 3.5 and 4 ms/year for P1, N1a and N1b, respectively). The gradual decrease in latencies probably occurs as a result of gradual increases in neural transmission speed due to age-related changes in myelination of underlying neural generators as well as increases in synaptic synchronization and efficacy (Huttenlocher, 1979; Courchesne, 1990). While there is not much information available regarding generators for the child P1/N1 responses, it is generally agreed that the generators of the N1 include the primary auditory cortex in adults (Näätänen and Picton, 1987) and that the P1 component is generated from thalamocortical sites, including the reticular activating system (Chen and Buchwald, 1986; Erwin and Buchwald, 1986a,b). Overall, the extended time course of maturation of the P1/N1 components in this study is consistent with the myelination and synaptic changes which continue to occur through the second decade of life (Yakovlev and Lecours, 1967; Courchesne, 1990).

While P1 and N1b components were observed in all children, the N1a response was inconsistently present. For example, some children showed an N1a at age 6 years, while others did not demonstrate a clearly identifiable N1a component until age 13 years. This may be a reflection of the fact that underlying myelinogenesis and synaptogenesis proceed at varying rates in different individuals (Brady et al., 1975). That is, although the overall the pattern of maturation is similar, it proceeds faster in some individuals than in others.

Our results indicate that maturational changes occurring in the cortical AEP components are complex and extend well into the late teenage years. Future studies are needed to understand the changes that occur in the N1a and N1b AEPs before culminating in the adult response. Addition-

ally, we need to determine whether P1 and N1 are related to behavioral aspects of auditory system development and function.

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### References

- Brady, H., Harman, D. and Ordy, J.M. Aging, Vol. 1, Clinical, Morphologic and Neuro-Chemical Aspects in the Aging Central Nervous System. Raven Press, New York, 1975.
- Chen, B.M. and Buchwald, J.S. Midlatency auditory evoked responses: Differential effects of sleep in the cat. *Electroenceph. clin. Neurophysiol.*, 1986, 65: 373–382.
- Courchesne, E. Chronology of postnatal human brain development: event-related potential, positron emission tomography, myelinogenesis, and synaptogenesis studies. In: J.W. Rohrbaugh, R. Parasuraman and R. Johnson (Eds.), *Event-related Brain Potentials: Basic Issues and Applications*. Oxford University Press, New York, 1990, pp. 210–241.
- Csepe, V. On the origin and development of the mismatch negativity. *Ear Hear.*, 1995, 16(1): 91–104.
- Eggermont, J.J. The onset and development of auditory function: contributions of evoked potential studies. *J. Speech Lang. Pathol. Audiol.*, 1989, 13(1): 5–16.
- Erwin, R. and Buchwald, J.S. Midlatency auditory evoked responses: differential effects of sleep in the human. *Electroenceph. clin. Neurophysiol.*, 1986a, 65: 383–392.
- Erwin, R. and Buchwald, J.S. Midlatency auditory evoked responses: differential recovery cycle characteristics. *Electroenceph. clin. Neurophysiol.*, 1986b, 64: 417–423.
- Fuchigami, T., Okubo, O., Fujita, Y., Okuni, M., Noguchii, Y. and Yamada, T. Auditory event-related potentials and reaction time in children: evaluation of cognitive development. *Dev. Med. Child Neurol.*, 1993, 35: 230–237.
- Glantz, S.A. How to compare regression lines. In: S.A. Glantz (Ed.), *Biostatistics*, 3rd edn. McGraw-Hill, New York, 1992, pp. 239–240.
- Goodin, D., Squires, K., Henderson, B. and Starr, A. Age-related variations in evoked potentials to auditory stimuli in normal human subjects. *Electroenceph. clin. Neurophysiol.*, 1978, 44: 447–458.
- Huttenlocher, P.R. Synaptic density in human frontal cortex—developmental changes and effects of aging. *Brain Res.*, 1979, 163: 195–205.
- Johnson, R. Developmental evidence for modality-dependent P300 generators: a normative study. *Psychophysiology*, 1989, 26: 651–667.
- Klatt, D. Software for a cascade/parallel formant synthesizer. *J. Acoust. Soc. Am.*, 1980, 67: 971–995.
- Kraus, N., Smith, D.I., Reed, N.L., Stein, L.K. and Cartee, C. Auditory middle latency responses in children: effects of age and diagnostic category. *Electroenceph. clin. Neurophysiol.*, 1985, 62: 343–351.
- Kraus, N., McGee, T.J., Carrell, T.D., Zecker, S.G., Nicol, T.G. and Koch, D.B. Auditory neurophysiologic responses and discrimination deficits in children with learning problems. *Science*, 1996, 273: 971–973.
- Martin, L., Barajas, J.J., Fernandez, R. and Torres, E. Auditory event-related potentials in well-characterized groups of children. *Electroenceph. clin. Neurophysiol.*, 1988, 71: 375–381.
- Näätänen, R. and Picton, T. The N1 wave of the human electric and magnetic response to sound: a review and an analysis of the component structure. *Psychophysiology*, 1987, 24: 375–425.
- Ohlrich, E.S., Barnett, A.B., Weiss, I.P. and Shanks, B.L. Auditory evoked potential development in early childhood: a longitudinal study. *Electroenceph. clin. Neurophysiol.*, 1978, 44: 411–423.
- Ponton, C., Don, M. and Masuda, A. Maturation of human cortical auditory function: differences between normal hearing and cochlear implant children. *Ear Hearing*, 1996, 17(5): 430.
- Shibasaki, H. and Miyazaki, M. Event-related potential studies in infants and children. *J. Clin. Neurophysiol.*, 1992, 9(3): 408–418.
- Tonnquist-Uhlen, I., Borg, E. and Spens, K.E. Topography of auditory evoked long-latency potentials in normal children, with particular reference to the N1 component. *Electroenceph. clin. Neurophysiol.*, 1995, 95: 34–41.
- Yakovlev, P.I. and Lecours, A.R. The myelogenetic cycles of regional maturation of the brain. In: A. Minkowski (Ed.) *Regional Development of the Brain in Early Life*. Blackwell Scientific, Oxford, 1967, pp. 3–70.