

Central Auditory Maturation and Babbling Development in Infants With Cochlear Implants

Anu Sharma, PhD; Emily Tobey, PhD; Michael Dorman, PhD; Sneha Bharadwaj, PhD; Kathryn Martin, MA; Phillip Gilley, MS; Fereshteh Kunkel, MS

Objective: To examine the relationship between the maturation of central auditory pathways and the development of canonical (speechlike) babbling in infants with cochlear implants.

Design: Comparison of the latencies of the P1 cortical auditory evoked potential and vocalizations produced by subjects before they were fitted with a cochlear implant and at several time points within the first year after implantation.

Subjects: Two congenitally deaf children who were implanted with a multichannel cochlear implant at ages 13 and 14 months.

Interventions: P1 response latencies were recorded in response to a /ba/ stimulus before implantation and at several time points following implantation. Vocalizations produced by the subjects while interacting with their caregiver were audiorecorded twice before implantation and at monthly sessions following implantation.

Results: Subjects showed a rapid decrease in P1 latencies resulting in normal P1 latencies within about 3 months after implantation. Before implantation, the vocalizations were primarily of a precanonical nature. After 3 months' experience with the implants, the proportion of canonical vocalizations increased dramatically relative to the number of precanonical utterances.

Conclusions: Results of this study suggest that the development of P1 response latencies and the development of early communicative behaviors may follow a similar developmental trajectory in children implanted early. Although preliminary, these findings indicate that the development of early communicative behaviors following implantation may be positively influenced by the rate of plastic changes in central auditory pathways.

Arch Otolaryngol Head Neck Surg. 2004;130:511-516

IT IS REASONABLE TO HYPOTHESIZE that normal maturation of central auditory pathways is a precondition for the normal development of speech and language skills in children. That is, if central auditory pathways do not develop normally, presumably the perceptual skills that underlie speech production and perception will not develop normally. To test this hypothesis, one prerequisite is an objective measure of the maturation of central auditory pathways.

In a series of previously conducted experiments, we have investigated the maturation of central auditory pathways in normal-hearing and hearing-impaired children fitted with multichannel cochlear implants. Our objective measure of the maturity of central auditory pathways has been the latency of the P1 cortical auditory evoked potential. The P1 response is generated by auditory thalamic and cortical sources.¹⁻⁴ P1 latencies vary as a func-

tion of chronological age, reflecting central auditory pathway maturation. The latencies, thus, infer the maturational status of auditory pathways in normal-hearing, hearing-impaired, and congenitally deafened children who regain some aspects of hearing with a cochlear implant.⁵⁻⁸

Sharma et al⁶⁻⁸ examined P1 latencies in 104 congenitally deaf children using multichannel cochlear implants and ranging in ages from 1.3 years to 17.5 years who had at least 6 months of experience with the devices. A comparison of P1 latencies in the children using implants with those of age-matched normal-hearing peers revealed that children with cochlear implants who had the longest period of auditory deprivation before implantation (≥ 7 years) demonstrated abnormally long cortical response latencies to speech stimuli, and those who had the shortest period of auditory deprivation (approximately 3.5 years or less) demonstrated age-

From The University of Texas at Dallas, Callier Advanced Hearing Research Center, Dallas (Drs Sharma, Tobey, and Bharadwaj, Mss Martin and Kunkel, and Mr Gilley); and Department of Speech and Hearing Science, Arizona State University, Tempe (Dr Dorman). The authors have no relevant financial interest in this article.

appropriate latency responses. These data suggested that there was a sensitive period of 3.5 years during which the central auditory system was maximally plastic. Moreover, implantation within this time period resulted in age-appropriate cortical responses to sound within months after implantation. On the other hand, abnormal cortical responses were found in children who received implants after age 7 years—in some cases, even after 10 years of stimulation.⁸

In children using cochlear implants, there are striking similarities between the critical age cutoffs for normal P1 latencies and age cutoffs associated with the development of speech and language skills. Several investigators report children younger than 3 to 4 years with implants show significantly higher speech perception and language skills compared with children receiving implants after age 6 to 7 years.⁹⁻¹¹ Taken together, our studies of the development of P1 latency and the studies of speech and language development suggest that maturation of central auditory pathways may be intimately tied to the development of speech and language behaviors.

In this study, we examined the relationship between P1 latency and the development of a critical pre-speech behavior, ie, canonical babbling, in 2 children who received multichannel cochlear implants at ages 13 and 14 months. Normal-hearing infants undergo several stages of speechlike development.¹²⁻¹⁴ During the first 2 months of life, newborns produce comfort sounds. These sounds appear to be the precursors of vowel production. Between 2 and 3 months, infants enter a “going” stage. During this stage, they learn to articulate in the back of the mouth and, therefore, acquire a repertoire of vowel-like and g-like sounds. Between 4 and 6 months, infants expand their vocal repertoire to include growls, yells, whispers, squeals, and isolated vowel-like sounds. Well-formed syllables appear between age 7 and 10 months during the *canonical* babbling stage. During this stage, the use of reduplicated sequences such as [mama-mama] or [dadadada] begins. Reduplicated babbling is particularly important since it signals the first use of adult-like syllables and paves the way for a child to develop his or her first words.

Early communicative efforts of profoundly hearing-impaired or deaf infants parallel normal-hearing infants in several stages; however, deaf infants do not achieve the reduplicated or canonical babbling stage at the same time as normal-hearing infants. Deaf infants do not approach the canonical babbling period until at least 11 months and some do not obtain reduplicated syllables until 2.5 years of age or later.¹² Early intervention appears to positively affect the development of canonical babbling in infants with hearing impairments and increases the likelihood that they will begin the canonical babbling stage at an earlier age than if they remain unaided.¹²

Given that early amplification increases the likelihood of canonical babbling at more nearly normal ages, it is also likely that cochlear implants will increase the likelihood of canonical babbling in young children using cochlear implants. To examine this possibility, we examined the relationship in time between the develop-

ment of central auditory pathways, as measured by P1 latencies, and the development of canonical babbling.

METHODS

SUBJECTS

Subjects were 2 young, female children with cochlear implants. Guidelines of the institutional review board at The University of Texas at Dallas regarding human subjects were followed to obtain informed consent from the parents of the subjects. Subject 1 was initially identified as having a bilateral, severe-to-profound hearing loss at 7 months of age. The etiology of hearing loss was congenital cytomegalovirus. She was implanted with a MedEL (Innsbruck, Austria) C40+ device in the left ear and activated using the TEMPO+ speech processor at 13 months of age. The TEMPO+ speech processor was programmed using a continuous interleaved sampler (CIS) strategy with monopolar stimulation.

Subject 2 presented with a bilateral, severe-to-profound hearing loss, identified at birth by negative findings of a newborn screening test. Radiological findings revealed cochlear malformations, bilaterally. A Nucleus 24 cochlear implant device (Cochlear Americas, Englewood, Colo) was implanted in the right ear at 13 months of age. Activation of the Sprint speech processor was at 14 months of age using an Advanced Combination Encoder (ACE) speech coding strategy incorporating 900-Hz, 8-maxima, monopolar stimulation.

PROCEDURES

Electrophysiologic Recordings

Cortical auditory-evoked responses were recorded in response to a synthesized speech syllable /ba/. A full description of the stimulus can be found in a previous article.¹⁵ The stimulus was presented via a loudspeaker placed at an angle of 45° to the implanted side. Processors were set to the children's usual processor settings. Subjects were seated comfortably in a reclining chair placed in a sound booth. Subjects watched a videotape movie or cartoon of their choice on a TV monitor placed in front of them in the sound booth. Videotape audio levels were kept below 45 dB sound pressure level. Evoked potentials were collected using Cz as the active electrode. Cz refers to the vertex midline placement. The reference electrode was placed on the mastoid and a ground electrode on the forehead. Eye movements were monitored using a bipolar electrode montage (lateral outer canthus–superior outer canthus). The reference electrode was placed on the nonimplanted ear and an eye-blink detection electrode was placed on the nonimplanted side. Averaging was automatically suspended by the recording computer when eye blinks were detected. The recording window included a 100-millisecond prestimulus time and 600-millisecond poststimulus time. Incoming evoked responses were analog filtered from 0.1 Hz to 100 Hz. Approximately 300 response sweeps were collected for each subject. The test session including electrode application and evoked response recording lasted for about 30 minutes. Sweeps greater than ± 100 μ V were rejected off-line and the remaining sweeps were averaged to compute a grand average waveform for the individual subjects. P1 was defined as the first robust positivity in the auditory evoked potential waveform in the 50- to 175-millisecond range. For subject 1, P1 data were collected at the following postimplantation times (and ages): at implant activation (1.07 years), 1 week after implantation (1.09 years), 1 month after implantation (1.17 years), 3 months after implantation (1.34 years), and 12 months after implantation (2.06

years). For subject 2, P1 data were collected at the following postimplantation times (and ages): at implant activation (1.13 years), 1 month after implantation (1.24 years), and 3 months after implantation (1.38 years).

Vocalization Recordings

Vocalizations produced by the children while interacting with the caregiver were audiorecorded during 2 sessions separated by 2 weeks before cochlear implantation and during several sessions following cochlear implantation. The postimplantation sessions were recorded once a month and all sessions lasted approximately 45 minutes. The audiorecordings were transcribed in 13 categories by 2 phonetically trained graduate students using a broad transcription system and verified acoustically. Half of the samples were retranscribed for an interjudge reliability (0.86 for subject 1 and 0.89 for subject 2). These categories included the following: (1) squeals, (2) growls, (3) grunts, (4) raspberries, (5) whispers, (6) yells, (7) vowel-like, (8) consonant-like, (9) vowel, (10) consonant, (11) consonant-vowel syllables, (12) vowel-consonant syllables, and (13) untranscribable/other (either untranscribable or a nonlinguistic utterance). Categories 1 through 8 were classified as precanonical vocalizations and categories 9 through 12 were classified as canonical vocalizations. For these preliminary examinations of the data, all canonical and precanonical appearing vocalizations were included in the counts since we were interested in the proportions of broad categories occurring within a given test session. Occasionally, utterances were deemed nontranscribable, usually because an environmental sound masked the utterance.

For subject 1, vocalization data were collected at the following times (and ages): before implantation (baseline 1, 0.94 years; baseline 2, 0.96 years), 1 month after implantation (1.17 years), 2 months after implantation (1.22 years), and 3 months after implantation (1.34 years). For subject 2, vocalization data were collected at the following times (and ages): before implantation (baseline 1, 1.02 years; baseline 2, 1.05 years), 1 month after implantation (1.26 years), and 3 months after implantation (1.41 years).

RESULTS

Figure 1 shows the 95% confidence intervals for the normal development of P1 latencies from Sharma et al.⁸ The mean P1 latencies for subject 1 and subject 2 as a function of their chronological age and experience of stimulation with the cochlear implant are superimposed on the normal curves. In **Figure 2**, the proportion of precanonical vs canonical vocalizations are shown for subjects 1 and 2. Two baseline points were obtained before implantation and the postimplantation data points were obtained at monthly intervals following implant activation. The averaged evoked response waveforms are shown for both subjects in **Figure 3**.

For subject 1 (Figure 1A), a rapid decrease in P1 latencies is observed and results in normal P1 latencies within 3 months after implantation. The P1 latencies continue to decrease at a normal rate 12 months after implantation. Changes in P1 latency and in the overall morphologic characteristics of the evoked response waveform can be seen in Figure 3. An early negativity seen at hookup and 1 week diminishes as the duration of stimulation with the implant increases. The vocalization data for subject 1 shown in Figure 2A indicate primarily precanonical behaviors

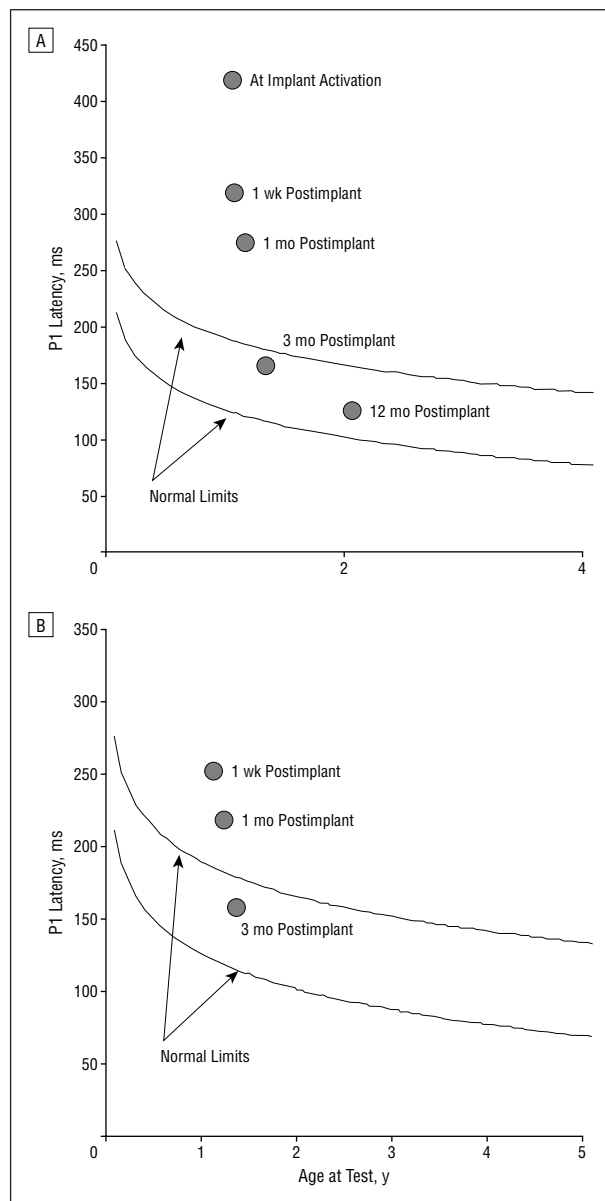


Figure 1. P1 latencies for are shown for a congenitally deaf child receiving a cochlear implant at 13 months (subject 1) (A) and at 14 months (subject 2) (B). The solid lines represent the 95% confidence intervals for normal development of P1 latencies. The circles represent the child's P1 latencies at different postimplantation intervals.

were produced when interacting with a caregiver before implantation and in the early months after implantation. However, by 3 months after implantation, predominantly canonical behaviors are observed when the child interacts with her caregiver.

For subject 2, a rapid decrease in P1 latencies occurs after implantation (Figure 1B). The morphologic changes in waveform that accompany the P1 latency decrease (Figure 3B) are similar to those described for subject 1. As can be seen in Figure 1B, normal P1 latencies for subject 2 are observable at 3 months after implantation. A parallel change in vocalizations is observed: the proportion of precanonical and canonical vocalizations before implantation shown in Figure 2B indicate the vocalizations were primarily of a precanonical nature. Af-

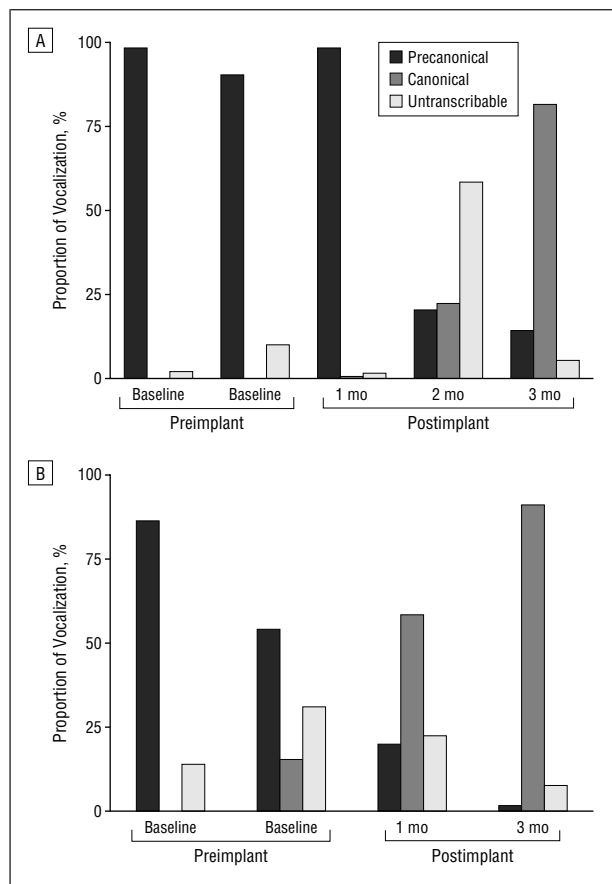


Figure 2. The proportion of precanonical vs canonical vocalizations are shown for subject 1 (A) and subject 2 (B). Two preimplantation (baseline) data points and several postimplantation intervals are indicated.

ter 3 months' experience with the implant, the proportion of canonical vocalizations increased dramatically with a comparable drop in precanonical utterances.

COMMENT

We examined the development of central auditory maturation (as evidenced by decreases in P1 latency) and the development of early communicative behavior (as evidenced by increases in canonical and decreases in precanonical vocalizations) in 2 children who received multichannel cochlear implants in early childhood. For both subjects, the electrophysiologic data demonstrate a rapid decrease in P1 latencies following implantation. These observations are consistent with our previous findings.^{7,8,16} In the present study, P1 latencies at 1 week after implantation were in the range of P1 latencies seen in normal-hearing newborns. These P1 latencies are consistent with our previous findings in children with early implantation.⁷ Morphologic changes in the evoked response waveform, mainly an early negativity that diminishes with auditory experience, are consistent with those reported previously.⁷ The 2 children receiving implants in this study at 13 and 14 months of age also demonstrated age-appropriate P1 latencies within 3 months after implantation. In our previous work with children receiving implants on average between 2.5 and 3.0 years of age, P1 latencies were not age appropriate until 6 to 8

months after implantation. These preliminary data suggest that hearing-impaired children receiving implants very early (around age 1 year) may benefit from relatively greater plasticity of the auditory pathways than children with implantation later within the developmentally sensitive period.

Are the rapid changes in P1 latency unique to electrical stimulation or do these changes reflect a general response of a deprived central auditory system to the initiation of stimulation? In order to speak to this issue we have examined the development of P1 latencies in children with congenital hearing loss who are fitted with hearing aids. In **Figure 4** and **Figure 5** we present data from one individual, a child with congenital, moderate-to-severe sensorineural hearing loss who was fitted with hearing aids at age 11 months. We describe this case because the age at intervention with the hearing aids (11 months) was similar to the age of implantation in our previously described cases (13 and 14 months). His unaided pure-tone average threshold was 75 dB HL (hearing level) and his aided pure-tone average threshold was 40 dB HL. As can be seen in Figure 4, his P1 latencies were outside normal limits at the time of initial fitting with the hearing aid and decreased to within normal limits 5 months after hearing aid use. The P1 latencies continued to develop normally with 12 months of hearing aid use. As auditory experience with the hearing aids increased, an early negativity in the waveform diminished (Figure 5). This change in response morphology was similar to that found for children fit with cochlear implants. These results suggest that rapid changes in P1 latencies and changes in response morphology are not unique to electrical stimulation but rather reflect the response of a deprived sensory system to new stimulation.

The exact neurophysiologic mechanisms for the rapid development of P1 latencies after early implantation are not clear. Kral et al¹⁷ have shown that congenitally deaf cats show a restricted (atypical) pattern of activation within the layers of the primary auditory cortex. Perhaps early stimulation with a cochlear implant initiates a more widespread (typical) sequence of activation within and between cortical layers resulting in robust cortical responses and shorter response latencies over time.

It would be useful to find converging evidence for rapid development within other deprived sensory systems following the onset of stimulation. Such evidence exists from the visual system. Maurer et al¹⁸ assessed visual acuity in human infants who were congenitally deprived of patterned visual input by cataracts. The cataracts were removed at 1 week to 9 months of age. They found that acuity improved rapidly, with some improvement apparent after as little as 1 hour of visual input. Critically, the rate of development of visual acuity following cataract removal was significantly greater than normal (relative to age-matched controls).

Both children in the present study showed rapid improvements in early communicative behavior as indicated by their vocalization data. Before implantation, both children produced predominantly precanonical vocalizations when interacting with their caregivers. Over 70% of the utterances produced during these preimplant interactive sessions were composed of early components

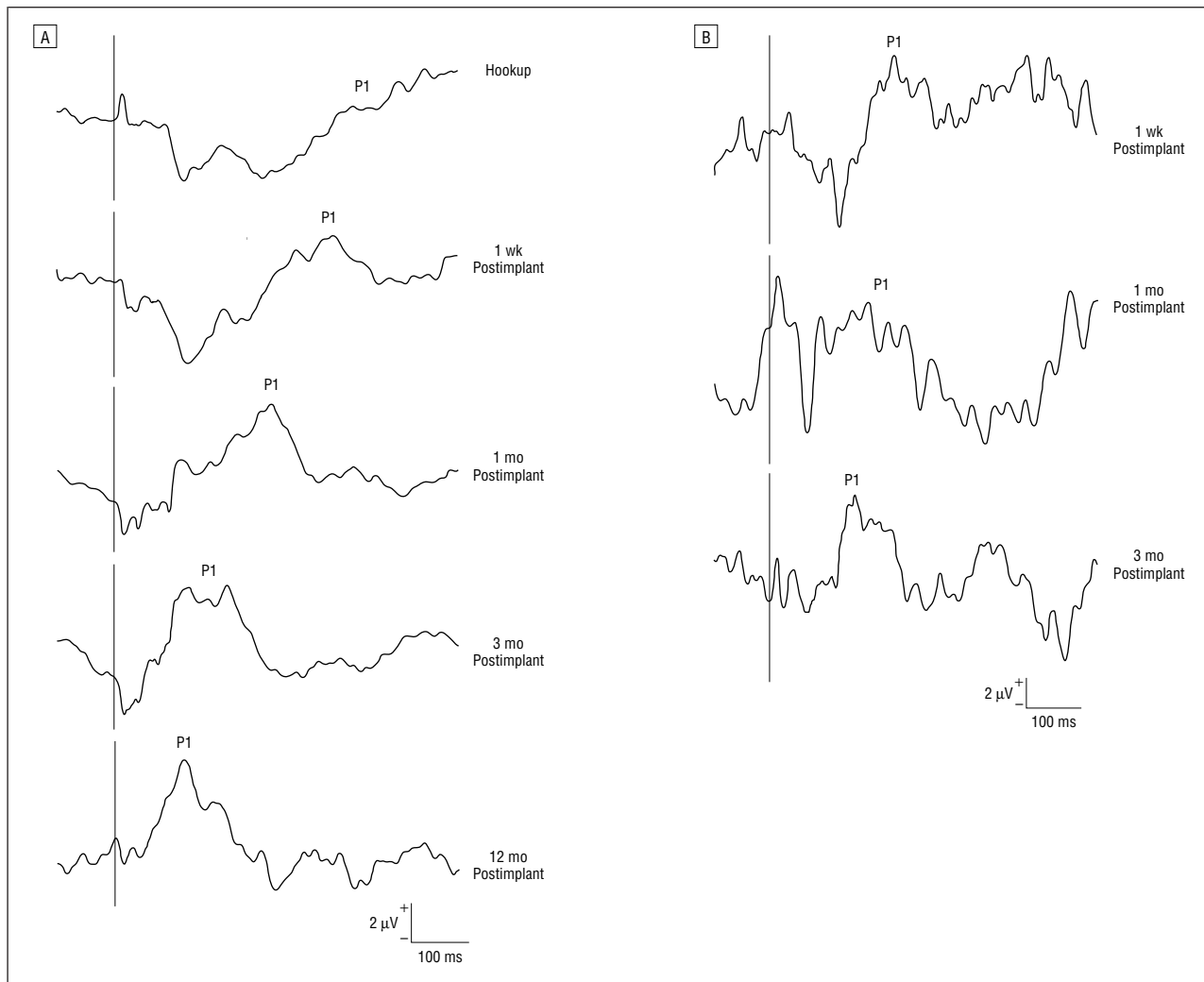


Figure 3. Averaged evoked response waveforms are shown for subject 1 (A) and subject 2 (B) at different postimplantation intervals. The P1 response peak is labeled.

of speech behaviors including growls, squeals, raspberries, grunts, whispers, and yells. In normal-hearing infants, these vocalization behaviors typically occur during 4 to 6 months of age. Prolonged use of these vocalizations at later chronological ages in the hearing-impaired children before implantation is consistent with previous reports indicating few changes in hearing-impaired children before intervention.¹² Following implantation, both children demonstrated consistent patterns of acquisition of vowel, consonant, and syllable utterances. Use of canonical utterances is observed within 1 month of auditory experience via the multichannel implants. The percentage of canonical behaviors continues to increase rapidly and predominates the utterances types as early as 3 months after implantation. The rapid progression to canonical behaviors after implantation suggests auditory experience with a cochlear implant promotes the acquisition of babbling behaviors underlying the motoric and early linguistic skills contributing to a young child's first word.

Taken together, the P1 latency and vocalization data for the 2 subjects in this study suggest that the development of P1 response latencies and the development of

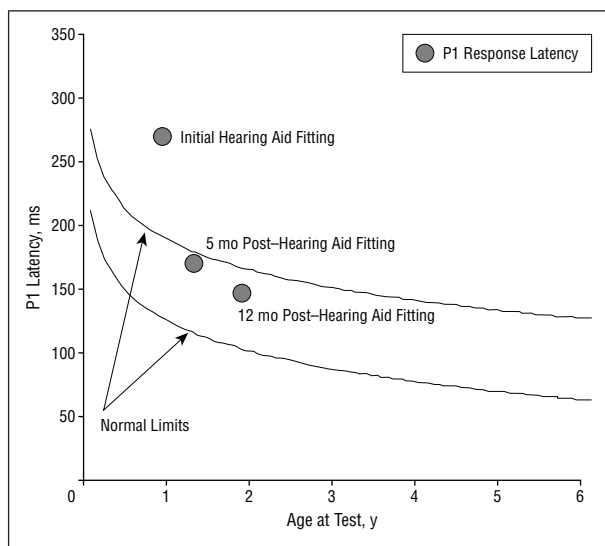


Figure 4. P1 latencies for are shown for a child with congenital hearing impairment who was first fitted with a hearing aid at age 11 months. The solid lines represent the 95% confidence intervals for normal development of P1 latencies. The circles represent the child's P1 latencies at the time of the initial hearing aid fitting and then 5 and 12 months later.

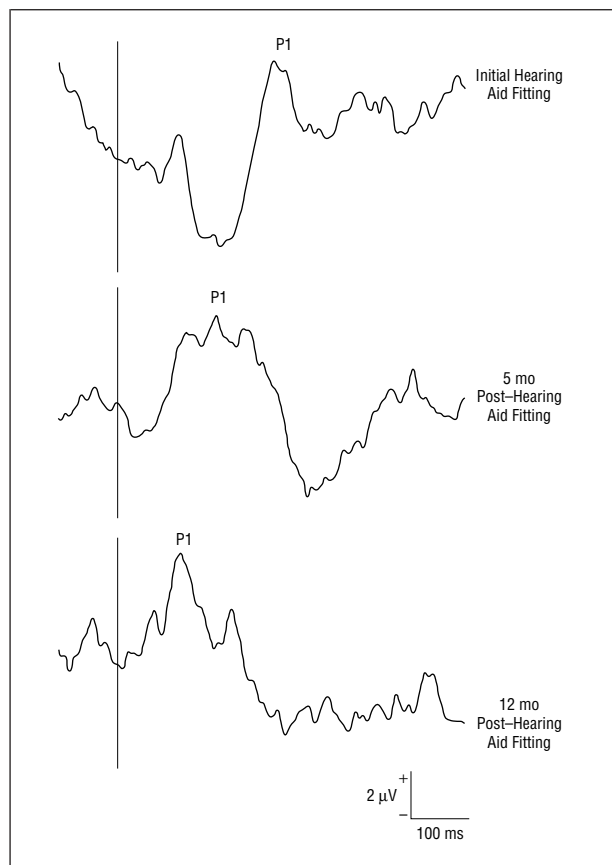


Figure 5. Averaged evoked response waveforms are shown for a child with congenital hearing impairment who was first fitted with a hearing aid at age 11 months. The waveforms are shown for different time intervals after hearing aid fitting. The P1 response peak is labeled.

early communicative behavior may follow a similar developmental trajectory. That is, after relatively minimal experience with the implant, rapid changes in P1 latency were observed in parallel with a major change in vocalization behavior from precanonical to speechlike canonical babbling. Although preliminary, these findings suggest that the development of early communication behaviors following implantation may be promoted by changes in central auditory pathways.

Submitted for publication September 15, 2003; final revision received January 30, 2004; accepted February 2, 2004.

This work was supported in part by the National Institute of Deafness and Other Communication Disorders and the National Institute of Child, Health and Development.

This study was presented at the Ninth Symposium on Cochlear Implants in Children; April 24, 2003; Washington, DC.

Corresponding author and reprints: Anu Sharma, PhD, Callier Advanced Hearing Research Center, The University of Texas at Dallas, 1966 Inwood Rd, Dallas, TX 75235 (e-mail: anu.sharma@utdallas.edu).

REFERENCES

1. Erwin RJ, Buchwald JS. Midlatency auditory evoked responses in the human and the cat model. *Electroencephalogr Clin Neurophysiol Suppl.* 1987;40:461-467.
2. McGee T, Kraus N. Auditory development reflected by middle latency response. *Ear Hear.* 1996;17:419-429.
3. Liegeois-Chauvel C, Musolino A, Badier JM, Marquis P, Chauvel P. Evoked potentials recorded from the auditory cortex in man: evaluation and topography of the middle latency components. *Electroencephalogr Clin Neurophysiol.* 1994;92:204-214.
4. Ponton CW, Eggermont JJ. Of kittens and kids: altered cortical maturation following profound deafness and cochlear implant use. *Audiol Neurootol.* 2001;6:363-380.
5. Eggermont JJ, Ponton CW, Don M, Waring MD, Kwong B. Maturation delays in cortical evoked potentials in cochlear implant users. *Acta Otolaryngol.* 1997;117:161-163.
6. Sharma A, Dorman MF, Spahr AJ. Rapid development of cortical auditory evoked potentials after early cochlear implantation. *Neuroreport.* 2002;13:1365-1368.
7. Sharma A, Dorman M, Spahr A, Todd NW. Early cochlear implantation in children allows normal development of central auditory pathways. *Ann Otol Rhinol Laryngol Suppl.* 2002;189:38-41.
8. Sharma A, Dorman M, Spahr T. A sensitive period for the development of the central auditory system in children with cochlear implants. *Ear Hear.* 2002;23:532-539.
9. Kirk KI, Miyamoto RT, Lento CL, Ying E, O'Neill T, Fears B. Effects of age at implantation in young children. *Ann Otol Rhinol Laryngol Suppl.* 2002;189:69-73.
10. Summerfield AQ, Nakisa MJ, McCormick B, Archbold S, Gibbin KP, Odonoghue GM. Use of vocalic information in the identification of /s/ and /sh/ by children with cochlear implants. *Ear Hear.* 2002;23:58-77.
11. Manrique M. Long term results with cochlear implants in children. Presented at: Sixth European Symposium on Paediatric Cochlear Implantation; 2002; Canary Islands, Spain.
12. Eilers RE, Oller DK. Infant vocalizations and the early diagnosis of severe hearing impairment. *J Pediatr.* 1994;124:199-203.
13. Eilers RE, Cobo-Lewis AB, Vergara KC, Oller DK, Friedman KE. A longitudinal evaluation of the speech perception capabilities of children using multichannel tactile vocoders. *J Speech Hear Res.* 1996;39:518-533.
14. Eilers RE, Gavin WJ, Oller DK. Cross-linguistic perception in infancy: early effects of linguistic experience. *J Child Lang.* 1982;9:289-302.
15. Sharma A, Kraus N, McGee TJ, Nicol TG. Developmental changes in P1 and N1 central auditory responses elicited by consonant-vowel syllables. *Electroencephalogr Clin Neurophysiol.* 1997;104:540-545.
16. Sharma A, Dorman M, Todd NW, Gilley P, Fainberg J, Martin K. Central auditory system development in children with hearing impairment. Presented at: Association for Research in Otolaryngology; February 26, 2003; Daytona Beach, Fla.
17. Kral A, Hartmann R, Tillein J, Heid S, Klinke R. Hearing after congenital deafness: central auditory plasticity and sensory deprivation. *Cereb Cortex.* 2002;12:797-807.
18. Maurer D, Lewis T, Brent HP, Levin AV. Rapid improvement in the acuity of infants after visual input. *Science.* 1999;286:108-110.