A Sensitive Period for the Development of the Central Auditory System in Children with Cochlear Implants: Implications for Age of Implantation

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Objective: **The aim of the present experiment was to assess the consequences of cochlear implantation at different ages on the development of the human central auditory system.**

Design: **Our measure of the maturity of central auditory pathways was the latency of the P1 cortical auditory evoked potential. Because P1 latencies vary as a function of chronological age, they can be used to infer the maturational status of auditory pathways in congenitally deafened children who regain hearing after being fit with a cochlear implant. We examined the development of P1 response latencies in 104 congenitally deaf children who had been fit with cochlear implants at ages ranging from 1.3 yr to 17.5 yr and three congenitally deaf adults. The independent variable was the duration of deafness before cochlear implantation. The dependent variable was the latency of the P1 cortical auditory evoked potential.**

Results: **A comparison of P1 latencies in implanted children with those of age-matched normal-hearing peers revealed that implanted children with the longest period of auditory deprivation before implantation—7 or more yr—had abnormal cortical response latencies to speech. Implanted children with the shortest period of auditory deprivation—approximately 3.5 yr or less—evidenced age-appropriate latency responses within 6 mo after the onset of electrical stimulation.**

Conclusions: **Our data suggest that in the absence of normal stimulation there is a sensitive period of about 3.5 yr during which the human central auditory system remains maximally plastic. Plasticity remains in some, but not all children until approximately age 7. After age 7, plasticity is greatly reduced. These data may be relevant to the issue of when best to place a cochlear implant in a congenitally deaf child.**

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A central issue in the field of pediatric cochlear implants is the optimal age range for implanting a congenitally deaf child. The prevailing wisdom is that implantation at an early age will produce better results than implantation at a relatively late age. This view stems, in part, from research on the effects of auditory deprivation on the development of auditory function in a variety of animals. Although studies have shown that the central auditory system establishes functional neural connections in the absence of sound (Hartmann, Shepard, Heid, & Klinke, 1997; Klinke, Kral, Heid, Tillein, & Hartmann, 1999), auditory deprivation causes widespread degeneration in the central auditory system (e.g., Hardie, & Shepherd, 1999; Leake, Snyder, Hradek, & Rebscher, 1992; Moore, 1994; Ryugo, Pongstaporn, Hutchton, & Niparko 1999; Ryugo, Rosenbaum, Kim, Niparko, & Saada, 1998). These changes include reduction of cell density in the spiral ganglion, anteroventral cochlear nucleus and ventral cochlear nucleus; changes in neural projections between brainstem nuclei (Nordeen, Killackey, & Kitzes, 1983); reduced cortical synaptic activity in cortico-cortical and cortico-thalamic connections (Kral, Hartmann, Tillein, Heid, & Klinke, 2000); a reduced number of primary dendrites in cortical pyramidal cells; and take over of auditory cortical areas by visual function (Lee et al., 2001; Finney, Fine, & Dobkins, 2001).

It is reasonable to suppose that the degenerative effects described above, if found in humans, would reduce the effectiveness of a cochlear implant. On this view, the best time to implant a child would be before the effects of sensory deprivation alter the development and plasticity of the central auditory system. This view is supported by data from congenitally deaf white cats (CDCs), and both mice and rats fit with cochlear implants. Klinke, Hartmann, Heid, Tillein, and Kral (2001) and Kral, Hartmann, Tillein, Heid, and Klinke (2002) report that as the duration of intracochlear stimulation increases for CDCs, the amount of cortical tissue activated increases, provided that cochlear implantation takes place before 6 mo of age. Kral et al. (2000) have shown that synaptic currents in young implanted CDCs are similar to those in hearing cats if stimu-

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lation is initiated early but not if stimulation is delayed until 6 mo of age. Hsu et al. (2001) examined Fos immunoreactivity as a functional marker for neuronal activity in the dorsal cochlear nucleus and inferior colliculus of neonatally deafened, implanted rats. The endogenous protein c-Fos is upregulated in response to transsynaptic stimulation and membrane electrical activity and can be detected by immunocytochemical techniques. Fos immunolabeling is used to study the pattern of neuronal activation in response to sound in the central auditory nervous system. Deafened rats who had been implanted at 21 days postdeafening had a larger number of Fos-immunoreactive cells in the dorsal cochlear nucleus and the inferior colliculus than deafened rats who were implanted at 120 days postdeafening. Hsu et al. (2001) concludes that electrical stimulation of the inner ear is more effective in eliciting gene expression associated with development of a functional network of central auditory pathways when initiated early in development than when initiated late in development. Ryugo and colleagues (Lee, Cahill, & Ryugo, Reference Note 1; Ryugo et al., 1997) examined the development of the endbulbs of Held in the cochlear nucleus (which are axosomatic and myelinated auditory nerve fibers that end in spherical bushy cells) in congenitally deaf Shaker-2 mice. They reported that, although the endbulbs of Held in deaf animals begin to develop normally, by about 2 to 7 mo of age these mice exhibit dramatic alterations in synaptic development that are clearly distinguishable from the endbulbs in normal-hearing littermates and that would impair the timing resolution of synaptic transmission. In sum, there is ample evidence from animal models of congenital deafness for the existence of a sensitive period for the development of auditory pathways.

We should assume the presence of a similar sensitive period in humans, but experimental data to support this assumption is, understandably, sparse. Ponton and colleagues (Eggermont, Ponton, Don, Waring, & Kwong 1997; Ponton, Don, Eggermont, Waring, Kwong, & Masuda, 1996a; Ponton & Eggermont, 2001; Ponton, Don, Eggermont, Waring, & Masuda, 1996b) have reported, for children with 4.5 yr or more of deafness, a delayed latency, relative to age-matched normal-hearing children, of the P1 cortical evoked potential. Ponton and Eggermont (2001) have suggested that children with cochlear implants who experience a sufficiently long period of deafness before the age of 6 to 8 yr never develop a fully functional set of axons in superficial layers of the auditory cortex.

Evoked potential studies and functional imaging studies have described recruitment of the auditory cortex by the visual and somatosensory systems in congenitally deaf humans (Finney et al., 2001; Lee et al., 2001; McFeely, Antonelli, Rodriguez, & Holmes, 1998; Nishimura et al., 1999). Lee et al. (2001) reported that the extent of cross-modal recruitment of the auditory cortex increases as the duration of deafness increases, deterring the restoration of auditory processing in the auditory cortex of long-term deafened individuals after cochlear implantation. Taken together, the scant data from humans is consistent with the concept of a sensitive period for the development of the auditory pathways but offer little or no information about the age that marks the end of the period or the factors that affect the duration of the period.

We have begun an investigation of the development, deterioration, and plasticity of the human central auditory system with the long-term goal of assessing the consequences of cochlear implantation at different ages during infancy, childhood and adolescence. Our experiments mirror the earlier experiments of Ponton et al. (1996a, 1996b) in concept, i.e., using the latency of the P1 cortical evoked response to assess the developmental status of the auditory pathway. In our studies, the P1 cortical auditory evoked potential (CAEP) was elicited in response to a speech stimulus—the syllable /ba/. The P1 response is generated by auditory thalamic and cortical sources (Erwin & Buchwald, 1987; Liegeois-Chauvel, Musolino, Badier, Marquis & Chauvel, 1994; McGee & Kraus, 1996). Ponton and Eggermont (2001) suggest that the surface positivity of the P1 response is consistent with "a relatively deep sink ([in cortical] layers IV and lower III) and a superficial current return." The latency of P1 reflects the accumulated sum of delays in synaptic propagation through the peripheral and central auditory pathways (Eggermont et al., 1997). Because P1 latency varies as a function of chronological age (Ceponiene, Cheour, & Näätänen, 1998; Cunningham, Nicol, Zecker, & Kraus, 2000; Ponton, Eggermont, Kwong, & Don 2000; Sharma, Kraus, McGee, & Nicol, 1997), P1 latency can be used to infer the maturational status of auditory pathways in congenitally deafened children who regain hearing after being fit with a cochlear implant. To assess the normal time course for development of P1 latency, we assessed P1 latency in 136 normal-hearing subjects ranging from 0.1 to 20 yr of age. Against these normative data, we compared the P1 latencies from 104 congenitally deaf children who were fit with implants at ages ranging from 1.3 yr to 17.5 yr and from three congenitally deaf adults fit with implants at 18, 31 and 34 yr.

METHODS

Subjects

CAEPs were recorded from 51 normal-hearing persons ranging in age from 0.1 yr to 20 yr of age. These data were combined with data from a previous study (Sharma et al., 1997) for a combined total of 136 normal-hearing persons ranging in age from 0.1 yr to 20 yr.

One hundred twenty-one persons with cochlear implants were tested. In approximately 12% of subjects (14 out of 121) cases the P1 response was obscured by the presence of a stimulus artifact in the first 100 msec of the recording. Although the artifact was more likely to be present in certain types of cochlear implants, it was seen in cochlear implant devices of all manufacturers. These subjects were excluded from the study. In the present study we included 107 subjects who wore cochlear implants and in whom a P1 CAEP was recorded and identified. These included 104 children and teenagers who ranged in age at the time of testing from 2.3 yr to 18 yr. Three congenitally deaf adults aged 20, 33 and 35 yr were also tested. Subjects were either congenitally deafened or presented with severe to profound hearing loss by age 1 yr. For the purpose of this paper we will refer to the children as a group as congenitally deaf. Recordings were made at least 6 mo after device switch on. Post hoc the implant users were divided into three groups, an early-, a middle-, and a late-implanted group. The early-implanted group consisted of 57 children who had been implanted by age 3.5 yr. The average age at implantation was 2.3 yr and the average number of years of implant use at the time of testing was 3 yr. The middle-implanted group consisted of 29 children who were implanted between ages 3.6 and 6.5 yr. The average age at implantation was 5 yr and the average number of years of implant use at the time of testing was 3 yr. The late-implanted group consisted of 21 persons who had been implanted after 7 yr of age. This group included three congenitally deaf adults. The average age of implantation for the children in the late group was 11.2 yr. The three congenitally deaf adults were implanted at ages 18, 31 and 34 yr. For the late group (including the three adults) the average number of years of implant use at the time of testing was 3.2 yr.

Procedures

Stimulus Presentation • Cortical auditory evoked responses were recorded in response to a synthesized speech syllable /ba/. The duration of the speech sound was 90 msec. This stimulus was identical to the one used in Sharma et al. (1997) and Sharma, Dorman, Spahr, and Todd (2002a). The 5 formant CV stimulus was generated using the Klatt (1980) synthesizer. The starting frequencies of F1 and F2 were 234 Hz and 616 Hz, respectively. The center frequencies for the formants of the vowel /a/ were 769 Hz, 1232 Hz, 2862 Hz, 3600 Hz, and 4500 Hz for F1, F2, F3, F4, and F5, respectively. F3, F4, and F5 were steady-state formants. 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 over 35 msec and then decreased to 80 Hz over 55 msec.

The stimulus was presented at an offset-to-onset interstimulus interval of 610 msec. The stimulus was delivered via a loudspeaker placed at an angle of 45° to the right of the normal-hearing subjects. For the implanted subjects, the speaker was moved to their implanted side. Implanted subjects were instructed to choose a setting on their processor at which they could hear the stimulus at a comfortable loudness level.

Evoked Response Recording Procedures • Subjects were seated comfortably in a reclining chair placed in a sound booth. Younger children were seated on their parent's laps. Subjects watched a video tape 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 SPL. We have found this to be an effective way of engaging young subjects (see also Kraus, McGee, Carrell, & Sharma, 1995). Evoked potentials were collected using Cz as the active electrode. The reference electrode was placed on the right mastoid and the ground on the forehead. Eye movements were monitored using a bipolar electrode montage (lateral outer canthus-superior outer canthus). For implanted children the reference electrode was placed on the nonimplanted ear and the eye-blink monitoring 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 msec prestimulus and 600 msec poststimulus time. Incoming evoked responses were analog filtered from 0.1 to 100 Hz. At least two runs of 300 response sweeps were collected for each subject. The test session including electrode application and evoked response recording lasted about 30 minutes.

Data Analysis • Sweeps greater than ±100 uV were rejected offline, after that the remaining sweeps were averaged to compute an averaged waveform. Individual subjects had at least two averaged AEP waveforms of 300 sweeps each. If the waveforms were judged replicable based on visual inspection, the waveforms were averaged together to

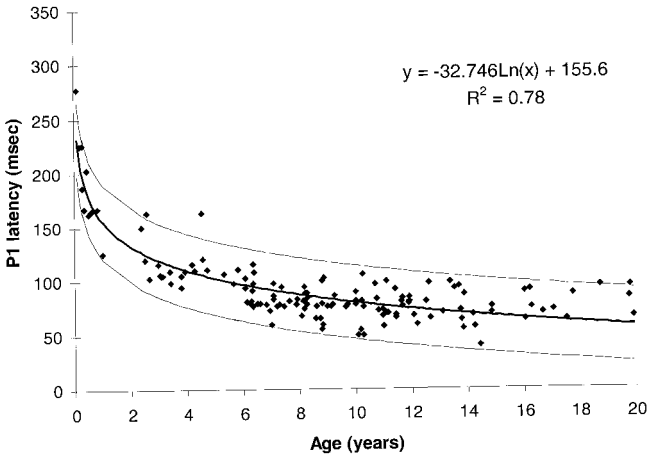

Figure 1. P1 latencies as a function of age for normal-hearing children. The line of best-fit and the 95% confidence interval are superimposed on the raw data.

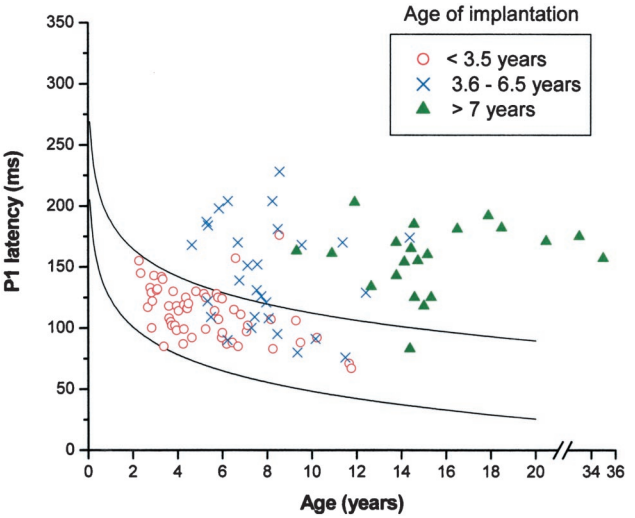

Figure 2. P1 latencies as a function of chronological age for children with cochlear implants. The solid functions are the 95% confidence limits for normal-hearing children. P1 latencies for children implanted before age 3.5 yr (early-implanted group) are shown as circles. P1 latencies for children implanted between age 3.5 yr and 6.5 yr (middle-implanted group) are shown as crosses. P1 latencies for children implanted after age 7 yr (late-implanted group) are shown as triangles.

create a grand average waveform for individual subjects. P1 was defined as the first robust positivity in the waveform. In the case of a double-peaked P1 response, P1 latency was typically marked on the first peak. Latency values were determined for P1 without regard to the chronological age and the age of implantation of subjects.

RESULTS

Normal-Hearing Children

The latency of P1 as a function of age is shown in Figure 1. The line of best fit and the 95% confidence interval are superimposed on the raw data. The data were best-fit by a growth function based on the natural log of age (latency = 155.6 +(-32.746)*(LN(Age)); $R^2 = 0.78$; $p < 0.0001$). Visual inspection indicates that latencies decrease rapidly in the first decade of life, and then decrease more gradually in the second decade of life. These results are consistent with those reported by Sharma et al. (1997), Ponton et al. (2000), and Cunningham et al. (2000). As can be seen in Figure 1, P1 latency continues to decrease from 15 to 20 yr of age. This finding is consistent with that reported by Cunningham et al. (2000) who showed that P1 latency decreased significantly from 13 to 15 yr to 19 to 27 yr of age.

Children with Implants

A 1-way ANOVA showed that the subjects in the early, middle and late-implanted groups were not significantly different with respect to their duration of implant use ($F = 0.05$; $p = 0.90$). The latencies for the implanted children are shown in Figure 2 as a function of chronological age at time of testing. The solid functions on each plot are the 95% confidence limits for normal-hearing children (taken from Fig. 1). P1 latencies for 20 out of 21 late-implanted persons (triangles) were outside the 95% confidence limit for age-matched normal-hearing children. The latencies of 19 of the 29 children in the middle group (crosses) were outside the range of normal. In contrast, 55 out of the 57 children in the early group (circles) had latencies within the range of normal. The proportion of latencies falling within the range of normal differed significantly between the early-implanted group and the late-implanted group (Fishers Exact Test for two proportions, $p = 0.0000001$). The proportion of latencies falling within the range of normal differed significantly between the early-implanted group and the middle-implanted group (Fishers Exact Test for two proportions, $p = 0.0000001$). Figure 3 shows a grand average waveform for a subset of 18 early-implanted children (average age 3.8 yr) and an age-matched group of normal-hearing peers (average age 3.5). A 1-way ANOVA showed that the ages of the children in the two groups was not significantly different ($F = 0.01$; $p = 0.18$). The average duration of implant use for the group of 18 early implanted children was 1.8 yr. As shown in Figure 3, the peak latencies of P1 for the early-implanted children and their normal-hearing peers are similar. Figure 4 shows a grand average waveform for a subset of 13 late-implanted children (average age 15.1 yr) and an age-matched

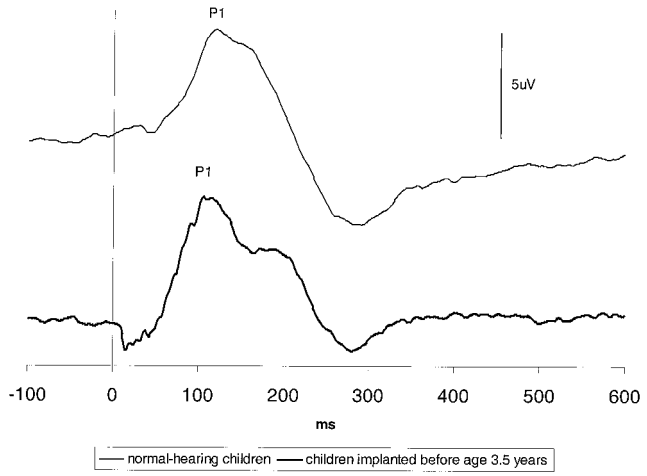

Figure 3. Grand average auditory evoked responses from a subset of 18 children with cochlear implants who were implanted under the age of 3.5 yr (bottom waveform) and age-matched normal-hearing peers (top waveform). P1 responses for the two groups are noted.

group of normal-hearing peers (average age 15.2 yr). A 1-way ANOVA showed that the ages of the children in the two groups was not significantly different ($F = 1.83$; $p = 0.18$). The average duration of implant use for the group of 13 late-implanted children was 3.1 yr. The P1 response is delayed by about 100 msec for the late-implanted children.

DISCUSSION

We examined P1 response latencies in congenitally deafened children fit with cochlear implants at an early, middle, or late age in childhood. Children in all three groups had used their implants on average for a similar time period (about 3 yr). However, as a group, congenitally deaf children implanted under 3.5 yr of age demonstrated age-appropriate P1 latencies by 6 mo postimplantation. In contrast, congenitally deaf subjects implanted after age 7 yr had delayed P1 latencies. The later finding is consistent with the previous findings of Ponton et al. (1996a, 1996b). Our results demonstrate that for congenitally deaf children there is a time period during early development of approximately 3.5 yr when the auditory system is relatively nondegenerate and/or maximally plastic. This finding is consistent with results of studies from different animal species showing sensitive periods in auditory development and showing that implantation within these periods produces the best results with respect to the functional development of the central auditory system (Hsu et al., 2001; Klinke et al., 2001; Kral et al., 2000, 2002).

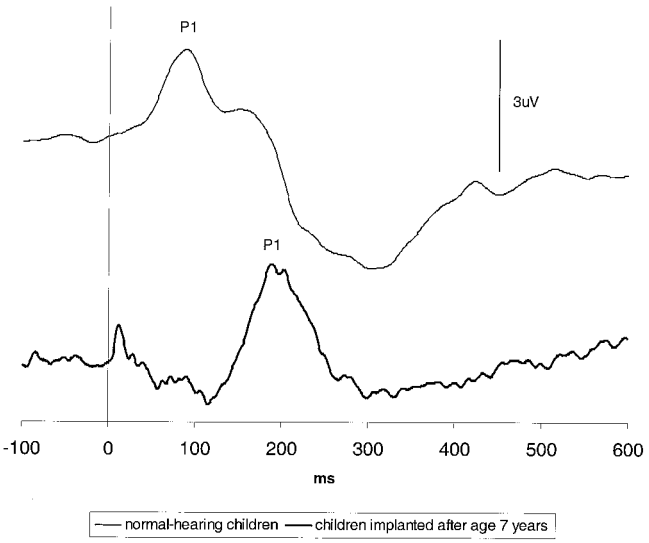

Figure 4. Grand average auditory evoked responses from a subset of 13 subjects with cochlear implants who were implanted later than age 7 yr (bottom waveform) and age-matched normal-hearing peers (waveform). P1 responses for the two groups are noted.

What developmental processes allow children deprived of sound for 3 to 4 yr to generate normal latency P1s within 6 mo after implantation? One possibility is that central auditory pathways develop normally in the absence of stimulation and remain minimally degenerate after periods of auditory deprivation lasting up to at least 3 to 4 yr. This possibility finds some support from the work of Huttenlocher and Dhabolkar (1997) on synaptogenesis, or synaptic proliferation, in the auditory cortex. Huttenlocher and Dhabolkar (1997) posthumously measured synaptic density in normal-hearing human subjects to estimate the timeline for synaptogenisis in the human auditory cortex. The data indicate that new synapse formation begins in the prenatal period and continues for the first 4 yr of life. After age 4, pathways undergo refinement mainly by elimination of synapses. Critically, Huttenlocher and Dhabolkar (1997) suggest that synaptogenisis is intrinsically regulated, i.e., is independent, to a large extent, of the auditory experiences of the child. On this view, central auditory pathways in early childhood may develop to a greater or lesser extent even in the absence of sound stimulation.

Another possibility is that the absence of early stimulation does alter the development of pathways but that the pathways are highly plastic for 3 to 4 yr. On this view, the effects of deprivation may be overcome within a short time after the initiation of stimulation. Evidence for this hypothesis comes from recent work in the visual system. Maurer, Lewis, Brent, and Levin (1999) assessed visual acuity in human infants who were congenitally deprived of patterned visual input by cataracts. The cataracts were removed at 1 wk to 9 mo of age.

Maurer et al. (1999) found that acuity improved rapidly, with some improvement apparent after as little as 1 hr of visual input. Critically, the rate of development of visual acuity after cataract removal was significantly greater than normal (relative to age-matched controls). It would be parsimonious to find a similar effect in the auditory system after implantation, and data described below suggest that this is the case.

Sharma, Dorman, and Spahr (2002b) examined changes in the cortical auditory evoked response waveform and in P1 latency in 22 prelingually deafened, early-implanted children. They found that the cortical auditory evoked response waveform undergoes rapid changes in morphology in the first 6 to 8 mo after implantation. During the same period, P1 latencies decrease rapidly. The rate of decrease of P1 response latencies in early-implanted children was greater than for age-matched normal-hearing controls and resulted in age-appropriate latencies within 6 to 8 mo after onset of stimulation.

In the present study, about two thirds of the children in the middle-implanted groups and nearly all of the subjects in the late-implanted group had delayed P1 latencies. This outcome is consistent with the previous results of Ponton et al. (1996a, 1996b). This result, relative to the results for early-implanted children, suggests that increasing periods of auditory deprivation progressively alter synaptic efficiency and transmission times.

As reviewed in the Introduction, there are many possible mechanisms for the presence of a deprivation effect in children implanted relatively late in childhood. One mechanism is cross-modal recruitment of the auditory cortex by the visual or somatosensory modalities (Finney et al., 2001; Lee et al., 2001; McFeely et al., 1998; Nishimura et al., 1999). Using PET scans, Lee et al. (2001) compared glucose metabolism rates in the auditory cortices of 17 prelingually deafened patients ranging in age from 2 yr to 20 yr before cochlear implantation. The degree of glucose metabolism preimplantation was taken to be an indicator of the degree to which cross modal recruitment of the auditory cortex had occurred. The authors report that the degree of hypo metabolism before implantation (which was greater for younger subjects) was positively correlated to the speech perception scores after implantation. Based on these results, Lee et al. (2001) conclude that the extent of cross-modal recruitment of the auditory cortex in humans increases as the duration of deafness increases. Lee et al. (2001) also suggest that the age beyond which the effects of cross-modal plasticity in the auditory cortex are more difficult to reverse is about 6.5 yr. This suggestion is concordant with our finding that implantation after age 7 leads to delayed P1 latencies. In citing the Lee et al. data we do not suggest that cortical take over is the principal mechanism leading to delayed cortical responses in late implanted children. We note the Lee et al. data only because of the convergence in estimates of the age beyond which abnormal cortical responses are more likely.

Ponton and Eggermont (2001) have recently proposed another mechanism for the abnormal development of P1 latencies in "late implanted" children. On this view, layer II axons in the auditory cortex do not mature normally in the absence of stimulation. As a consequence, the N1b wave of the cortical response, which arises from layer II, is not generated normally. Because the presence of the N1b wave is one of the factors that contributes to the shortening of P1 latency, the absence of the wave contributes to the abnormal latencies seen in the late implanted children.

The morphology of the CAEP may also be relevant to this issue. As reported in Sharma et al. (2002b), a large negative peak at approximately 150 msec is found in congenitally deaf children ($N = 22$) soon after device switch on. A similar negative waveform at approximately 100 to 150 msec is seen in deaf white cats after the initiation of stimulation with a cochlear implant (Klinke et al., 1999; Kral, 2002, personal communication). In early-implanted children the latency and amplitude of this response changes over time—the latency becomes shorter and the amplitude smaller (Sharma et al., 2002). On the other hand, this initial negativity persists for a longer period in late-implanted children, perhaps contributing to the abnormally long P1 latencies in late-implanted children. This negativity may provide another marker for assessing the plasticity of the auditory pathway.

While approximately two-thirds of the children in the middle age group (3.5 to 6.5 yr old) demonstrated delayed P1 latencies, the rest showed age-appropriate latencies. Critically, there were several children in the latter half of the age range (e.g., children fit at ages 5.1, 5.5, 5.8, and 6.6 yr) who had age-appropriate latencies. This outcome suggests that age at implantation is not the only variable that influences central auditory development. In an unpublished study we have found that several children who were implanted later in life, but who had significant aided benefit before implantation, have age-appropriate P1 responses. The extent to which variables such as preimplant unaided and aided hearing thresholds; age of first use of amplification; duration of hearing aid use and amount and nature of aural habilitation influence development postimplantation will be one focus of future investigations.

Longitudinal Changes in Latency • In the present study there were three late-implanted subjects who even after long periods of stimulation with the implant (8, 8.7 and 8.9 yr, respectively) demonstrated delayed P1 latencies. Therefore, an issue of interest is how long it would take for evoked potential latencies to reach values within the range of normal.

In recent experiments, we have assessed longitudinal changes in P1 latency for children implanted at different ages. At device switch-on, latencies for most implanted children are similar to those of a newborn or infant less than several months old (Sharma et al., 2002b). This is consistent with the suggestion from Ponton et al. (1996a, 1996b) that little or no development occurs in a pathway deprived of stimulation. In a sample of children implanted at age 11 or older we have found very little or no change in P1 latency after 1 to 2 yr of electrical stimulation. In contrast, in children implanted by age 7 we have found decreases in P1 latency within a year of implantation. Critically, in children implanted before age 3.5 we have found very large decreases in latency within the first several months after device switch on (Sharma et al., 2002b). The rate of change in latency immediately after implantation may be a powerful, and clinically useful, tool in assessing the residual plasticity in the auditory pathway of children fit with cochlear implants.

In summary, when viewed from the perspective of P1 latencies in congenitally deaf children, the auditory system appears maximally plastic for a period of approximately 3.5 yr. Implantation within this time window allows normal P1 latencies to sound within months after initiation of electrical stimulation. The auditory system in some, but not all, children remains plastic for up to 7 yr. The factors that promote a prolonged period of plasticity in these children are not known. Auditory deprivation for more than 7 yr substantially alters the latency of the P1 cortical response to sound. This effect is due, most likely, to a marked reduction of neural plasticity. These data may be relevant to the issue of when best to place a cochlear implant in a congenitally deaf child.

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