



ELSEVIER

Available online at www.sciencedirect.com

 ScienceDirect

Journal of Communication Disorders 40 (2007) 284–294

Journal of
**Communication
Disorders**

Central auditory development: Evidence from CAEP measurements in children fit with cochlear implants

Michael F. Dorman^{a,*}, Anu Sharma^b, Phillip Gilley^b,
Kathryn Martin^c, Peter Roland^d

^a *Department of Speech and Hearing Science, Arizona State University, AZ, USA*

^b *Department of Speech, Language and Hearing Science, University of Colorado, Boulder, CO, USA*

^c *The Callier Advanced Hearing Research Center, University of Texas at Dallas, TX, USA*

^d *University of Texas Southwestern Medical Center, Dallas, TX, USA*

Received 27 February 2007; accepted 1 March 2007

Abstract

In normal-hearing children the latency of the P1 component of the cortical evoked response to sound varies as a function of age and, thus, can be used as a biomarker for maturation of central auditory pathways. We assessed P1 latency in 245 congenitally deaf children fit with cochlear implants following various periods of auditory deprivation. If children experience less than 3.5 years of auditory deprivation before implantation, P1 latencies fall into the range of normal following 3–6 months of electrical stimulation. Children who experience greater than 7 years of deprivation, however, generally do not develop normal P1 latencies even after years of stimulation. Moreover, the waveforms for these patients can be markedly abnormal. Cortical reorganization stimulated by deprivation is likely to be a significant factor in both variation in the latency and morphology of the cortical evoked response to sound for children fit with a cochlear implant and variation in the development of oral speech and language function.

Learning outcomes: The reader will be introduced to research using cortical evoked responses (CAEPs), positron emission tomography (PET) scans and in-depth recording from the auditory cortex of congenitally deaf cats that converges on the existence of a sensitive period for the development of central auditory pathways in children. The reader will also be provided with two case studies that illustrate the use of the P1 response as biomarker for development of central auditory pathways. Finally, suggestions for future research will be provided.

© 2007 Elsevier Inc. All rights reserved.

* Corresponding author at: Coor Hall 2211, Tempe, AZ 85287-0102, USA. Tel.: +1 480 965 3345; fax: +1 480 965 8516.

E-mail address: mdorman@asu.edu (M.F. Dorman).

1. Introduction

It is now well documented that congenitally deaf children fit with cochlear implants can achieve high levels of oral speech and language skills (e.g., Pisoni, Cleary, Geers, & Tobey, 1999; Svirsky, Teoh, & Neuburger, 2004). However, success depends very critically on the age at which a child receives an implant (e.g., Colletti et al., 2005; Connor, Craig, Raudenbush, Heavner, & Zwolan, 2006; Kirk et al., 2002; Lee et al., 2004; Manrique, 2002; Nikilopoulos, O'Donoghue, & Archbold, 1999; Summerfield, 2002). Fig. 1, redrawn from Lee et al. (2004), shows scores on the Korean version of the CID sentences as a function of a child's age at the time of implantation. Children implanted before the age of 4 generally achieve high scores on the task of sentence recognition. Children implanted after the age of 7 generally achieve poor scores. Children implanted between age 4 and 7 show a complete range of scores—some are excellent; some are very poor. These data, and the data reported in the references cited above, suggest that there is a sensitive period during early development during which stimulation must be delivered if high levels of oral speech and language skills are to be acquired. In this paper, we review research that we, and others, have conducted which speaks to, and provides an account for, the existence of a sensitive period for central auditory development.

2. The P1 cortical auditory evoked potential as a biomarker

The P1 response (shown in Fig. 2, inset) is a robust positivity at a latency of 100–300 ms in young children. P1 is generated by auditory thalamic and cortical sources (Erwin & Buchwald, 1987; Liegeois-Chauvel, Musolino, Badier, Marquis, & Chauvel, 1994; McGee and 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” (p. 366). The latency of P1 reflects the accumulated sum of delays in synaptic propagation through the peripheral and central auditory pathways (Eggermont, Ponton, Don, Waring, & Kwong, 1997).

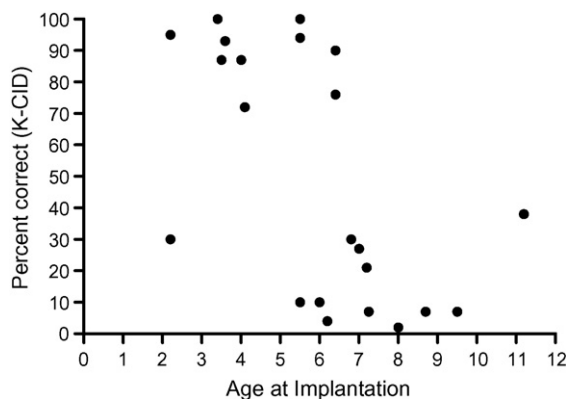


Fig. 1. Sentence recognition as a function of age at implantation (figure redrawn from Lee et al., 2004).

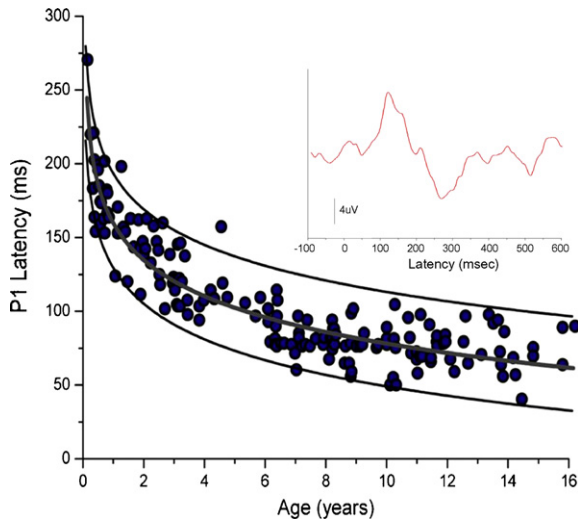


Fig. 2. P1 latency as a function of age for 190 normal-hearing children. The upper and lower solid lines indicate the 95% confidence limits for the data. The middle solid line is the line of best fit. The inset is a CAEP from a 3-year-old normal-hearing child.

As shown in Fig. 2, the latency of P1 changes during infancy and childhood (Ceponiene, Cheour, & Naatanen, 1998; Cunningham, Nicol, Zecker, & Kraus, 2000; Gilley, Sharma, Dorman, & Martin, 2005; Ponton, Eggermont, Kwong, & Don, 2000; Sharma, Dorman, & Spahr, 2002; Sharma, Kraus, McGee, & Nicol, 1997). In normal-hearing newborns the mean P1 latency is approximately 300 ms. Over the first 2–3 years of life there is a rapid decrease in latency (to approximately 125 ms at age 3) and then a more gradual decrease into the second decade of life. The mean P1 latency in normal-hearing adults (ages 22–25 years) is approximately 60 ms. Ninety-five percent confidence intervals for normal development of P1 latency are described in Sharma et al. (2002) and are shown in Fig. 2. Because P1 latency varies as a function of chronological age, P1 latency can be used as a biomarker to infer the maturational status of auditory pathways in infants and children. Of particular interest are infants and children with significant hearing loss.

3. A sensitive period for the development of the human central auditory pathways

Studies of congenitally deaf children fit with cochlear implants at different times during childhood have allowed us to infer the existence and time limits of a sensitive period for the development of central auditory pathways. In Fig. 3, we show P1 latencies from 245 congenitally deaf children who received electrical stimulation of the auditory pathway for a period of at least 6 months. P1 latencies are plotted against the 95% confidence intervals of P1 latencies derived from 190 normal-hearing children. Children who were deprived of sound for greater than 7 years (diamonds), show delayed P1 latencies. These data are

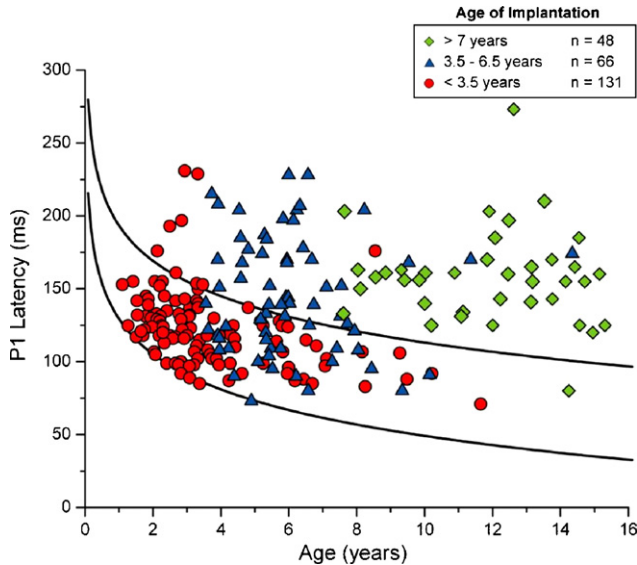


Fig. 3. P1 latency as a function of age at implantation for 245 children fit with a cochlear implant. Diamonds indicate latencies for children implanted at age 7 years and greater. Triangles indicate latencies for children implanted between ages 3.5 and 6.5 years. Circles indicate latencies for children implanted at less than 3.5 years.

consistent with data from animal models (Kral & Tillein, 2006) and provide clear evidence of the effects of sensory deprivation on central auditory pathways in humans. About one-half of the children who experienced fewer years of deprivation, between 3.5 and 7 years (triangles), had normal P1 latencies and almost all children who experienced fewer than 3.5 years of deprivation (circles) showed normal P1 latencies. These results are consistent with those from a previous study (Sharma et al., 2002) and suggest that central auditory pathways are maximally plastic (in response to auditory stimulation) for a period of about 3.5 years in early childhood. If stimulation is delivered within that period, then P1 latency and morphology reach age-normal values within 3–6 months following the onset of stimulation. However, if stimulation is withheld for more than 7 years, then most children exhibit a delayed P1 latency and abnormal P1 morphology, even after years of implant use. Indeed, the different morphology of the response suggests that the P1 measured in normal-hearing children and early implanted children has a different generator than the response we have labeled as ‘P1’ in late implanted children. This outcome suggests reorganization of auditory cortex in the face of long-term auditory deprivation (Gilley, Sharma, & Dorman, 2006).

Our electrophysiological data and data from PET imaging converge to suggest a time line for a sensitive period. Kang et al. (2004), Lee et al. (2004, 2001), and Oh et al. (2003) recorded resting glucose-metabolism rates in the auditory cortices of prelingually deafened children and adults *before* cochlear implantation and related these rates to speech perception scores *after* implantation. The degree of glucose metabolism before implantation was taken to be an indicator of the degree to which cross-modal recruitment of the auditory cortex had occurred. That is to say, the auditory cortices should be ‘silent,’

i.e., should be hypometabolic, in the face of years of auditory deprivation. However, if the cortices have been recruited by other cortical functions, then the cortices would not be hypometabolic. Lee et al. (2004) reported that the degree of hypometabolism before implantation was greatest for younger subjects and was positively correlated with the speech perception scores after implantation. That is, children who were implanted before age 4 showed the largest amount of hypometabolism in the auditory cortices before implantation and, following implantation, had the highest speech perception scores (as shown in Fig. 1). This age cut-off (4 years) is consistent with the 3.5 years cut-off for maximal plasticity of the central auditory pathways suggested by Sharma et al. (2002). The Lee et al. (2001) data also suggest that following 6.5–7.5 years of deprivation the auditory cortices experience significant cross-modal reorganization, i.e., they were found to be less hypometabolic than the cortices of younger children. This finding fits with the Sharma et al. (2002) finding of delayed P1 latencies following 7 years of auditory deprivation. It was also the case the speech perception scores of the children in Lee et al. (2004) implanted over the age of 7 were much poorer than the scores of the children implanted under the age of 4.

Other studies which have explored the issue of sensitive periods for central auditory development in cochlear implanted children have found roughly similar age cut-offs in early development. Based on their finding of absent N1 in cochlear implanted subjects who had been deaf for a period of at least 3 years under the age of 6, Eggermont and Ponton (2003) suggest that this time period reflects a critical period for cortical maturation and speech perception. Gordon, Papsin, and Harrison (2005) suggest that the auditory system in children who have experienced longer periods of deprivation (>5 years) shows less potential for plastic change (as measured by middle latency responses) than in children who have experienced fewer years of deprivation (<5 years).

In general, there are similarities between the critical age cut-offs for normal P1 latencies and from PET data and age cut-offs associated with the development of speech and language skills. Several investigators have reported that children implanted under ages 3–4 years show significantly higher speech perception scores and better language skills than children implanted after age 6–7 years (Kirk et al., 2002; Manrique, 2002; Summerfield, 2002). For a review of sensitive periods as they relate to speech perception and language acquisition in children with cochlear implants see Harrison, Gordon, and Mount (2005) and Tomblin, Barker, and Hubbs (in press).

To summarize, electrophysiologic and functional brain imaging (PET) data in humans suggests that there is a sensitive period of about 3.5–4.0 years in early development during which auditory stimulation is delivered to a highly plastic central auditory system. If more than 7 years of deprivation precede the onset of stimulation, then that stimulation is delivered to a re-organized auditory cortex which shows reduced plasticity.

4. Cortical mechanisms underlying the sensitive period

Congenitally deaf cats can be used as a model system to study cortical activity after the end of the sensitive period. In kittens, the sensitive period for development of central auditory pathways lasts up to 5 months of age (Kral, Hartmann, Tillein, Heid, & Klinke,

2000). When electrical stimulation is started after 4 months of deafness there is a delay in the activation of supragranular layers of the cortex and a near absence of activity at longer latencies and in infragranular layers (layers V and VI) (Kral et al., 2000). The near-absence of outward currents in layers IV and III of congenitally deaf cats suggests incomplete development of inhibitory synapses and an alteration of information flow from layer IV to supragranular layers. This abnormal pattern of activity within the auditory cortex is likely to be the basis for the abnormalities in evoked potential morphology and latency we find in children implanted after the end of the sensitive period. Because the higher order auditory cortex projects back to A1 (primary auditory cortex) mainly to the infragranular layers, the absence of activity in infragranular layers suggests a decoupling of primary cortex from higher order auditory cortex (Kral et al., 2000; Kral, Hartmann, Tillein, Heid, & Klinke, 2002; Kral, Tillein, & Heid, 2005). Such a decoupling would allow other sensory input to predominate in the higher order auditory cortex in children deprived of sound for a long period. Decoupling of primary cortex from higher order auditory may result in the recruitment of the higher order auditory cortex by other modalities (such as vision) (Bavelier & Neville, 2002; Lee et al., 2004, 2001). Decoupling between primary and higher order language cortex in children deprived of sound for a long period and a reorganization of higher order auditory cortex provides a mechanism for the language learning difficulties of children who receive an implant after the end of the sensitive period.

5. Clinical use of the P1 biomarker

The plot of P1 latency as a function of age for normal-hearing listeners, shown in Fig. 2, has allowed us to assess the development of central auditory pathways in a number of clinical circumstances. In the next section, we present two cases to demonstrate clinical use of the P1 CAEP.

5.1. Case 1

The patient was a female child with an unremarkable birth history who failed her newborn hearing screening. Auditory brainstem response (ABR) testing at age 2 weeks suggested a bilateral, severe-to-profound hearing loss. Further testing suggested congenital cytomegalovirus (CMV) infection as the etiology underlying the hearing loss. The patient was fitted with a hearing aid at age 5 months. When tested in soundfield the unaided PTA for the right ear was 100 dB; there was no response at any frequency for the left ear. Soundfield testing using binaural hearing aids revealed an aided PTA of 78 dB HL. After 7 months of hearing aid use CAEPs were recorded in an aided soundfield setting. A P1 response could not be detected at the maximum output levels of the equipment. This suggested that the auditory stimulation provided by the hearing aid was likely to be inadequate for development of central auditory pathways. The patient met the standard criteria for cochlear implantation and was fitted with a cochlear implant in her right ear at age 19 months. Fig. 4 shows P1 latencies at the time of implant activation, 1 week, 1 month, 4 months and 7 months after implantation. The latency of P1 decreased by 200 ms over a 4-month period. At 4 months the P1 latency was within normal limits and

continued to be within normal limits when tested 7 months after implantation. Results of an informal speech and language evaluation indicated progress in acquisition of speech and language.

In this case, the absence of a P1 response after 7 months of hearing aid use provided clear evidence that the auditory stimulation provided by the hearing aid was not sufficient for central auditory development. After implantation, the latency of the P1 decreased rapidly to within normal limits indicating that the implant was providing stimulation not provided by the hearing aid.

5.2. Case 2

The patient was a male child diagnosed with a hearing loss after failing a newborn hearing screening. Hearing testing at age 5 months using ABR testing revealed a severe-to-profound hearing loss. Further testing revealed that the hearing loss was secondary to Goldenhar syndrome. In addition to the sensorineural hearing loss, the child also had microtia and atresia in the right ear. He was fitted with a hearing aid in the left ear at age 9 months. Behavioral audiometric testing revealed a profound hearing loss in the right ear and a severe-to-profound hearing loss in the left ear. Soundfield testing revealed no aided benefit in the left ear. CAEP testing was performed in an aided soundfield setting 17 months after hearing aid fitting. A P1 response could not be elicited at the maximum output levels of the equipment. The absence of a P1 response suggested that the hearing aid was not providing the stimulation necessary for development of the central pathways. The patient met the standard criteria for cochlear implantation and was fitted with a cochlear implant in the left ear at age 2 years 7 months. CAEP recordings were repeated at the time of implant hookup, and then 6 and 13 months post-implantation. As shown in Fig. 5 the P1 latency was significantly delayed at hookup—an outcome consistent with a relatively unstimulated central auditory system. Moreover, the P1 latency remained prolonged at 6 and 13 months

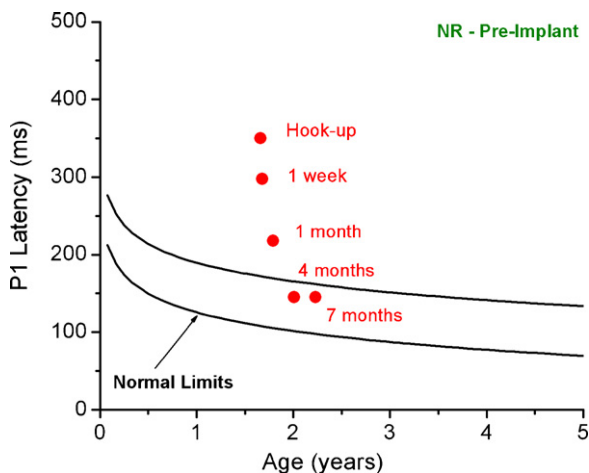


Fig. 4. Case 1: P1 latency as a function of age. The parameter is the time since device hookup.

post-implantation. The prolonged latency of the P1 indicated a lack of normal central–auditory pathway development. This finding is consistent with the audiologist’s observation that the child did not wear his cochlear implant consistently and was behaviorally unresponsive to auditory stimuli. This case demonstrates that not all early-implanted children implanted achieve normal development of central auditory pathways. For that reason, the CAEP may be useful in monitoring changes in central auditory development in children with complex medical conditions.

6. Research needs

Our initial clinical results are promising with respect to the use of P1 latency as a measure of central auditory development in children who receive intervention with a hearing aid or a cochlear implant. However, before the measurement of P1 latencies can gain widespread clinical use a number of problems and issues must be addressed. In the case of cochlear implants the most critical problem is an electrical artifact from the pulse train generated by the cochlear implant. The artifact is especially prominent when using a long duration speech stimulus, e.g., the 90 ms ‘ba’ stimulus used to generate the latency-by-age norms in Fig. 2. We have developed time-intensive techniques to minimize this artifact in our laboratory (Gilley, Sharma, Dorman, Finley, et al., 2006) but have not found a technique that can be easily and quickly used in the clinic. A rapid and effective solution to the artifact problem will need to be found before CAEPs can be used with cochlear implant patients in a busy clinical setting.

Finally, if we wish children, who experience a disconnection syndrome and/or cortical reorganization, to achieve high levels of oral speech and language development, then it will be necessary to create stimulation programs and rehabilitation programs that take into account cortical reorganization. Programs of this type would be of enormous practical benefit.

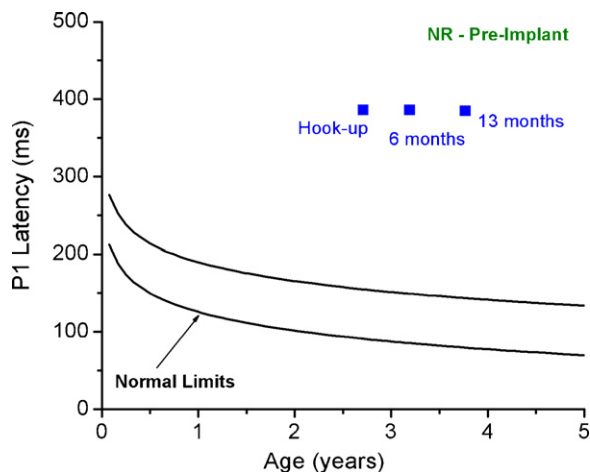


Fig. 5. Case 2: P1 latency as a function of age. The parameter is time since device hookup.

Acknowledgement

The research described in this paper was supported by grants NIDCD R01-DC004552 and R01-DC 006257 to author AS.

Appendix A. Continuing education

1. Studies of oral speech and language development in congenitally deaf children fit with a cochlear implant find that:
 - a. Children implanted late in childhood achieve levels of performance equal to that of children implanted early in childhood
 - b. There is a positive correlation between age of implantation and achievement of speech and language skills
 - c. There is a negative correlation between age at implantation and achievement of speech and language skills
 - d. Cochlear implants have little effect on a deaf child's development of speech and language skills
2. The latency of the P1 response:
 - a. Is about 300 ms in children
 - b. Is about 150 ms in children
 - c. Varies as a function of a child's age
 - d. Changes little as a function of a child's age
3. Children fit with a cochlear implant:
 - a. Show abnormal P1 latencies
 - b. Show abnormal P1 latencies if implanted after the age of 3.5
 - c. Show normal P1 latencies if implanted under the 3.5 years
 - d. Show normal P1 latencies following a long period of stimulation with a cochlear implant
4. The results of PET scans in children taken before implantation:
 - a. Suggest that unstimulated auditory cortex remains 'silent' after years of auditory deprivation
 - b. Suggest that unstimulated auditory cortex is reorganized after years of auditory deprivation
 - c. Suggest that early stimulation is not sufficient to prevent reorganization of auditory cortex
 - d. Suggest that reorganization of auditory cortex is independent of the duration of deprivation
5. The 'decoupling hypothesis' of [Kral et al. \(2005\)](#) suggests that the relatively poor oral speech and language abilities of children implanted after a long period of auditory deprivation are due to:
 - a. A decoupling of the cochlea from the brain stem
 - b. A decoupling of the cochlear nucleus from the inferior colliculus
 - c. A decoupling of anterior and posterior portions of the cortex
 - d. A decoupling of primary auditory cortex from higher order auditory cortex

References

- Bavelier, D., & Neville, H. J. (2002). Cross-modal plasticity: Where and how? *Nature reviews. Neuroscience*, *3*, 443–452.
- Ceponiene, R., Cheour, M., & Naatanen, R. (1998). Interstimulus interval and auditory event-related potentials in children: Evidence for multiple generators. *Electroencephalography and Clinical Neurophysiology*, *108*, 345–354.
- Colletti, V., Carner, M., Miorelli, V., Guida, M., Colletti, L., & Fiorino, F. G. (2005). *Laryngoscope*, *115*(3), 445–449.
- Connor, C. M., Craig, H., Raudenbush, S., Heavner, K., & Zwolan, T. (2006). The age at which young deaf children receive cochlear implants and their vocabulary and speech production growth: Is there an added value for early implantation? *Ear & Hearing*, *27*(6), 628–644.
- Cunningham, J., Nicol, T., Zecker, S., & Kraus, N. (2000). Speech-evoked neurophysiologic responses in children with learning problems: Development and behavioral correlates of perception. *Ear & Hearing*, *21*(6), 554–568.
- Eggermont, J. J., & Ponton, C. W. (2003). Auditory-evoked potential studies of cortical maturation in normal hearing and implanted children: Correlations with changes in structure and speech perception. *Acta Otolaryngologica*, *123*, 249–252.
- Eggermont, J. J., Ponton, C. W., Don, M., Waring, M. D., & Kwong, B. (1997). Maturation delays in cortical evoked potentials in cochlear implant users. *Acta Otolaryngologica*, *117*, 161–163.
- Erwin, R. J., & Buchwald, J. S. (1987). Midlatency auditory evoked responses in the human and the cat model. *Electroencephalography and Clinical Neurophysiology Supplement*, *40*, 461–467.
- Gilley, P. M., Sharma, A., & Dorman, M. (2006b). Reorganization of auditory and visual cortical pathways during auditory deprivation. *Paper presented at the 2nd Shanghai International Conference on Physiological Biophysics: Audition & Vision*.
- Gilley, P. M., Sharma, A., Dorman, M., Finley, C. C., Panch, A., & Martin, K. (2006a). Minimization of cochlear implant stimulus artifact in cortical auditory evoked potentials. *Clinical Neurophysiology*, *117*(8), 1772–1782.
- Gilley, P. M., Sharma, A., Dorman, M., & Martin, K. (2005). Developmental changes in refractoriness of the cortical auditory evoked potential. *Clinical Neurophysiology*, *116*(3), 648–657.
- Gordon, K., Papsin, B., & Harrison, R. (2005). Effects of cochlear implant use on the electrically evoked middle latency response in children. *Hearing Research*, *204*(1–2), 78–89.
- Harrison, R. V., Gordon, K. A., & Mount, R. J. (2005). Is there a critical period for cochlear implantation in congenitally deaf children? Analyses of hearing and speech perception performance after implantation. *Developmental Psychobiology*, *46*(3), 252–261.
- Kang, E., Lee, D. S., Kang, H., Lee, J. S., Oh, S. H., Lee, M. C., et al. (2004). Neural changes associated with speech learning in deaf children following cochlear implantation. *Neuroimage*, *22*(3), 1173–1181.
- Kirk, K., Miyamoto, R., Lento, C., Ying, E., O'Neill, T., & Fears, B. (2002). Effects of age at implantation in young children. *Annals of Otolaryngology, Rhinology, and Laryngology Supplement*, *189*, 69–73.
- Kral, A., Hartmann, R., Tillein, J., Heid, S., & Klinke, R. (2000). Congenital auditory deprivation reduces synaptic activity within the auditory cortex in a layer-specific manner. *Cerebral Cortex*, *10*, 714–726.
- Kral, A., Hartmann, R., Tillein, J., Heid, S., & Klinke, R. (2002). Hearing after congenital deafness: Central auditory system plasticity and sensory deprivation. *Cerebral Cortex*, *12*, 797–807.
- Kral, A., & Tillein, J. (2006). Brain plasticity under cochlear implant stimulation. *Advances in Otorhinolaryngology*, *64*, 89–108.
- Kral, A., Tillein, J., & Heid, S. (2005). Postnatal cortical development in congenital auditory deprivation. *Cerebral Cortex*, *15*, 552–562.
- Lee, H. J., Kang, E., Oh, S. H., Kang, H., Lee, D. S., Lee, M. C., et al. (2004). Preoperative differences of cerebral metabolism relate to the outcome of cochlear implants in congenitally deaf children. *Hearing Research*, *203*, 2–9.
- Lee, D. S., Lee, J. S., Oh, S. H., Kim, S. K., Kim, J. W., Chung, J. K., et al. (2001). Cross-modal plasticity and cochlear implants. *Nature*, *409*, 149–150.
- Liegeois-Chauvel, C., Musolino, A., Badier, J. M., Marquis, P., & Chauvel, P. (1994). Evoked potentials recorded from the auditory cortex in man: Evaluation and topography of the middle latency components. *Electroencephalography and Clinical Neurophysiology*, *92*, 204–214.

- Manrique, M. (2002). Long term results with cochlear implants in children. *Paper presented at the 6th European Symposium on Pediatric Cochlear Implantation*.
- McGee, T., & Kraus, N. (1996). Auditory development reflected by middle latency response. *Ear & Hearing, 17*, 419–429.
- Nikilopoulos, T., O'Donoghue, G., & Archbold, S. (1999). Age at implantation: Its importance in pediatric cochlear implantation. *Laryngoscope, 109*, 595–599.
- Oh, S. H., Kim, C. S., Kang, E. J., Lee, D. S., Lee, H. J., Chang, S. O., et al. (2003). Speech perception after cochlear implantation over a 4-year time period. *Acta Otolaryngologica, 123*(2), 148–153.
- Pisoni, D., Cleary, M., Geers, A., & Tobey, E. (1999). Individual differences in effectiveness of cochlear implants in children who are prelingually deaf: Some new process measures of performance. *Volta Review, 101*, 111–164.
- Ponton, C. W., & Eggermont, J. (2001). Of kittens and kids: Altered cortical maturation following profound deafness and cochlear implant use. *Audiology & Neuro-Otology, 6*, 363–380.
- Ponton, C. W., Eggermont, J. J., Kwong, B., & Don, M. (2000). Maturation of human central auditory system activity: Evidence from multi-channel evoked potentials. *Clinical Neurophysiology, 111*, 220–236.
- Sharma, A., Dorman, M., & Spahr, A. (2002). A sensitive period for the development of the central auditory system in children with cochlear implants: Implications for age of implantation. *Ear & Hearing, 23*, 532–539.
- Sharma, A., Kraus, N., McGee, T. J., & Nicol, T. G. (1997). Developmental changes in P1 and N1 central auditory responses elicited by consonant–vowel syllables. *Electroencephalography and Clinical Neurophysiology, 104*, 540–546.
- Summerfield, Q. (2002). The costs, effectiveness, and cost-effectiveness of cochlear implantation: What have we learned in the last decade? *Lecture presented at the 7th international cochlear implant conference*.
- Svirsky, M., Teoh, S. W., & Neuburger, H. (2004). Development of language and speech perception in congenitally, profoundly deaf children as a function of age at cochlear implantation. *Audiology & Neuro-Otology, 9*, 224–233.
- Tomblin, B., Barker, B., & Hubbs, S. (in press). Ontogenic constraints on language development in children with cochlear implants. *International Journal of Audiology*.