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The Development of Speech Perception in Children Using Cochlear Implants: Effects of Etiologic Factors and Delayed Milestones

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Abstract

Hypothesis: Speech perception outcomes for cochlear implantation of children

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Outline

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vary over a wide range, and it is hypothesized that central pathologic states associated with certain causes of hearing impairment account for a substantial part of the variance.

Study Design: A retrospective analysis was carried out to ascertain the relationships between speech perception, etiologic factors, and central pathologic states as indicated by preoperative delayed motor milestones and/or cognitive delays.

Setting: Data were obtained from the pre-and postoperative records of patients attending a hospital cochlear implant clinic.

Patients: Results for 75 consecutive patients up to age 5 years who underwent implantation were included in the study.

Intervention: Patients received a 22-electrode cochlear prosthesis and were seen by the clinic for regular tune-up and assessments. Home-and school-based habilitation was recommended by the clinic.

Main Outcome Measures: Speech perception measures were classified on a five-point scale to allow for different evaluation procedures at different ages and developmental stages.

Results: The incidence of motor and cognitive delays were fairly evenly spread across etiologic factors, except for cytomegalovirus, which had a much higher than average incidence. Children with motor and/or cognitive delays were significantly slower than other children in the development of speech perception skills after implantation. Etiologic factors did not have a statistically significant effect on speech perception outcome.

Conclusions: It is likely that central pathologic states account for a substantial part of the variance among children using cochlear implants. Specific indicators of central pathologic states should be used to assess a child's prognosis in preference to less specific information based on etiologic factors alone.

It is well known that speech perception results with cochlear implants vary over a very wide range in both children and adults (1,2). Duration of deafness, duration of implant experience, age at onset of deafness, age at implantation, amount of residual hearing preoperatively, number of electrodes in use, and educational setting have been reported to have statistically significant effects, accounting for <=50% of the variance in large populations (1–4). Some studies have found relatively small but statistically significant differences between the mean scores of groups of patients with differing causes of deafness. For example, Battmer et al. (5) found a difference between patients deafened by meningitis and others. A similar result was reported in a multifactorial study by Blamey et al. (2). The pathologic changes in deafness may be either peripheral (in the cochlea) or central (in the brainstem or the brain) or may be both. To our knowledge, no study to date has attempted to relate the effects of etiologic factors on cochlear implant perception scores to the potential site(s) of the pathologic condition (central or peripheral). On the basis of the known pathologic course of the disorders

affecting the hearing of children, it may be possible to test this thesis. For instance, the rubella embryopathy, cytomegalovirus embryopathy, the combination of prematurity with hypoxia and jaundice, certain syndromes such as CHARGE-associated disorders, and bacterial meningitis are all associated with mental retardation in some cases (6).

We hypothesize that central pathologic conditions and/or processing disorders associated with some etiologic factors may contribute to the unexplained variance in studies of implant patients' speech perception. Clearly, the most objective evaluation of this hypothesis would involve a prospective study of the long-term results in a group of children whose candidacy for implantation was assessed without consideration of the etiologic and pathologic factors to be assessed. Given the time scale and ethical considerations involved in a prospective study, we have elected to carry out a retrospective pilot study as a first step. In this study, we analyze the results in 75 children who underwent implantation in The University of Melbourne/Royal Victorian Eye and Ear Hospital Cochlear Implant Clinic before May 1998, grouped by cause and classified according to evidence of motor and/or cognitive delays recorded in their preoperative medical histories.

METHODS

Population under Study

In this retrospective review, 75 consecutive children (<=5 years of age) who underwent implantation with the Cochlear Limited (Sydney, Australia) 22-electrode cochlear implant at the Royal Victorian Eye and Ear Hospital, Melbourne, between January 1985 and May 1998 were studied. Two patients who were followed up at other institutions were excluded from the study. Children who received implants after age 5 were not included in the study to reduce the effect of a possible loss of plasticity in the brain's ability to listen, process, and recognize sound in older children. Information for each patient was obtained from the preoperative medical, audiology, and speech pathology records. When a patient had been referred for developmental evaluation by a psychologist, this source of information was also used. The data collected included basic demographic data, cause of deafness, motor and cognitive developmental status, and speech perception.

Etiologic factors

The medical record was used to determine the cause of deafness when it was clearly documented and had been determined without ambiguity. If the cause was classified as “unknown, most likely other cause,” this “other cause” was used only when the medical record implied that it was the only identifiable etiologic factor and that it had directly preceded the deafness, e.g., history of maternal cytomegalovirus infection during pregnancy. If this was not clearly defined, the cause was classified as unknown.

Milestones

The detailed history from parents and the thorough examinations performed separately by the attending consultant physician and the audiologist/speech therapist were used to determine whether there was cognitive or motor delay immediately preceding cochlear implantation. The patients' cognitive and motor milestones were classified as either normal or delayed, determined by the guidelines set out by Illingworth (7).

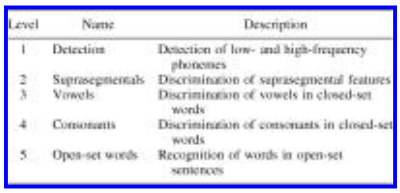
Motor milestones were classified as delayed only if the medical record clearly stated that gross or fine motor function was impaired for that patient's age or if the patient had received such a diagnosis after a formal developmental assessment by a child psychologist. Patients in whom meningitis was the cause were monitored for at least 1 year before cochlear implantation. Transient gait or other motor disturbances, frequently seen in postmeningitic patients, were classified as normal.

Cognitive milestone delay was determined by similar methods with attention to social behavior, interaction with environment and other children, and personality development. Speech and verbal communication development per se were not used as markers of cognitive development. Whereas differences in social, religious, and cultural backgrounds were accounted for, patients were deemed to have delayed cognitive milestones only when there was clear evidence for such a delay on clinical examination by the attending consultant physician. Although it was not standard practice for every child in the clinic, 11 of the 20 children classified as delayed in this study were referred to a child psychologist or neurologist. In every case, the psychologist or neurologist confirmed the physician's opinion that the child was significantly delayed.

Speech Perception

All patients were followed up weekly in the initial period following implantation. Later evaluations occurred at 6-monthly or 12-monthly intervals, depending on the age of the child and his or her individual clinical requirements. The primary outcome measures were improvement in speech perception and time to improvement. No single speech

perception assessment was appropriate for all patients because of the large range in age, speech production ability, language, and overall development among the study group members. For this reason, a classification of performance levels similar to that of Dowell et al. (3) was used. In this system, five levels define ascending degrees of complexity of speech perception (Table 1). The time from date of implantation until the attainment of each level was measured in months for each patient. The preimplant level and highest level reached were also noted.



Level	Name	Description
1	Detection	Detection of low- and high-frequency phonemes
2	Suprasegmentals	Discrimination of suprasegmental features
3	Vowels	Discrimination of vowels in closed-set words
4	Consonants	Discrimination of consonants in closed-set words
5	Open-set words	Recognition of words in open-set sentences

TABLE 1. *Speech perception levels*

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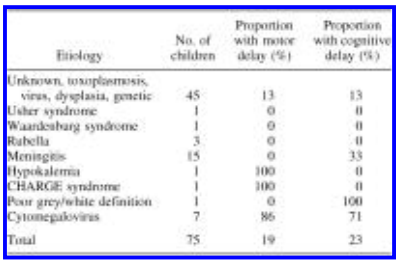
Statistical Analysis

Information for each patient was recorded in a Microsoft Excel spreadsheet, and statistical analysis was performed with Minitab Release 10.51 Xtra. Chi-square analysis was used to investigate the relationships between cause and milestones. A generalized linear model analysis was used to investigate the relationships between the speech perception categories, as dependent variable, and the independent variables: time after implant, and milestones or cause.

RESULTS

The ages at implantation ranged from 1 year 5 months to 5 years 9 months (mean 3.2 years, SD 1.2 years). The duration of implant experience ranged from 5 months to 12 years (mean 4.1 years, SD 2.8 years). From 1985 to 1998, speech processing strategies for the Cochlear Limited implant evolved from F0F1F2 through MPEAK to the SPEAK strategy, with successive improvements in speech perception for the population of postlinguistically deafened adults (8). Similarly, speech processing changes will have influenced the speech perception measures reported here, but will have approximately equivalent effects across different groups of children classified according to cause and milestones.

Table 2 shows the numbers of children classified by cause. The third and fourth columns of the table show the percentages of children with delayed motor and cognitive milestones for each etiologic group. A chi-square analysis of the data in Table 2 was conducted to determine whether there was a significant association between cause and delayed milestones. Because of the small numbers of patients, the groups for Usher syndrome, Waardenburg syndrome, rubella, hypokalemia, CHARGE syndrome, and poor gray/white matter differentiation were combined into a single group. The analysis indicated a highly significant association between cause and the incidence of delays (chi-square = 17.4, $df = 3$, $p < 0.001$). Cytomegalovirus was associated with a significantly higher incidence of identified motor and cognitive delays than the other causes combined (chi-square = 13.8, $df = 1$, $p < 0.001$). A high incidence of disabilities additional to deafness in children affected by congenital cytomegalovirus has previously been reported by Schildroth (6) and others. The high proportions of delays for hypokalemia, CHARGE syndrome, and undifferentiated gray/white matter are not statistically significant for this population because of the small number of subjects involved. The meningitis group showed no significant difference with respect to delayed milestones compared with all other causes (excluding cytomegalovirus).



Etiology	No. of children	Proportion with motor delay (%)	Proportion with cognitive delay (%)
Unknown, toxoplasmosis, virus, dysplasia, genetic	45	13	13
Usher syndrome	1	0	0
Waardenburg syndrome	1	0	0
Rubella	3	0	0
Meningitis	15	0	33
Hypokalemia	1	100	0
CHARGE syndrome	1	100	0
Poor grey/white definition	1	0	100
Cytomegalovirus	7	86	71
Total	75	19	23

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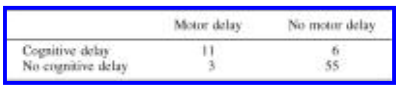
TABLE 2. *Classification of children by etiology and motor and cognitive delay*

Table 3 is a contingency table relating the incidence of motor and cognitive delays. The chi-square value for this table is highly significant (chi-square = 30.7, *df* = 1, p < 0.001). This indicates, as expected, that the two classifications are not independent. In other words, children who show evidence of cognitive delay are also likely to show evidence of motor delay, and vice versa. In view of this, the children were divided into two groups for further analysis. Group A consisted of children who showed evidence of neither type of delay. Group B consisted of children who showed evidence of motor delay or cognitive delay or both.



	Motor delay	No motor delay
Cognitive delay	11	6
No cognitive delay	3	55

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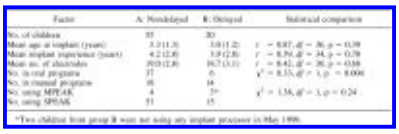
TABLE 3. *Crossclassification of children by motor and cognitive delay*

In considering the relative speech perception levels for groups A and B, it is prudent to check for significant differences between the groups on other variables that may have prognostic value. Dowell et al. (1,3) and Sarant (9) found significant effects for age at implantation (which is equivalent to duration of deafness in congenitally deaf children), duration of experience with the implant, educational setting (oral/aural or total communication or sign), and the processing strategy used in the evaluation (MPEAK or SPEAK 8). Blamey et al. (10) found a significant effect for the number of electrodes in use by adult implant users. Table 4 summarizes the values of these variables for the delayed and nondelayed groups of children in this study. The proportion of children in oral/aural rather than total communication and other manual communication educational settings was the only one of these variables that was significantly different for groups A and B. This variable is problematic in many studies of this nature because the choice of educational placement may be influenced by the level of speech perception achieved by the child. In other words, there is no way of knowing, from these data, whether the educational placement was a factor in the speech perception level achieved by the child, or vice versa.



Factor	A: Nondelayed	B: Delayed	Statistical comparison
No. of children	35	20	
Mean age at implant (years)	3.2 (1.3)	3.0 (1.2)	$t = 0.87$, $df = 36$, $p = 0.39$
Mean implant experience (years)	4.2 (2.8)	3.9 (2.8)	$t = 0.39$, $df = 34$, $p = 0.70$
Mean no. of electrodes	20.0 (2.8)	19.7 (3.1)	$t = 0.42$, $df = 36$, $p = 0.68$
No. in oral programs	27	6	$\chi^2 = 8.15$, $df = 1$, $p = 0.004$
No. in manual programs	8	14	
No. using MPEAK	4	10	$\chi^2 = 1.38$, $df = 1$, $p = 0.24$
No. using SPEAK	31	15	

*Two children from group B were not using any implant processor in May 1998.

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TABLE 4. *Comparison of groups A and B for factors known to affect speech perception scores in children and adults using cochlear implants**Two children from group B were not using any implant processor in May 1998.

Figures 1, 2, and 3 show the development of speech perception levels over time postoperatively for the two groups of children. The differences are immediately obvious from the graphs. Group A showed higher proportions of children reaching each speech perception level at each time postoperatively. An alternative way of looking at this is to say that the mean speech perception level reached by children in group A was greater than for those in group B at each time after implant (see Fig. 3). This observation

was confirmed statistically by use of a general linear model analysis, group (A or B) being used as a categorical factor and time postoperatively as a covariate. The dependent variable was the highest speech perception level attained by each child. The general linear model analysis indicated a significant difference between the two groups ($p < 0.001$, mean speech perception level of 4.2 for group A and 3.3 for group B), and the fitted slope of speech perception level versus time postoperatively was 0.60 levels per year. The slope was significantly greater than zero ($p < 0.001$). The proportion of variance accounted for in the analysis was 39%.

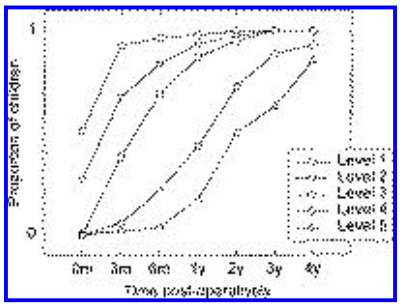

FIG. 1. Proportions of nondelayed children who had attained each speech perception level as a function of time postoperatively. Note that the time scale has been expanded nonlinearly for the first year to show details clearly. The numbers of children in the group at each time point from 0 months to 4 years were 55, 55, 55, 46, 40, 35, and 28, respectively.

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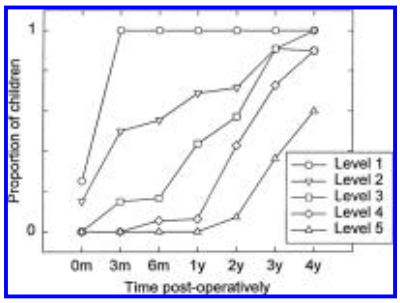

FIG. 2. Proportions of delayed children who had attained each speech perception level as a function of time postoperatively. The numbers of children in the group at each time point from 0 months to 4 years were 20, 20, 18, 16, 14, 11, and 10, respectively.

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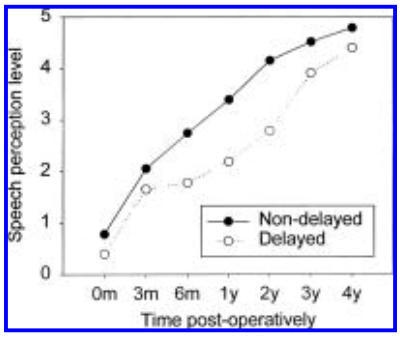

FIG. 3. Mean speech perception levels reached by the nondelayed and delayed groups of children as a function of time postoperatively. The numbers of children in each group at each time point are given in the captions for Figures 1 and 2.

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A similar generalized linear model analysis with cause as a categorical factor instead of the delayed milestones factor indicated no significant effect of cause ($p > 0.4$),

although postoperative time was still highly significant.

CONCLUSIONS

As one might expect, children who show evidence of motor and/or cognitive delays before cochlear implantation tend to progress more slowly than other children through the speech perception levels. They also seem to start at lower levels than other children, and some may never reach level 5, the ability to recognize a useful amount of open-set speech materials without lipreading. They may, however, obtain some useful benefits from cochlear implantation, including the ability to recognize words from a closed set without lipreading, and improved open-set speech perception with the aid of lipreading.

This result has obvious consequences for the candidacy of children for cochlear implantation and for the counseling of parents and children before and after cochlear implantation. Practice seems to vary between clinics. Some take the line that children with motor and/or cognitive delays should not receive implants because they are unlikely to perform well. By contrast, some clinics offer cochlear implants to profoundly deaf children with delays on the grounds that they will derive some benefit and there is improved speech perception over time, albeit slower than in other children. The sharing of information such as that obtained in this study may make these decisions easier for both the families and the clinicians involved. As pointed out in the discussion, the educational placement of the children in this study was significantly different postoperatively for the delayed and nondelayed groups. This difference may be the result of decisions made by the family after considerations of the lower level and slow development of speech perception (without the use of manual supplements) in the delayed group of children. Alternatively, the availability of signed information may have slowed the rate of development of oral/aural speech perception skills in some children.

Except for cytomegalovirus, cause of deafness was not a good indicator of motor or cognitive delay. Other more direct indicators should be searched for and used when a child's candidacy for cochlear implantation is being considered. The lack of a strong association between cause of deafness and delayed milestones may help to explain the relatively small influence of cause on speech perception measures in this and previous studies. In other words, the incidence and seriousness of central pathologic conditions can vary widely between individual children with the same cause, thus giving rise to a wide variation in outcomes between children with the same cause.

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