EFFECTS OF DEAFNESS AND COCHLEAR IMPLANT USE ON DEVELOPMENT OF HUMAN AUDITORY FUNCTION

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Profound deafness in children disrupts and delays the development of language and communication skills. The extent to which these skills are affected depends on the age of onset and the duration of deafness (Busby et al., 1989). For profoundly deaf children, meaningful auditory sensation may be restored by electrical stimulation of auditory nerve fibers through a cochlear implant. By examining children fitted with a cochlear implant, it may be possible to determine the effects that deafness and the subsequent reintroduction of stimulation have on the maturation of those areas of the brain that are deprived of their normal sensory input.

A direct and objective measure of auditory cortical maturation can be obtained by recording electrophysiological activity such as the cortical auditory evoked response. Early components of the cortical auditory evoked response reflect stimulus-onset detection in sensory cortex (Courchesne, 1978). Maturational changes in cortical activity are reflected in age-related decreases in the latency of these components.

To assess the effects of deafness and cochlear implant use on cortical maturation, age-related changes in the latency of the positive peak, P1, were examined in implanted and normal-hearing children and adults. For normal-hearing subjects, cortical activity was evoked by applying 100 μs voltage pulses to a headphone. For implant users, special computer hardware that bypassed the speech processor was used to deliver 200 μs/phase biphasic current pulses directly to the implant. Each stimulus consisted of a brief train of ten acoustic clicks or electric pulses. Click stimuli were presented monaurally to the left ear of normal-hearing subjects at approximately 65 dB above threshold. Stimulation levels were set individually for each implant user at a loud but comfortable level. Evoked responses were recorded at 30 electrode locations on the scalp. Evoked response latencies are reported for the vertex electrode Cz, as this is the most commonly used electrode location in both research and clinical settings.

Large wave-shape differences are apparent between the younger children (< age 10) and adults for both normal-hearing and implanted subjects. For the six and seven year olds, the AER is dominated by a large positive peak at about 100 ms. The general pattern of age-related latency decrease for this peak suggests that it is probably equivalent to the adult P1. Age-related latency changes for P1 are non-linear, with the difference between children and adults decreasing exponentially for both normal-hearing and implanted children. Curve fit analyses of the P1 data showed that the rate of latency decrease was the same for normal-hearing and implanted children. However for implanted children, the age at which P1 latency becomes adult-like is delayed. To further examine the nature of this delay, the implanted children were divided into short (≤ 2 years), medium (4-6 years), and long-term (8-9 years) auditory deprivation groups based on the duration of deafness (see Figure 1). A near perfect correspondence exists between the duration of deprivation and P1 maturational delay if it is assumed that some time elapses between the onset of deafness and its detection.

These findings suggest that in absence of cortical activation during the period of deafness, maturation of activity in auditory cortex does not progress. However, once stimulation of the auditory pathway is restored, some, if not all, maturational processes resume a normal course.


Figure 1: Maturational delay for P1 latency as a function of duration of auditory deprivation (time between detection of deafness and time of implantation).