342 Effects of Long-Term Deafness and Delayed Chronic Intracochlear Electrical Stimulation on the Primary Auditory Cortex

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Behaviorally relevant chronic intra-cochlear electrical stimulation (ES), when delivered from a young age, is known to alter spectral (spatial) and temporal processing in the auditory system [1,2]. Whether these effects are limited to stimulation that is initiated during the early critical periods, or also occurs when stimulation is commenced after long-term deafness, is less clear. We neonatally deafened five cats via daily neomycin injections, and at two months of age implanted a multi-channel scala tympani electrode array. A modified clinical cochlear implant was used to deliver environmentally derived chronic ES from eight to fourteen months of age. We recorded from single- and multi-unit clusters ($n = 300$) in the primary auditory cortex (AI) using a combination of single tungsten and multi-channel silicon electrode arrays. We assessed spectral processing in AI by measuring the cochlea-to-cortex mapping, and the spread of cortical activation. We assessed temporal resolution in AI by measuring the jitter in response latency and the maximum rate at which clusters could be driven. Similar to chronic ES initiated early in life [1], delayed ES had little effect on the basic response properties of AI neurons, but did reverse the disruption of the cochlea-to-cortex mapping and reduction in maximum driven rate (Mann-Whitney; $p < 0.05$) seen with long-term deafness. The late initiation of ES did not, however, reverse the increase in spread of activation or increase in response latency jitter seen with long-term deafness. We hypothesize that the inability of electrical activation of the cochlea after the closure of the normal critical period to reverse the increased spread of activation and response latency jitter contributes to the poorer performance observed among congenitally deaf human patients implanted later in life.