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ScienceDirect
Hearing Research 228 (2007) 44–57
Hearing Research
www.elsevier.com/locate/heares

Research paper

Effects of auditory pathway anatomy and deafness characteristics? Part 2: On electrically evoked late auditory responses

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Received 13 September 2006; received in revised form 12 January 2007; accepted 12 January 2007
Available online 30 January 2007

Abstract

The purpose of this study was to distinguish the effects of different parameters on latencies of wave N1, wave P2, and inter-peak interval N1–P2 of electrical late auditory responses (ELARs). ELARs were recorded from four intra-cochlear electrodes in fourteen adult HiRes90K® cochlear implant users who had at least three months of experience. The relationship between latencies and stimulation sites in the cochlea was characterized to assess the influence of the auditory pathway anatomy on ELARs, i.e., whether the speed of neural propagation varies according to the place that is activated in the cochlea. Audiograms before implantation, duration of deafness, and psychophysics at first fitting were used to describe the influence of deafness characteristics on latencies. The stimulation sites were found to have no effect on ELAR latency and, while there was no influence of psychophysics on latency, a strong relationship was shown with duration of deafness and the pre-implantation audiogram. Thus, ELAR latency was longer for poorer audiograms and longer durations of deafness and this relationship appeared to be independent of stimulation parameters such as stimulation site. Comparison between these findings and those from the equivalent study on EABR waves IIIe and Ve latency [Guiraud, J., Gallego, S., Arnold, L., Boyle, P., Truy, E., Collet, L., 2007. Effects of auditory pathway anatomy and deafness characteristics? (1): On electrically evoked auditory brainstem responses. Hear. Res. 223 (1–2), 48–60] shows that, while ELAR and EABR latencies are related with parameters that reflect the integrity of the auditory pathway, ELAR latency is less dependent on stimulation parameters than EABR latency.

Keywords: ELAR latencies; Tonotopy; Cochlear implant; Deafness characteristics

1. Introduction

Cochlear implantation is a treatment option for adults and children with bilateral severe-to-profound sensorineural hearing loss who do not benefit from traditional amplification. A cochlear implant functions by bypassing the stimulation of the hair cells of the inner ear and rather activates the auditory nerve fibers directly, resulting in the transmission of impulses to the central auditory pathway (Abbas, 1993; Kiang and Moxon, 1972; Loeb et al., 1983). Most users demonstrate improved performance when compared with their pre-implant abilities, although

Abbreviations: ELAR, electrical late auditory response; EABR, electrical auditory brainstem response; EEP, electrically evoked potential; MMN, mismatch negativity; ANOVA, analysis of variance; M level, most comfortable level; dB HL, decibel (hearing level); CU, charge unit; CPI, clinician programming interface; PTA, pure tone audiometry

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doi:10.1016/j.heares.2007.01.022

there is wide variation in outcome. This variation may depend on parameters such as duration of implant use, age at implantation, speech production, lip-reading performance, intelligence quotient, motivation, and family background (Waltzman et al., 1995; Blamey et al., 1996). However, other factors that are linked to deafness may have even more influence on cochlear implant benefit. Such factors include, etiology (Brimacombe & Eisenberg, 1984), duration of deafness (Blamey et al., 1996), poor auditory nerve survival or atrophy of the central auditory nervous system (Hall, 1990; Pfingst et al., 1980; Jyung et al., 1989; Shepherd et al., 1983; Walsh & Leake-Jones, 1982; Kraus et al., 1993b, 1998; Micco et al., 1995; Oviatt and Kileny, 1991; Stypulkowski et al., 1986; Ponton et al., 1996; Blamey, 1997; Fayad et al., 1991; Pauler et al., 1986). Studies investigating the relationship between performance and evoked potentials yield controversial results (Makhdoum et al., 1998; Firszt et al., 2002b; Kelly et al., 2005), which may be linked to the fact that different protocols were used. It is indeed known that evoked potentials are dependent on the stimulus characteristics in a conflicting way (for a review: Näätanen and Picton, 1987). We propose to study the influence of some characteristics of deafness on the auditory pathway using electrically evoked potentials (EEPs) in response to electrical pulses delivered from various sites in the cochlea.

Many characteristics of the neural responses elicited by electrical stimulation of the peripheral auditory system can be demonstrated using far-field evoked potential measurements. Electrically evoked late responses (ELARs), such as the N1, P2, N2, P300 and mismatch negativity (MMN) occur with a latency beyond 50 ms and have complex generators that reflect various levels of cortical processing, including subcortical and thalamo-cortical projections, primary auditory cortex and association areas (Cunningham et al., 2000; Kraus et al., 1993a; Kraus and McGee, 1992; Näätanen and Picton, 1987; Scherg and von Cramon, 1986; Tremblay et al., 2001; Vaughan and Ritter, 1970). The generators of N1 and P2 are centered in the primary and secondary auditory complex (belt and parabelt regions in the anatomical model of Hackett et al., 2001 and planum temporale according to Näätanen and Picton, 1987). ELARs have been used widely to investigate auditory function in cochlear implant users (e.g., Wable et al., 2000; Groenen et al., 1996; Pelizzone et al., 1989). Their latency is less variable than their amplitude (Eggermont, 1988) and may therefore provide a better tool to assess the auditory pathway integrity. Similar ELAR latencies have been reported for cochlear implant users and normal hearing subjects in studies using speech stimuli delivered through a loudspeaker in the sound field (Kileny et al., 1997; Micco et al., 1995; Oviatt and Kileny, 1991). However, click stimuli delivered directly to the implanted electrodes and bypassing the speech processor resulted in shorter ELAR latencies in cochlear implant users (Ponton and Don, 1995; Ponton et al., 1996; Firszt et al., 2002a; Maurer et al., 2002). This shortening may be due to the fact that the delay in the travelling wave and the transduction delay are bypassed in implantees. This may also be a consequence of much better synchronization of a larger number of neural units by the electrical current pulses, resulting in reduced mean synaptic delays. Hence, the total time to reach the primary auditory cortex and the synaptic delays of cortico-cortical reactivation will be shorter. When the auxiliary input of the sound processor and then the recipient's program are used, this latency decrease may be compensated by the time the cochlear implant takes to process the sound, resulting in latencies similar to those of normal-hearing subjects. Nevertheless, the latency of the electrically evoked potential may be linked with the degeneration of auditory fibers (Guiraud et al., 2007) and reflect the maturity of the auditory pathway (Ponton et al., 1996a,b, 1999, 2000; Ponton and Don, 1995; Ponton and Eggermont, 2001; Eggermont et al., 1997; Sharma et al., 2002, 2005). Hence, it seems appropriate to use ELAR latency to investigate the impact of deafness on the auditory pathway if they are recorded in the same way for all subjects.

The effects of deafness on the auditory pathway have been investigated in many studies. There is abundant evidence indicating that, in mammals and birds, neural activity within the sensory pathway plays an important role in the development and maintenance of that pathway (Cowan, 1970; Globus, 1975; Purves and Lichtman, 1985). More specifically, widespread auditory degeneration is caused by auditory deprivation in the central auditory system (e.g., Hardie and Shepherd, 1999; Leake et al., 1992; Moore, 1994; Ryugo et al., 1997, 1998). Degeneration of cochlear hair cells results in secondary degeneration of the spiral ganglion cells of the auditory nerve (Kohenen, 1965. Johnsson, 1974; Spoendlin, 1975; Otte et al., 1978). The extent of survival depends on factors such as etiology, severity of the pathology, and duration of deafness (Nadol et al., 1989). Other changes due to auditory deprivation include reduction of cell density in the anteroventral cochlear nucleus and ventral cochlear nucleus, changes in the neural projections between brainstem nuclei (Nordeen et al., 1983), reduced cortical synaptic activity in cortico-cortical and cortico-thalamic connections (Kral et al., 2000), reduced number of primary dendrites in cortical pyramidal cells, and encroachment of auditory cortical areas by visual and somatosensory systems in congenitally deaf humans (Lee et al., 2001; Finney et al., 2001; McFeely et al., 1998; Nishimura et al., 1999). Some of these alterations can be objectively evidenced with ELAR latency. Less numerous neural cells may result in a decrease in neural transmission speed and an increase in synaptic delays (Rattay, 1987). More generally, a relationship between the number of neural fibers and conduction velocity was indeed shown for both sensory and motor pathways (respectively, Cavalcanti do Egito Vasconcelos et al., 2003; Morgan and Proske, 2001). Damage to the myelin sheaths also reduces the neural transmission speed (Zhou et al., 1995). Such alterations of conduction velocity could