Human Auditory System Maturation: A Neurophysiological Comparison Between Normal-Hearing Children And Children Who Use A Cochlear Implant

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Abstract: Scalp-recorded thalamo-cortical auditory evoked potentials (AEPs) appear to reflect activity originating from at least two central auditory system pathways and a third modality non-specific pathway originating in the reticular activating system. Age-related changes in the AEPs were used to estimate maturational time courses of these pathways for normal-hearing children as well as for profoundly deaf children who use a cochlear implant. Results suggest that under conditions of normal auditory input, each pathway contributing to the evoked response may have a unique developmental sequence. Under conditions of altered input due to (1) a period of profound deafness followed by (2) cochlear implant use, the developmental sequences for these pathways may be differentially affected depending on onset and duration of deafness.

INTRODUCTION

Numerous studies have investigated the maturational changes of AEPs. Most of these studies limited their scope by restricting analyses to specific AEP peaks, recording from small numbers of electrodes, and testing narrow age ranges of children. Nevertheless, the combined results of these investigations have greatly increased our understanding of AEP maturation.

Age-related changes in AEPs are presumed to reflect maturational changes in the activity of central nervous system sensory pathways. Auditory stimulation generates activity in at least three central nervous system pathways. These include two auditory system pathways (the lemniscal and lemniscal-adjunct pathways) as well as a third modality non-specific pathway originating in the reticular activating system (RAS) (1,2). Our knowledge of the neural generators that contribute to AEPs remains incomplete, but the findings from a number of studies (e.g., 3,4) suggest a relationship between activity in each of these pathways and the generation of specific AEP peaks. Based on this relationship, it is possible to propose a model of auditory system maturation based on AEP peak maturation.

The first goal of this study is to assess age-related AEP changes across an extended age range in a large group of normal-hearing children. The second goal is to relate these changes to maturation of the pathways presumed to generate activity contained in the AEPs. The third goal is to assess age-related changes in the AEPs of implanted children and to relate any differences between normal-hearing and implanted children to the possible consequences of deafness and cochlear implant use on the maturing auditory system.

METHODS

Subjects: The AEPs reported in this study were recorded from 156 normal-hearing subjects, 5-20 years of age. AEP data are also reported for 6 adults and 12 children (ages 6 to 15) who use a cochlear implant. For the implanted children, the age at which deafness was detected ranged from birth to 5 years 1 month. Three of the implanted children were congenitally deaf. The interval between detection of the profound hearing loss and implantation of the cochlear prosthesis ranged from 5 months to 8 years 10 months.

Procedure: For normal hearing subjects, AEPs were generated using acoustic clicks (100 μs in duration) presented monaurally to the left ear at approximately 70 dB nHL. For implant users, specialized computer-controlled hardware communicated directly with the implant, bypassing the speech processor, to generate 200 μs/phase biphasic current pulses. For each implant user, stimulation levels were set individually at a loud but comfortable level.

Each stimulus consisted of a brief train of 10 clicks or pulses. The interval between the onset of successive clicks or pulses within the train was 2 ms, while the interval between trains was 760 ms. In order to assess maturational changes in the mismatch negativity (MMN), AEPs were recorded using an oddball presentation sequence. A standard/deviant duration difference was created by contrasting the 10 click stimulus with a one
Averaged AEPs were based on at least 2150 stimulus presentations containing approximately 200 deviants and 1950 standards. The AEPs were recorded at 30 standard electrode locations. For analysis, electrode pair combinations were identified that optimally maximized and isolated the different AEP peaks across the 5-20 year age range tested. These electrode pair combinations were identified by a systematic iterative search of the 30 electrode array. To allow inter-hemispheric comparisons between homologous electrode pairs, the search was constrained so that optimal electrode pairs had to be located over one hemisphere, either contralateral (contra) or ipsilateral (ipsi) to the stimulated ear.

RESULTS AND CONCLUSIONS

Normal-hearing Children: The AEP peaks presumed to reflect activation of the RAS pathway include an early maturing component of Pa as well as Pb and P2. All of these peaks appear adult-like by age 6. The MMN is presumed to reflect activity originating in the lemniscal-adjunct pathway. Two MMN components were identified based on latency (early and late) and optimal recording montages. The early MMN component was adult-like contralateral to the stimulated ear by age 6. This component emerged ipsilateral to the stimulated ear at age 6. The late MMN component was also present contralateral to the stimulated ear in the youngest group of children tested and was present ipsilateral to the stimulated ear from age 7 onward. Unlike the early component, the latency of the late MMN clearly decreases as a function of age, most notably after age 10. The AEPs peaks presumed to reflect lemniscal pathway activity include TP41 and N1. The maturation of these peaks follow a longer time course, becoming adult-like during mid to late adolescence.

The early maturation of Pa, Pb, and P2 suggests that activity in an auditory driven RAS pathway matures early, and is mostly adult-like by age 5 or 6. Based on the pattern of MMN maturation, the lemniscal-adjunct pathway also appears to be functional, if not entirely mature, by age 6. The amplitude and latency of TP41 and N1, assumed to reflect activation of the lemniscal pathway, do not become adult-like until middle or late adolescence.

Cochlear Implant Children: Thus far, the analysis of AEP data for the implanted children has been limited to three AEP peaks; one identified as TP41 as well as N1 and P2. Results of the analyses for TP41 latency change suggest that maturation of this peak is delayed approximately by the period of deafness prior to implantation. The N1 and P2 peaks are affected differently by deafness and cochlear implant use. The N1 peak is absent from the AEPs of all the implanted children tested thus far. With the exception of two implanted children, the P2 peak is also absent from the AEPs. Implanted children whose AEPs contain a P2 peak had normal hearing at least until the age of 3. Thus far, the MMN data have not been analyzed in sufficient detail to reach any conclusion except that the MMN evoked by a salient difference in stimulus duration is present in implanted children as young as age 6.

These results indicate that profound deafness and cochlear implant stimulation significantly alter the activity of neural sources generating the auditory evoked potentials. In implanted children, the maturation of at least one AEP peak was delayed while others were entirely absent. While the work with implanted subjects must be regarded as preliminary, these initial findings may indicate that the effects of deafness and cochlear implant use are not uniform across the pathways contributing components to the AEPs. More importantly, these data suggest that the absence of auditory input delays central auditory system maturation.

REFERENCES