Available online at www.sciencedirect.com
SCIENCE DIRECT®
Hearing Research 204 (2005) 78–89
HEARING RESEARCH
www.elsevier.com/locate/heares

# Effects of cochlear implant use on the electrically evoked middle latency response in children

Karen A. Gordon [a,b,*], Blake C. Papsin [b], Robert V. Harrison [a,b]

[a] Department of Otolaryngology, The Hospital for Sick Children, University of Toronto, Toronto, Ont., Canada M5G 1X8
[b] Brain and Behaviour Program, The Hospital for Sick Children, University of Toronto, Toronto, Ont., Canada M5G 1X8

Received 16 February 2004; accepted 6 January 2005
Available online 8 February 2005

**Abstract**

The electrically evoked middle latency response (eMLR) reflects central auditory activity in cochlear implant users. This response was recorded repeatedly in 50 children over the first year of cochlear implant use and in 31 children with $5.3 \pm 2.9$ years of implant experience. The eMLR was rarely detected at the time of implantation in anaesthetized or sedated children and was detected in only 35% of awake children at initial device stimulation. The detectability of the eMLR increased over the first year of implant use becoming 100% detectable in children after at least one year. Acutely evoked responses were more likely to be present in older children despite longer periods of auditory deprivation. Within six months of implant use, most children had detectable eMLRs. At early stages of device use, eMLR amplitudes were lower in children implanted below the age of 5 years compared to children implanted at older ages; amplitudes increased over time in both groups. Latencies after six months of implant use were prolonged in the younger group and decreased with implant use. EMLR changes with chronic cochlear implant use suggest an activity-dependent plasticity of the central auditory system. Results suggest that the pattern of electrically evoked activity and development in the auditory thalamocortical pathways will be dependent upon the duration of auditory deprivation occurring in early childhood.


*Keywords:* Deafness; Congenital; Pre-lingual; Peri-lingual; Evoked potentials; Electrical stimulation; Sensitive periods; Auditory; Human; Children; Thalamus; Cortex; Synchrony; Development; Plasticity

## 1. Introduction

It is reasonable to assume that activation of the thalamocortical projections of the auditory pathways are compromised by severe to profound hearing loss. For children who experience deafness during early periods of development, the limited or lack of auditory input might disrupt activity-dependent developmental processes which normally take place in the central auditory system. Moreover, with ongoing duration of auditory deprivation, degenerative processes could occur along the auditory pathways and limit the potential for plasticity in the system. The present study provides evidence to test these suppositions. As our experimental tool, we have monitored the electrically evoked middle latency response (eMLR) potentials over time in children who use cochlear implants. The eMLR is known to have similar wave morphology and latencies as the acoustically evoked MLR (Kileny et al., 1989) which reflects activity in the auditory thalamus and cortex (Kraus and McGee, 1995).

### 1.1. Effects of auditory deprivation in the thalamus and cortex

Long term auditory deprivation experienced by prelingually deafened adults who communicate using sign

* Corresponding author. Tel.: +416 813 6683; fax: +416 813 5036.
*E-mail address:* karen.gordon@utoronto.ca (K.A. Gordon).

0378-5955/$ - see front matter © 2005 Elsevier B.V. All rights reserved.
doi:10.1016/j.heares.2005.01.003

language has been shown to result in cross-modal reorganization of the auditory cortex (Neville et al., 1997; Nishimura et al., 1999; Bavelier et al., 2001; Finney et al., 2001; Giraud et al., 2001). Auditory input delivered by a cochlear implant activates the auditory cortex (Ponton and Eggermont, 2001; Sharma et al., 2002a) (Miyamoto et al., 1999), but the activity is disrupted by interactions from visual input if reorganization has occurred during the period of deprivation (Nishimura et al., 2000). Evidence from Lee and colleagues (Lee et al., 2001) suggests that the degree of cross-modal plasticity indicated by glucose metabolism in the auditory cortex predicts the ability to hear speech with a cochlear implant; in that study, reduced hypometabolism correlated with poor speech perception skills post-implantation.

Deprivation or manipulation of auditory input does not disrupt afferent neural connections between the thalamus and cortex (Gao and Pallas, 1999; Stanton and Harrison, 2000; review: Pallas, 2001). Normal thalamocortical connections have been found in congenitally deaf cats which, in contrast, show deficits in infragranular layers of the cortex responsible for corticocortical and corticothalamical connections (Kral et al., 2000). Stanton and Harrison (2000) conclude that the stability of thalamocortical pathways in the presence of cortical reorganization implies that similar reorganization has occurred in the thalamus. This group has also shown that neonatal high frequency hearing loss results in abnormal frequency maps in the inferior colliculus (Harrison et al., 1993) and cortex (Harrison et al., 1991).

### *1.2. Plasticity in auditory thalamus and cortex*

Changes along the human auditory system evoked by cochlear implant use have been inferred by improvements in speech perception skills in both adults (e.g., Tyler et al., 1997a; Shipp et al., 1997; Staller et al., 1997) and children (e.g., Tyler et al., 1997b; Waltzman et al., 1997; Nikolopoulos et al., 1999a). However, children with pre or peri-lingual deafness who receive their cochlear implant at older ages achieve lower speech perception skills than their younger peers at similar durations of implant use (Osberger et al., 1991; Dawson et al., 1992; Fryauf-Bertschy et al., 1992; Tyler et al., 1997b; Nikolopoulos et al., 1999b). There may be a specific age at implantation before which speech perception outcomes of cochlear implantation are most promising. Significant differences have been found between two groups of children divided at ages 3 (Hassanzadeh et al., 2002; Kirk et al., 2002), 4 (Tyler et al., 1997b), and 5 (Papsin et al., 2000; Geers et al., 2002) years, however, the best "divider" age might change depending on the particular speech perception test used to measure outcome (El-Hakim et al., 2002). The fact that two groups of children divided at one age show significantly different outcomes implies that there are sensitive periods during development of the speech and language pathways, the central auditory system, or both.

Some evidence suggests that central auditory plasticity evoked by electrical stimulation in subjects with profound hearing loss is limited by sensitive periods. In congenitally deaf cats, chronic stimulation from a cochlear implant resulted in higher amplitude middle latency responses and the emergence of long latency responses (Klinke et al., 1999) but only if the cats were implanted by the age of 6 months (Kral et al., 2001). Evoked potential data suggest that the initial rate of cortical development with respect to time in sound (chronological age – duration of auditory deprivation) in children using cochlear implants is comparable to that of normal hearing children (Ponton et al., 1996). However, with longer durations of implant use, long latency potentials may not develop into adult-like responses reflecting a persistent immaturity or deficit in cortical activity (Ponton and Eggermont, 2001). Children implanted at ages below 3.5 years have been shown to have age-appropriate long latency responses after 6 months of implant use (Sharma et al., 2002b) although it is not yet known if these children received auditory input early enough to develop adult-like long latency responses.

Sensitive periods in the brainstem have not been found after stimulation with electrical pulses. Chronic stimulation from a cochlear implant promotes similar expansions of spatial tuning curves in the inferior colliculus of adult cats (Moore et al., 2002) as found in kittens (Snyder et al., 1990) and increased metabolic activity occurs after cochlear implantation in cats regardless of age at implant (Seldon et al., 1996). In children using cochlear implants, neural conduction time in the auditory brainstem decreases in response to chronic stimulation during the first year of implant use (Gordon et al., 2002). The rate of change in brainstem conduction is not related to children's age at implantation suggesting that the human auditory brainstem remains plastic throughout childhood despite early onset severe to profound hearing loss (Gordon et al., 2003).

The effects of auditory deprivation on the thalamus are more poorly understood than effects at the brainstem or cortical levels. In one report, thalamic plasticity occurred following unilateral manipulation of auditory input in young barn owls (Miller and Knudsen, 2003) and, in another, reorganization in the thalamus was indirectly shown in cats with neonatal high frequency hearing loss (Stanton and Harrison, 2000). There is no prior evidence that the human auditory pathways extending from the thalamus to cortex are impacted by a lack of auditory input during early development; moreover, the presence of a sensitive period in thalamocortical development of the auditory system remains unclear.

Activity from the auditory thalamus to cortex has been recorded in adults using cochlear implants using eMLRs (Shallop et al., 1990; Firszt et al., 2002) but,