Author/s:
Shepherd, R. K.; Matsushima, J; Clark, Graeme M.

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ELECTRICAL STIMULATION OF THE AUDITORY NERVE IN DEAF KITTENS: EFFECTS ON THE SPIRAL GANGLION
R.K. Shepherd, J. Matsushima* and G. M. Clark, Department of Otolaryngology, University of Melbourne, Parkville, Victoria 3052, Australia.

Cochlear pathology following the administration of ototoxic drugs results in a widespread and rapid loss of sensory hair cells followed by a gradual degeneration of auditory nerve fibres and their cell bodies, the spiral ganglion. Recently, two studies have described increased spiral ganglion cell survival in the cochleas of deafened animals following chronic electrical stimulation of the auditory nerve (Hartshorn et al., 1991; Leake et al., 1991). If electrical stimulation is shown to have a trophic effect on degenerating auditory nerve fibres, these findings will significantly influence the preoperative management of cochlear implant patients. The aim of the present study was to corroborate these earlier reports and to evaluate the general tissue response of deafened cochleae in young animals following chronic electrical stimulation.

Four normal hearing kittens were deafened at six weeks of age by co-administration of kanamycin and ethacrynate acid (Xu et al., 1990). At two months of age each animal was bilaterally implanted with scala tympani electrode arrays under halothane/methoxyflurane anaesthesia. Unilateral electrical stimulation commenced two weeks following surgery using charge balanced biphasic current pulses of 200 µs per phase, presented at a rate of 100 pulses per second. Charge densities at the electrode surface were in the range 12 to 24 µC cm⁻² geom. per phase. Periodically during the stimulation program electrically-evoked auditory brainstem responses (EABRs) were recorded under sodium sedation (12 mg kg⁻¹ i.m.). Following total stimulation periods of 1100 - 1600 hours each animal was killed with an overdose of pentobarbitone sodium (i.m.) and the cochleas removed for histology. Spiral ganglion cell densities were determined for a region adjacent to the stimulating electrodes and were compared statistically with similar populations from the contralateral control cochlea using the paired t-test.

Histopathological examination of the control cochleas showed a minimal tissue response to the chronically implanted electrode array. In general, the stimulated cochleas exhibited a slightly more extensive soft tissue response together with small amounts of new bone. Significantly, there was no statistical difference in spiral ganglion cell density adjacent to the stimulating electrodes compared with similar populations from control cochleas. Finally, chronically recorded EABR's showed stable thresholds together with a monotonic increase in the response amplitude with implant duration.

In contrast with previous reports, the present findings show no evidence of a trophic influence of electrical stimulation on the residual spiral ganglion. They do, however, indicate that chronic electrical stimulation does not adversely affect the spiral ganglion population in young deafened animals. The results also show that these cochleas may evoke a slightly more extensive tissue reaction in response to electrical stimulation than observed in normal hearing kittens (Shepherd et al., 1991). The mechanism underlying this increased tissue response requires further investigation. Finally, the stability of the EABR thresholds observed throughout this study are consistent with our histological findings. The mechanism underlying the increased response amplitude with implant duration remains unclear, however, we have observed similar increases in normal hearing kittens (Shepherd et al., 1991).


* Department of Otolaryngology, Hokkaido University, Sapporo, Japan.
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