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Research Paper

Delayed changes in auditory status in cochlear implant users with preserved acoustic hearing

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ABSTRACT

This retrospective review explores delayed-onset hearing loss in 85 individuals receiving cochlear implants designed to preserve acoustic hearing at the University of Iowa Hospitals and Clinics between 2001 and 2015. Repeated measures of unaided behavioral audiometric thresholds, electrode impedance, and electrically evoked compound action potential (ECAP) amplitude growth functions were used to characterize longitudinal changes in auditory status. Participants were grouped into two primary categories according to changes in unaided behavioral thresholds: (1) stable hearing or symmetrical hearing loss and (2) delayed loss of hearing in the implanted ear. Thirty-eight percent of this sample presented with delayed-onset hearing loss of various degrees and rates of change. Neither array type nor insertion approach (round window or cochleostomy) had a significant effect on prevalence. Electrode impedance increased abruptly for many individuals exhibiting precipitous hearing loss; the increase was often transient. The impedance increases were significantly larger than the impedance changes observed for individuals with stable or symmetrical hearing loss. Moreover, the impedance changes were associated with changes in behavioral thresholds for individuals with a precipitous drop in behavioral thresholds. These findings suggest a change in the electrode environment coincident with the change in auditory status. Changes in ECAP thresholds, growth function slopes, and suprathreshold amplitudes were not correlated with changes in behavioral thresholds, suggesting that neural responsiveness in the region excited by the implant is relatively stable. Further exploration into etiology of delayed-onset hearing loss post implantation is needed, with particular interest in mechanisms associated with changes in the intracochlear environment.

1. Introduction

Recent modifications to surgical procedures and electrode array designs facilitate the preservation of intracochlear structure and function and have thus expanded the number of patients who benefit from cochlear implantation. Although retention of acoustic hearing following cochlear implant (CI) surgery has become a common outcome, the degree ranges from minimal to complete, with some individuals experiencing total loss of acoustic hearing in the implanted ear (e.g. Helbig et al., 2016; Moteki et al., 2016; Gantz et al., 2016; Roland et al., 2016; van Abel et al., 2015; Hunter et al., 2016; Santa Maria et al., 2013). A decrease in hearing sensitivity

Abbreviations: AIC, Akaike's Information Criterion; ANOVA, Analysis of Variance; CI(s), cochlear implant(s); ECAP(s), electrically evoked compound action potential(s); dB, decibels; FDA, Food and Drug Administration; Hz, Hertz; IDE, Investigational Device Exemption; N, number; NRT, Neural Response Telemetry; PTA, pure tone average; sd, standard deviation; Z, impedance

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identified at the first post-operative appointment is commonly attributed to surgical trauma, an acute inflammatory response, or change in the system mechanics due to the presence of the array, but decreases in hearing sensitivity have also been observed months to years after surgery; the cause: undetermined. It is this delayed-onset hearing loss that is the focus of the present report.

The University of Iowa has been involved in hearing-preservation research since the 1990s (see Gantz et al., 2016 for an overview of research involving the first short-electrode array). Retrospective review of data from all individuals who have undergone hearing-preservation CI surgery at this institute allowed us to characterize delayed hearing loss across a longer time span than previously reported (up to 15 years for participants implanted with earliest generation devices). We also explored whether electrode impedance (Z) or electrically evoked compound action potential (ECAP) measures might provide insight into the underlying etiology.

Delayed-onset hearing loss as observed in this population may result from the body's reaction to the implanted materials. Histologic analysis often reveals fibrotic encapsulation of the electrode array and tissue comprised of various cell types within the cochlear labyrinth (e.g. Linthicum et al., 1991; Nadol et al., 2008; Seyyedi and Nadol, 2014; Quesnel et al., 2015). A change in the physical structure of the inner ear scalae has the potential to change the passive mechanics of the system. Modeled as an increase in damping, fibrotic tissue has the potential to reduce basilar membrane vibration. Reduced movement in apical cochlear regions, which would decrease the ear's sensitivity to low-frequency stimuli, could occur when fibrotic tissue invades the scala tympani (Choi and Oghalai, 2005). Moreover, the biological processes responsible for the fibrotic tissue may be toxic to the neurosensory structures of the inner ear (Bas et al., 2015; Eshraghi et al., 2015), which could also account for a decrease in acoustic hearing. Significant correlations between the degree of tissue reaction and the amount of acoustic hearing loss have been observed in animals (e.g. O'Leary et al., 2013), which suggests that the tissue response may also be relevant to humans experiencing delayed loss of acoustic hearing.

Changes in the physical structure of the cochlear scalae cannot be detected using current noninvasive imaging techniques; however, clues may be gleaned from impedance measures that are routinely recorded from each intracochlear electrode during clinical appointments. Electrode impedance provides information about the status of the electrode and the surrounding environment, and has been shown to be sensitive to changes in tissue growth around the electrode array (Wilk et al., 2016). Histological analysis of a temporal bone from a deceased CI recipient who experienced a complete loss of acoustic hearing in the implanted ear between one and four months after implantation revealed that although post mortem hair cell and neural counts were not significantly different for implanted and unimplanted ears, the implanted ear presented with extensive fibrous tissue in the cochlear scalae. Electrode impedance values available at the final pre mortem audiological appointment were similar to impedance values recorded shortly after implantation; however, a transient increase occurred around the time of the acoustic hearing loss (see Fig. 3B in Quesnel et al., 2015). CI audiologists at this institute (and from other institutes) have also observed variations in electrode impedance at the time a drop in acoustic hearing is identified, but these anecdotal reports have yet to be systematically evaluated.

Electrically evoked compound action potentials (ECAPs) can also be recorded using clinical software and are routinely measured from many individuals implanted at the University of Iowa during research appointments. These potentials are the synchronous response of electrically stimulated auditory neurons, and as such, provide information about the status of the auditory nerve. Correlations between spiral ganglion neuron loss and the degree of inflammation responses have been observed (e.g. Xu et al., 1997; Fayad et al., 2009), which is consistent with the suggestion that an inflammatory response also may be toxic to the neurosensory structures in the cochlea (Bas et al., 2015; Eshraghi et al., 2015). A hypersensitivity reaction to the electrode array may be more common than originally thought (e.g. Seyyedi and Nadol, 2014), and although ECAP measures generally have been shown to be relatively stable over time (e.g. Hughes et al., 2001; Brown et al., 2010), a closer evaluation of within-subject changes, particularly for individuals who demonstrate changes in acoustic hearing, is warranted.

This retrospective review aims to characterize the prevalence and nature of delayed loss of acoustic hearing by compiling repeated measures of unaided audiometric thresholds, electrode impedance, and ECAP amplitude growth functions in a relatively large sample of hearing-preservation CI recipients. Systematic changes over time were interpreted as reflecting a change in the status of the auditory system. Because the data set contained several electrode array designs and surgical approaches, they were evaluated as factors relating to the prevalence of delayed hearing loss. Evaluation of the physical and physiological measures also allowed us to explore etiology, albeit indirectly. It was hypothesized that an increase in electrode impedance would be observed more often for individuals with delayed hearing loss than individuals with stable hearing if a change in the intracochlear environment, as would be the case with fibrous tissue growth, was in fact a contributing mechanism. Correlation analysis was used to evaluate whether changes in ECAPs (i.e. threshold, slope and suprathreshold amplitude) and changes in unaided audiometric thresholds were related. It was hypothesized that a significant correlation would be consistent with a global change in neural status as a contributing factor.

2. Methods

2.1. Participants

As of October 14, 2015, 143 adults (ages 18 years and older) had been implanted at the University of Iowa with hearing preservation electrode arrays manufactured by Cochlear Ltd (Cochlear Americas, CO, USA): S8 (24M and 24RE based receiver-stimulator), S12, L24, and 422. Characteristics of these arrays are provided in Table 1. The electrode arrays vary in physical dimensions, number of electrodes, overall length and insertion depth. Some electrode arrays were designed specifically for insertion via a cochleostomy while others were designed for insertion through either a cochleostomy or the round window (Table 1).

The “S” arrays were all implanted under an Investigational Device Exemption (IDE) approved by the Food and Drug Administration (FDA; S8: G990155; S12: G070016). Preoperative unaided audiometric thresholds $\leq$60 dB HL at 125 and 250 Hz and $\geq$75 dB HL above 1500 Hz were required for candidacy. The L24 array was approved by the FDA for clinical use in March of 2014; arrays implanted prior to that date were approved using an IDE (G070191 and G110089). For L24 arrays implanted under IDE G070191, audiometric threshold criteria were the same as for the S8 and S12 arrays. For individuals implanted under IDE G110089, a five-frequency pure tone average (PTA; 125, 250, 500, 750 and 1000 Hz) between 60 and 90 dB HL was required. The 422 array is FDA approved for clinical use. It was designed to preserve cochlear structures rather than acoustic hearing, and has been used in individuals both with and without acoustic hearing prior to surgery. In this report, only individuals with 422s whose preoperative audiometric thresholds were $\leq$60 dB HL at 125 and 250 Hz were