

Table 1. Comparison of histological study and MRI study

	CN	SON	LL	IC
Histological study				
Yakovlev and Lecours		23–24th fw (last of 5th fetal month)		
Rorke and Riggs		<40th fw (before mature born)		
Moore		26th and 29th fw		
MRI study				
Martin (2.35 T)	–	–	–	<40th fw (T2)
Counsell (1.0 T)	–	–	26th fw (T1)	25th fw (T2)
Our results (1.5 T)	37th fw (T2)	37th fw (T2)	37th fw (T1)	38th fw (T2)

fw: fetal weeks; CN: cochlear nucleus; SON: superior olivary nucleus; LL: lateral lemniscus; IC: inferior colliculus; T: Tesla.

brainstem auditory nuclei and pathway and why MRI reflects the myelination, past histological research was consulted. Rorke and Riggs [3] presented in their book a photograph of the sharp contrast of myelinating tissue of the cochlear nucleus, superior olivary nucleus, lateral lemniscus, and inferior colliculus with non-myelinating surrounding tissue. Rorke's staining dye has an affinity for lipids of the myelin sheath. T1- and T2-weighted images also reflect the change in the lipid content in the developing myelin sheath. Moore et al. [4] also presented the histology of myelination on the brainstem auditory nuclei and pathway; by 29 fetal weeks, the ventral cochlear nucleus was filled with myelinated axons and the superior olivary nucleus was also visible because of a pervasive fine-fiber myelinated neuropil. Magnetic resonance imaging is considered to detect this myelinated axon congregation and change the signal intensity for a reason that is explained below.

The myelin sheath is composed of multiple layers that are wound radially around the long axis of an axon. Analysis by radiographic diffraction and polarized microscopy has shown that the layers have a characteristic structure: protein–lipid–protein–lipid–protein [33,34]. The changes in signal intensity associated with myelination on T1- and T2-weighted images are due to changes in the lipid and water contents of developing myelin [35]. Myelination is probably shown as high signal intensity on T1-weighted images because of T1 shortening caused by an increase in cholesterol and glycolipids in the myelin sheath. The hypointense appearance of myelination on T2-weighted images corresponds to the time of tightening of myelin around the axon and the saturation of polyunsaturated fatty acids within the myelin membrane [35]. The reduction in signal intensity on T2-weighted images is probably due to a reduction in the number of aqueous protons due to the development of the hydrophobic inner phospholipids layer [35]. Other maturational changes, such as glial cell multiplication, increases in synaptic density, and dendrite formation, occur at

the same time as myelination in gray matter nuclei and may reduce the amount of free water at these sites, thereby contributing to the hypointense signal on T2-weighted images [35] (which may be the reason why T2-weighted imaging is more suitable for evaluation of gray matter nuclei). Blurring phenomenon of MRI [13] can be explained from the Moore histological report [4] that compared adult material with fetus material regarding the superior olivary complex; in adult material, myelinated axons fill the surrounding reticular formation and the superior olivary complex becomes less prominent than in the fetal period.

Our MRI study, the cochlear nucleus and the superior olivary nucleus showed myelinational signal change by the time of –3 CPW (37th fetal week) and blurring occurred at least 13 CPW on T2-weighted imaging. The lateral lemniscus, which is the white matter tract, showed myelinational signal change at –3 CPW (37th fetal week) and blurring occurred by at least 8 CPW on T1-weighted imaging. On T2-weighted imaging, myelinational signal change occurred at –1 CPW and blurring occurred by at least 13 CPW. In the inferior colliculus, myelinational signal changes were observed at –2 CPW (38th fetal week) and blurring occurred by at least 39 CPW. Magnetic resonance imaging can detect the myelinational signal intensity change; however, subsequently, blurring occurs and myelination can no longer be detected. In these results, it turned out that it is much later, about 3.5 months (11–18 weeks) than histological research previously demonstrated, that MRI shows signal intensity change. A proposed explanation for this difference is that myelination does not take place suddenly but happens gradually, and after definite myelination with full change of myelin sheath ingredients (loss of water and gain of lipids), it takes a minimal concentration of myelin to have a significant effect on the signal intensity detectable by MRI.

In radiological studies, numerous authors [13,15,19–22,28,36–38] have documented central nervous system changes on MRI corresponding to

myelination in the developing neonate and infant. Although most of these radiological reports did not mention the brainstem auditory nuclei and pathway, a few papers about MRI study mentioned about brainstem auditory pathway. Only lateral lemniscus and inferior colliculus which is a part of the auditory pathway were mentioned by Martin et al. [21] and Counsell et al. [35] (Table 1). According to Martin's study inferior colliculus myelinated before 40 fetal weeks, and Counsell mentioned the lateral lemniscus is myelinated at 26 fetal weeks (T1-weighted) and 27 fetal weeks (T2-weighted) and the inferior colliculus is myelinated at 25 fetal weeks (T2-weighted).

Our results and Martin's study showed solidarity. On the other hand, Counsell's study showed earlier myelination period than our study or Martin's study, and reflects more closely previous histological study results. However, Serena's study is performed by 1.0 T MRI which is not so high-resolution than Martin's study (2.35 T) or our study (1.5 T), in addition, imaging slices were used for visual evaluation which is not so objective method, so it was considered difficult to evaluate correctly the small structure of very early brain (their subjects were 26 preterm infants with a median fetal age of 28 weeks). The reason of time lag between Serena's study and Martin's study or our study is considered in performance of the MRI unit or in methodology of evaluation (visual evaluation and ROI analysis).

4.3. The difference of T1- and T2-weighted imaging

Our study shows that in the cochlear nucleus, superior olivary nucleus, and inferior colliculus, myelinational intensity change did not show a significant difference in T1-weighted images and myelinational intensity change was identified only in T2-weighted images, although in the lateral lemniscus, T1-weighted images showed the change of intensity earlier than in T2-weighted imaging. T2-weighted sequences were superior to T1-weighted sequences in demonstrating the contrast between gray matter nucleus and surrounding white matter and were therefore more suitable for evaluating gray matter nucleus [35]. This might be explained by the fact that in a high-field-strength system, the difference in the values of the T1 relaxation times of gray matter nucleus and white matter is not sufficiently large [35]. T2-weighted MRI was superior at showing myelin in deep gray matter nuclei and T1-weighted MRI was better at showing myelin in white matter tracts. This could be due to the characteristics of the

anatomic area; other MRI studies dealing with brainstem regions also confirm this observation [34,35]. These results suggest that partially modified protocols may be useful for assessing myelination in the brainstem.

4.4. Myelination progress from other aspects

We have also taken into account physiological study results to consider the myelination progress of the brainstem auditory nuclei and pathway. In the visual system, the duration of functional maturation (spatiotemporal vision) correlates with the duration of myelination of the optic radiation [33,39,40]. Moore et al. [4] mentioned that the time of onset of myelination coincides with the onset of acousticomotor reflexes or auditory startle reaction. Auditory function is also considered to correlate with myelination of the brainstem auditory nuclei and pathway. The myelin sheath surrounding an axon is composed of multiple segments of myelin [34]. Each segment is a modified plasma membrane that originates as an extension of an oligodendroglial cell process [34]. Small segments of bare axon are situated between the myelin sections, exposed to the interstitial space. These segments are called nodes of Ranvier. When the axonal membrane receives an action potential, the electrical impulse is unable to propagate through the high-resistance myelin sheath; therefore, the impulse "jumps" to the next node, which might be 1 mm or farther away [34]. Because of the low capacitance of the sheath, the remaining axonal membrane between the nodes depolarizes with little energy requirement and markedly increased speed. The increasing speed of fiber conduction involving the auditory brainstem nuclei and pathway can be measured by auditory brainstem response (ABR). Many researchers [5-9] have described a decrease in peak and interpeak latencies of ABR in early infancy and attributed the phenomenon to myelination of the brainstem auditory nuclei and pathway. The ABR wave I comes from the distal end of the auditory nerve, the generation of wave II is from the cochlear nucleus, wave III involves the superior olivary nucleus, wave IV is from the lateral lemniscus, and wave V is from the inferior colliculus. These ABR studies indicate that the speed of axonal conduction is low in children under 1 year of age and rapid axonal conduction gradually develops in the brainstem auditory nuclei and pathways. The increase of myelin density is likely to be a factor in the steady decrease in ABR interval between wave I and waves III-V.

5. Conclusion

Regarding the brainstem auditory nuclei and pathway, 1.5 T MRI revealed the signal intensity change by myelination at an average of 3.5 months (11–18 weeks) later than those reported in the histological literature. This time lag suggests that apart from histological research (comparing with histological work is not correct when evaluate the maturation of central auditory pathway using MRI because of time lag as shown in this study), the necessity for the new milestones of brainstem auditory pathway maturation using MRI is suggested. We suggest the new milestone of MRI used evaluation for brainstem auditory pathway in this study.

Myelination does not take place suddenly but happens gradually, so definite myelination, with a full change of myelin sheath ingredients (loss of water and gain of lipids), is needed to be detectable by MRI. This study shows the progress pattern of myelination in the brainstem auditory nuclei and pathway on MRI. These results can be used to assess with MRI the auditory system maturation of infants.

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References

- [1] P. Flechsig, *Anatomie des Menschlichen Gehirn und Rueckenmarks auf Myelogenetischer Grundlage*, Thime, Leipzig, 1920.
- [2] P.I. Yakovlev, A. Lecours, The myelogenetic cycles of regional maturation of the brain, in: Minkowski (Ed.), *Regional Development of the Brain in Early Life*, Blackwell Scientific Publication, Oxford, 1967, pp. 3–70.
- [3] L.B. Rorke, H.E. Riggs, *Myelination of the Brain in the Newborn*, Lippincott Company, Philadelphia, 1969.
- [4] J.K. Moore, L.M. Perazzo, A. Braun, Time course of axonal myelination in the human brainstem auditory pathway, *Hear Res.* 87 (1/2) (1995) 21–31.
- [5] C.W. Ponton, J.J. Eggermont, S.G. Coupland, R. Winkelaar, The relation between head size and auditory brainstem response interpeak latency maturation, *J. Acoust. Soc. Am.* 94 (4) (1993) 2149–2158.
- [6] P.A. Despland, R. Galambos, The auditory brainstem response (ABR) is a useful diagnostic tool in the intensive care nursery, *Pediatr. Res.* 14 (2) (1980) 154–158.
- [7] A.G. Pettigrew, D.J. Henderson-Smart, D.A. Edwards, Evoked potentials and functional development of the auditory system, in: M. Rowe, L. Aitkin (Eds.), *Information Processing in Mammalian Auditory and Tactile Systems*, Alan R. Liss, Inc., New York, 1990, pp. 295–308.
- [8] K. Kaga, Y. Tanaka, Auditory brainstem response and behavioral audiometry, developmental correlates, *Arch. Otolaryngol.* 106 (1980) 564–567.
- [9] M. Inagaki, Y. Tomita, S. Takashima, K. Ohtani, G. Andoh, K. Takeshita, Functional and morphometrical maturation of the brainstem auditory pathway, *Brain Dev.* 9 (6) (1987) 597–601.
- [10] M. Brant-Zwadzki, D.R. Enzmann, Using computed tomography of the brain to correlate low white-matter attenuation with early gestational age in neonates, *Radiology* 139 (1981) 105–108.
- [11] R.D. Penn, B. Trinko, L. Baldwin, Brain maturation followed by computed tomography, *J. Comput. Assist. Tomogr.* 4 (1980) 614–616.
- [12] L. Picard, M. Claudon, J. Roland, E. Jeanjean, M. Andre, P. Plenat, et al., Cerebral computed tomography in premature infants, with an attempt at staging developmental features, *J. Comput. Assist. Tomogr.* 4 (1980) 435–444.
- [13] H. Nakagawa, S. Iwasaki, K. Kichikawa, A. Fukusumi, T. Taoka, H. Ohishi, et al., Normal myelination of anatomic nerve fiber bundles: MR analysis, *Am. J. Neuroradiol.* 19 (1998) 1129–1136.
- [14] M.A. Johnson, J.M. Pennock, G.M. Bydder, Clinical NMR imaging of the brain in children: normal and neurologic disease, *Am. J. Roentgenol.* 141 (1983) 1005–1018.
- [15] B.A. Holland, D.K. Haas, D. Norman, M. Brant-Zwadzki, T.H. Newton, MRI normal brain maturation, *Am. J. Neuroradiol.* 7 (1986) 201–208.
- [16] R.B. Dietrich, W.G. Bradley Jr., MR evaluation of early myelination patterns in normal and developmentally delayed infants, *Am. J. Neuroradiol.* 9 (1998) 69–76.
- [17] C.B. McArdle, C.J. Richardson, D.A. Nicholas, M. Mirfakhraee, C.K. Hayden, E.G. Amparo, Developmental features of the neonatal brain: MR imaging, gray-white matter differentiation and myelination, *Radiology* 162 (1987) 223–229.
- [18] M.S. van der Knaap, J. Valk, MR imaging of the various stage of normal myelination during the first year of life, *Neuroradiology* 31 (1990) 459–470.
- [19] K. Hayakawa, Y. Konishi, M. Kuriyama, K. Konishi, T. Matsuda, Normal brain maturation in MRI, *Eur. J. Radiol.* 12 (1990) 208–215.
- [20] E. Martin, R. Kikinis, M. Zuerrer, C. Boesch, J. Briner, G. Kewitz, et al., Development stages of human brain: an MR study, *J. Comput. Assist. Tomogr.* 12 (1988) 917–922.
- [21] E. Martin, S. Krassnitzer, P. Kaelin, MR imaging of the brainstem: normal postnatal development, *Neuroradiology* 33 (1991) 391–395.
- [22] A.J. Barkovich, B.O. Kjos, D.E. Jackson Jr., D. Norman, Normal maturation of the neonatal and infant brain: MR imaging at 1.5 T, *Radiology* 166 (1988) 173–180.
- [23] C.R. Bird, M. Hedberg, B.P. Drayer, P.J. Keller, R.A. Flom, J.A. Hodak, MR assessment of myelination in infants and children: usefulness of marker sites, *Am. J. Neuroradiol.* 10 (1989) 731–740.
- [24] J.A. Stone, D.W. Chakeres, P. Schmalbrock, High-resolution MR imaging of the auditory pathway, *Magn. Reson. Imag. Clin. N. Am.* 6 (1) (1998) 195–217.
- [25] D. Bergerbest, D.G. Ghahremani, D.E. Gabrieli, Neural correlates of auditory repetition priming: reduced fMRI activation in the auditory cortex, *J. Cogn. Neurosci.* 16 (2004) 966–977.
- [26] K. Hittmair, D. Wimberger, T. Rand, L. Prayer, G. Bernert, J. Kramer, et al., MR assessment of brain maturation: comparison of sequences, *Am. J. Neuroradiol.* 15 (1994) 425–433.

- [27] B.D. Flannigan, W.G. Bradley Jr., J.C. Mazziotta, W. Rauschnig, J.R. Bentson, R.B. Lufkin, G.B. Hieshima, Magnetic resonance imaging of the brainstem: normal structure and basic functional anatomy, *Radiology* 154 (2) (1985) 375–383.
- [28] J.T. Curnes, P.C. Burger, W.T. Djang, O.B. Boyko, MR imaging of compact white matter pathways, *Am. J. Neuroradiol.* 9 (6) (1988) 1061–1068.
- [29] A. Fee-Higgins, J.C. Larroche, *Development of the Human Foetal Brain*, INSERM Masson, Paris, 1987.
- [30] Gaining and growing: calculating corrected age. University of Washington's homepage, <http://depts.washington.edu/growing/Assess/Grca.html>.
- [31] B.A. Brody, H.C. Kinney, A.S. Kloman, F.H. Gilles, Sequence of central nervous system myelination in human infancy, an autopsy study of myelination, *J. Neuropathol. Exp. Neurol.* 46 (1987) 283–301.
- [32] M. Damska, M. Laure-Kaminowska, Myelination as a parameter of normal and retarded brain maturation, *Brain Dev.* 12 (1990) 214–220.
- [33] D.A. Kirschner, A.E. Blaurock, Organization, phylogenetic variations, and dynamic transitions of myelin, in: R.E. Martenson (Ed.), *Myelin: Biology and Chemistry*, CRC Press, Boca Raton, FL, 1991, pp. 413–448.
- [34] A.J. Barkovich, Magnetic resonance techniques in the assessment of myelin and myelination, *J. Inher. Metab. Dis.* 28 (3) (2005) 311–343.
- [35] S.J. Counsell, E.F. Maalouf, A.M. Fletcher, P. Duggan, M. Battin, H.J. Lewis, et al., MR imaging assessment of myelination in the very preterm brain, *Am. J. Neuroradiol.* 23 (5) (2002) 872–881.
- [36] T. Stricker, E. Martin, C. Boesch, Development of the human cerebellum observed with high-field-strength MR imaging, *Radiology* 177 (2) (1990) 431–435.
- [37] W. Grodd, Normal and abnormal patterns of myelin development of the fetal and infantile human brain using magnetic resonance imaging, *Curr. Opin. Neurol. Neurosurg.* 6 (3) (1993) 393–397.
- [38] M. Staudt, I. Krageloh-Mann, W. Grodd, Normal myelination in childhood brains using MRI—a meta analysis, *Rofo* 172 (10) (2000) 802–811.
- [39] H.C. Kinney, B.A. Brody, A.S. Kloman, F.H. Gilles, Sequence of central nervous system myelination in human infancy, patterns of myelination in autopsied infants, *J. Neuropathol. Exp. Neurol.* 47 (1988) 217–234.
- [40] H.R. Wilson, Development of spatiotemporal mechanisms in infant vision, *Vis. Res.* 28 (1988) 611–628.

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聋婴幼儿助听后的认知、交流及言语发育

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【摘要】 目的 探索聋婴幼儿助听后的认知、交流与言语发育的特征。方法 将确诊为先天性重度聋的婴幼儿 39 例分为 A 组(20 例,8 个月以前开始助听)和 B 组(19 例,8 个月以后开始助听)。通过数字摄像机对受试儿童的行动、交流方式及发声进行录音和录像。对两组适应助听器和注视交流成立所需的时间,手指发声及其他言语发育的出现时间等作了统计学分析,并对两组的发声进行了声谱分析。结果 适应助听器所需时间、注视交流成立所需时间和手指发声出现时间:A 组分别为(0.5±0.2)个月,(0.6±0.2)个月和(12.1±2.1)个月;B 组分别为:(2.3±0.5)个月,(2.2±0.3)个月和(16.1±4.5)个月;各项成绩 A 组优于 B 组,两组间的差异均有统计学意义。从言语发育分析,两组间过渡喃语出现时间的差异无统计学意义,标准喃语和有意义语出现时间的差异有统计学意义。结论 早期助听具有适应助听器时间短、易于形成注视交流、手指发声出现早和言语发育良好等特点;提示早期和有效的助听,是激发认知发育和促进言语发育的前提。

【关键词】 聋; 助听器; 儿童发育; 交流障碍; 言语声学

Development of cognition, communication, and vocalization in hearing impaired infants fitted with hearing aids HUANG Li-hui*, HAN De-min, WANG Tao, KAGA Kimi-taka. *Beijing Institute of Otolaryngology, Beijing Tongren Hospital, Capital University of Medical Sciences, Beijing 100005, China

【Abstract】 **Objective** To study the characteristics of development of cognition, communication, and vocalization in congenitally hearing impaired infants with hearing aids. **Methods** Hearing aids were fitted to 39 congenitally hearing impaired infants: 20 of them were fitted with the hearing aids before the age of 8 months (Group A), and 19 of them after the age of 8 months (Group B). Digital video camera was used to record the action, communication, and vocalization of the children. **Results** The time needed to be adaptable to the hearing aids of Group A was(0.5±0.2) months, significantly shorter than that of Group B [(2.2±0.3) months, $P < 0.01$]. The time needed to establish communication by eyes was (0.6±0.2) months in Group A, significantly shorter than that in Group B [(2.3±0.5) months, $P < 0.01$]. The time needed to present canonical babbling, communication by pointing behaviors, and meaningful words of Group A were (15.0±1.8) months, (12.1±2.1) months, and (17.3±2.2) months respectively, all significantly shorter than those of Group B [(23.2±8.0) months, (16.1±4.5) months, and (32.6±10.9) months respectively, all $P < 0.05$]. However, the time needed to present pre-canonical babbling of Group A was (4.3±0.5) months, not significantly different from that of Group B [(4.8±0.6) months, $P > 0.05$]. **Conclusion** Using the hearing aids early and validly helps acquire good development of cognizance and speech in hearing impaired infants.

【Key words】 Deafness; Hearing aids; Child development; Communication disorders; Speech acoustics

认知,是人获得知识和使用知识的过程。瑞士著名儿童心理学家和认知论开创者 Piaget^[1,2]将儿童认知发育分为四个阶段:感觉运动阶段(0~2岁)、前运演阶段(2~7岁)、具体运演阶段(7~11岁)和形式运演阶段(11~15岁)。他强调,正常

儿童在言语尚未发生的感觉运动阶段,可以进行各种认知运演活动,这使得儿童具有丰富的发现。国内研究表明,正常儿童2岁以前(包括2岁)已经形成并掌握了基本的交流技能^[3],而心理理论发展和执行功能发展在聋童身上表现出较大的不一致性^[4]。本研究通过对不同助听年龄的先天性聋婴幼儿的行为、交流方式及发声变化等进行对比和分析,了解这些先天性聋童早期认知、交流和言语发育的特征,为正确和有效地进行聋儿康复指导提供科学的依据。

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对象与方法

一、对象

日本东京大学医学部耳鼻咽喉科交流障碍门诊 1999 年 4 月至 2004 年 2 月期间患儿 39 例,男 18 例,女 21 例,所有患儿均确诊为先天性双侧重度(或极重度)感音神经性聋,并经耳鼻咽喉科、小儿内科及神经生理学检查,除耳聋外无其他智力及发育障碍。智力发育检测采用美国 WPPSI (wechsler preschool and primary scale of intelligence) 智力评分方法。根据助听开始年龄的不同,将其分为 A 组(20 例,8 个月以前开始助听)和 B 组(19 例,8 个月以后开始助听)。

二、方法

1. 录音录像及声学分析:在门诊的隔音室,当婴幼儿和母亲游玩,或认字,或读卡片,或听力检查时,对其行为动作、交流方式和发声进行录像和录音。录音录像采用日本 SONY 公司生产的 CCD-TRV92 型数字摄像机,每 1~2 周记录一次。声谱分析采用美国 GM 公司生产的 Sound Scope 软件,分析条件:分析滤波带宽 150~300 Hz,频率 2048 Hz/div,分析时间 100~200 ms/div,声谱分析表横轴为时间(ms),纵轴为频率(Hz)。

2. 认知、交流和言语发育关联因子的相关定义:(1)适应助听器所需时间:正式佩戴助听器后,从佩戴到完全习惯所需的时间。(2)注视交流成立:听到声音时会寻找声源并注视对方,或被叫名字时,头转向呼叫者,或主动发出声音吸引对方注意而注视对方,每次注视时间 ≥ 3 s 者,定义为注视交流成立。(3)言语发育关联因子:按言语前期言语发育的标准,定义为过渡喃语、标准喃语、手指发声和有意语^[3]。

3. 统计学方法:根据本研究数据正态分布的不一致性,采用非参数统计方法。对 A 组和 B 组 2 组间适应助听器和注视交流成立所需的时间,以及手指发声的出现年龄等作了统计分析,并对言语发育关联因子的出现年龄作了比较,采用 Kruskal-Wallis 检测进行分析,用 Wilcoxon 法计算评分,统计软件采用 SAS 8.0 标准统计软件包。

结 果

平均听阈为 A 组(91 ± 10) dB HL, B 组(94 ± 10) dB HL,两组差异无统计学意义($P > 0.05$)。平均助听开始年龄:A 组(6.8 ± 1.2)个月, B 组

(20.9 ± 6.2)个月($P < 0.01$);A 组观察开始至终止年龄为 3~35 个月,观察开始平均年龄 4.5 个月,观察终止平均年龄 29 个月;B 组观察开始至终止年龄为 8~40 个月,观察开始平均年龄 15 个月,观察终止平均年龄 31 个月。

1. 认知和交流的比较:(1)适应助听器所需时间:A 组平均(0.5 ± 0.2)个月与 B 组平均(2.3 ± 0.5)个月的差异有统计学意义($P < 0.01$)。(2)注视交流成立所需时间:A 组平均(0.6 ± 0.2)个月与 B 组平均(2.2 ± 0.3)个月的差异有统计学意义($P < 0.01$,表 1)。

表 1 适应助听器与注视交流所需时间

组别	例数	时间(月)	统计量(Z)
A			
X1	20	0.5 ± 0.2	-2.91*
X2	20	0.6 ± 0.2	-2.93*
B			
X1	19	2.3 ± 0.5	3.32
X2	19	2.2 ± 0.3	3.01

注:Z 为近似正态统计量;X1 为适应助听器所需时间;X2 为注视交流成立所需时间;与 B 组比较,* $P < 0.01$

2. 言语发育关联因子的比较:A 组(4.3 ± 0.5)个月和 B 组间(4.8 ± 0.6)个月的过渡喃语差异无统计学意义($P > 0.05$);标准喃语[A 组(15.0 ± 1.8)个月, B 组(23.2 ± 8.0)个月],手指发声[A 组(12.1 ± 2.1)个月, B 组(16.1 ± 4.5)个月]及有意语[A 组(17.3 ± 2.2)个月, B 组(32.6 ± 10.9)个月]3 个方面的出现年龄, A 组与 B 组均有统计学意义(均 $P < 0.05$)。从言语发育的平均出现年龄看, A 组和 B 组间过渡喃语出现时间的差异较小;而标准喃语、手指发声和有意语的出现时间, A 组较 B 组出现早约 8、4 和 15 个月(表 2)。

3. 声谱分析的比较:A 组和 B 组配戴助听器的年龄不同,两组间声谱特征的差异较大。单纯就过渡喃语而言, A 组和 B 组的声调都偏高,但发声的特征无明显差异。从标准喃语和有意语的声谱分析, A 组发声音节清晰,而 B 组多为语调平板无抑扬,甚至呈浊音化。

讨 论

我国学者强调,婴幼儿和小龄儿童助听器验配是聋婴幼儿早期听力干预的重要环节^[5];诸多研究表明^[6-8],婴幼儿的早期听觉补偿较晚期听觉补偿,可以获得较好的言语发育。Piaget^[1]认为,儿童认知

表 2 A 组与 B 组言语发育的比较

组别	例数	时间(月)	统计量(Z)
A			
X1	20	4.3±0.5	-1.54
X2	18	15.0±1.8	-2.38 ^b
X3	17	12.1±2.1	-1.89 ^a
X4	12	17.3±2.2	-2.58 ^b
B			
X1	19	4.8±0.6	2.05
X2	12	23.2±8.0	3.23
X3	14	16.1±4.5	2.85
X4	13	32.6±10.9	3.61

注:Z 近似正态统计量;X1 为过渡喃语;X2 为标准喃语;X3 为手指发声;X4 为有意义语;与 B 组比较,^a $P < 0.05$,^b $P < 0.01$

发育各阶段是密切相关的,创造条件,促使儿童与外界相互作用,才能使认知结构不断成熟和发展。注视交流在我们的日常生活中常常反映一个人的心理活动,它是人与人交流中不可缺少的一个重要组成部分。进藤美津子等^[9]指出,言语发育前期婴幼儿的注视交流和发声行为是获得言语的基础。加我牧子强调^[10],手指发声是婴幼儿对外界的一种关心,是儿童想要认识人和新事物的一种表现;从言语发育的观点看,手指发声的出现是一个重要的里程碑。傍氏和香等^[11]对 2 例聋儿的手势交流和言语发育作了长期观察,指出手指发声与婴幼儿的言语发育密切相关。可见,儿童注视交流和手指发声的成立,对于其言语发育的获得是至关重要的。本研究结果表明,早期佩戴助听器的 A 组,注视交流成立所需的平均时间明显早于晚期佩戴助听器 B 组;手指发声出现时间, A 组成绩明显优于 B 组,两组间的差异具有统计学意义。以上提示,早期的听觉刺激和各种交流活动,可以激发大脑的整体认知功能,从而进一步促进言语发育。因此,为聋婴幼儿早期选配合适的助听器,创造条件使其与外界进行多方面交流,尽早激发大脑的认知发育,对于促进言语发育具有重要的意义。

Oller 等^[12-13]将正常婴幼儿的言语前期归纳为:发声期,原始调声期,扩张期(前三个时期又称为过渡喃语期)和标准喃语期四个阶段;并且认为在言语前期言语发育的过程中,标准喃语期是言语发育最重要的时期。前期研究提示^[6],过渡喃语是不受听觉反馈作用的一种发声活动;而标准喃语和有意义语,则明显受到听觉反馈作用的影响。本研究过渡喃语的出现年龄, A 组与 B 组之间的差异无统计学

意义,标准喃语和有意义语的出现年龄,两组间的差异均有统计学意义,说明这与前期研究的结果一致。另外,从声学分析看, A 组和 B 组过渡喃语的发声特征无明显差异。但在标准音节的形成、语调的变化、共振峰移行时间的缩短和共振峰的分化上看, A 组的成绩明显优于 B 组。以上结果充分说明,早期听觉补偿对于聋婴幼儿的早期言语发育具有积极的促进作用。

本研究对不同助听年龄聋婴幼儿助听后的认知、交流及言语发育进行了对比分析,发现早期助听的婴幼儿具有适应助听器时间短、易于形成注视交流,手指发声出现早和言语发育良好等特点,提示早期和有效的助听,可以激发认知发育和促进言语发育,而聋婴幼儿的认知及言语信号处理的机制等,将有待进一步深入研究和探讨。

参 考 文 献

- [1] Piaget J. Operational structures and cybernetics. *Annee Psychol*, 1953, 53: 379-388.
- [2] Piaget J. Language and thinking. *Rev Prat*, 1965, 15: 2253-2254.
- [3] 江敏红. 0-6 岁儿童非言语交流发展的初步研究. *中国特殊教育*, 2004, 47: 79-84.
- [4] 李一员, 吴睿明, 胡兴旺, 等. 聋童执行功能发展: 聋童与正常儿童的比较. *心理学报*, 2006, 38: 356-364.
- [5] 黄治物, 常伟, 吴展元, 等. 应做好早期听力康复的干预工作. *中华医学杂志*, 2003, 83: 270-271.
- [6] 黄丽辉, 加我君孝, 韩德民, 等. 正常与先天性重度聋婴幼儿言语前期言语发育的比较研究. *中华医学杂志*, 2005, 85: 765-768.
- [7] 黄麗輝, 加我君孝, 今泉敏, 他. 補助月齡の異なる先天性高度難聴児の前言語期における音声の発達について. *音声言語医学*, 2002, 43: 134-140.
- [8] Yoshinaga-Itano C, Sedey AL, Coulter DK, et al. Language of early- and later-identified children with hearing loss. *Pediatrics*, 1998, 102: 1161-1171.
- [9] 進藤美津子, 玉井ふみ, 山崎和子, 他. 前言語期における認知・コミュニケーション行動の発達評価チェック・リストの作成. *広島県立保健福祉短大紀要*, 1999, 4: 93-101.
- [10] 加我牧子. 小児言語障害のみかた. *小児内科*, 1988, 20: 1535-1543.
- [11] 傍氏和香, 森望, 大森千代美, 他. 難聴児の指さしについて. *Audiology Japan*, 2000, 43: 371-372.
- [12] Oller DK, Eilers RE, Neal AR, et al. Precursors to speech in infancy: the prediction of speech and language disorders. *J Commun Disord*, 1999, 32: 223-245.
- [13] Oller DK, Eilers RE, Neal AR, et al. Late onset canonical babbling: a possible early marker of abnormal development. *Am J Ment Retard*, 1998, 103: 249-263.

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