THE AUDITORY CORTEX AND AUDITORY DEPRIVATION: EXPERIENCE WITH COCHLEAR IMPLANTS IN THE CONGENITALLY DEAF.

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The primary auditory cortex (AI) exhibits a topographic representation of the organ of Corti in normal hearing animals. Plasticity studies have shown that this orderly representation of frequency can be modified following a restricted hearing loss or by behavioural training[1,2]. Little is known, however, of the effects of a profound hearing loss on AI, although a number of early studies have suggested an enhancement of activity from other modalities[3]. Knowledge of the functional status of the central auditory pathway in the profoundly deaf, and the ability of these structures to undergo reorganization - particularly following long periods of auditory deprivation - are important issues for the clinical management of cochlear implant patients. In this paper we review our recent clinical and experimental experience with cochlear implants in the congenitally deaf.

Speech perception skills among cochlear implant patients show a significant negative correlation with duration of deafness and a positive correlation with auditory experience obtained both prior to the hearing loss and with use of the implant[4]. The trend towards implantation of younger children, supported by theses findings, have resulted in some congenitally deaf children - after a number of years of experience - obtaining speech perception skills equivalent to postlinguistically deafened adults. While there are many factors contributing to clinical performance using a cochlear implant, improvement observed with experience suggests that the ability of the central auditory system to undergo functional reorganisation may play a significant role.

We have used the congenitally deaf white cat to study cortical evoked potential (CEP) and single unit activity in the AI evoked by electrical stimulation of the auditory nerve. While we studied the animals as adults, the absence of auditory brainstem responses at one month of age demonstrated that these animals had little prior auditory experience. Under barbiturate anaesthesia, CEP and single unit activity were recorded from the contralateral AI in response to bipolar electrical stimulation of the cochlea. Threshold, morphology and latency of the CEP depended on location within the AI, with latency varying from ~10 - 20 ms. Although limited, there was evidence of threshold shifts with site of electrical stimulation in accordance with the known cochleotopic organization of AI. Thresholds also varied with geometry of the stimulating electrodes in accordance with changes we have previously observed in normal hearing animals. If present at all, CEP s recorded outside AI exhibited very high thresholds. Single unit recordings exhibited properties similar to CEP s. Increasing stimulus intensity resulted in an increase in spike rate and a decrease in latencies to a minimum of ~ 10 ms, and thresholds varied with geometry of the stimulating electrodes. Strongly driven responses showed adaptation of spontaneous activity, however, even at saturation currents the degree of synchronization was less than we have observed when recording from brainstem nuclei. Finally, while not studied exhaustively, we could not evoke visual or somatosensory CEP s from the AI.

These studies help illustrate the functional status of the AI following auditory deprivation and provide evidence of potential reorganization with clinical experience.

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