

## ELECTRICAL STIMULATION OF THE AUDITORY NERVE: STIMULUS INDUCED REDUCTIONS IN NEURAL EXCITABILITY

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Electrical stimulation of the auditory nerve elicits highly synchronised neural activity (Javel et al., in press). As the stimulus current is increased the neural response becomes highly deterministic with every current pulse eliciting a spike even at stimulus rates of 600-800 pulses per second (pps). Our previous acute experimental studies have shown that high stimulus rates (>200 pps) and high stimulus currents (>1.0 mA) can result in temporary and sometimes permanent reductions in the excitability of the auditory nerve (Shepherd and Clark, 1986). The present study was designed to examine the mechanisms underlying these stimulus induced reductions in excitability. These results will have implications for the maximum safe and effective stimulus rates that can be employed in cochlear implants.

Anaesthesia was induced in normal hearing cats using ketamine hydrochloride (20 mg kg<sup>-1</sup> i.p.), and maintained for the duration of the experiment with supplemental doses of pentobarbitone sodium (i.p.). Bipolar scala tympani electrodes were implanted bilaterally. One cochlea was electrically stimulated for 30-60 minutes using stimulus intensities and rates both within and above the maximum range used clinically. The second cochlea was a non-stimulated control. Electrical stimuli consisted of charge balanced biphasic current pulses. Stimulus induced reductions in the excitability of the auditory nerve were monitored by recording wave iv of the scalp recorded electrically-evoked auditory brainstem response (EABR) or the electrically-evoked compound action potential (ECAP) recorded from the auditory nerve. In some experiments strychnine, a potent efferent blocker, was administered (0.5-1.0 mg kg<sup>-1</sup> i.p.) in order to examine cochlear efferent contributions to these stimulus induced changes. The effect of anoxia was studied in several animals by ventilating the animal with 100% nitrogen during periods of acute electrical stimulation. Following varying periods of anoxia the animal was allowed to recover by ventilating it with 100% oxygen. EABRs were monitored prior, during and following the period of anoxia. At the conclusion of the experiment each animal was killed with an overdose of pentobarbitone sodium (i.p.) and tissue samples were removed for examination.

Acute periods of electrical stimulation produced both per- and post-stimulus reductions in the EABR and ECAP. The extent of these changes increased with stimulus rate and stimulus current. The EABR reflected changes occurring in the ECAP, suggesting that these effects originated at the level of the auditory nerve. The administration of strychnine had no effect on the stimulus induced reductions observed in evoked potentials. In contrast, EABRs recorded from acutely stimulated cochleae in animals made temporarily anoxic, exhibited extensive reductions. Initially, an increase in the latency of all EABR waves was observed. With continued anoxia the amplitudes of all waves decreased significantly. Moreover, post-stimulus recovery of these EABRs was prolonged. Control EABRs exhibited little change during the period of anoxia.

The present results suggest that the extent of these stimulus induced reductions in the EABR and ECAP were dependent on the degree of electrically evoked neural activity and that these changes appeared to originate at the level of the auditory nerve. The potentiation of these stimulus induced changes in the presence of anoxia suggests that they are of metabolic origin.

Javel, E., Tong, Y.C., Shepherd, R.K. & Clark, G.M. *Annals of Otolaryngology and Laryngology*, in press.

Shepherd, R.K. & Clark, G.M. (1986) *Proceedings of the Australian Physiological and Pharmacological Society*, 17, 16P.



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**Title:**

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[Abstract]

**Date:**

1987

**Citation:**

Shepherd, R. K., & Clark, G. M. (1987). Electrical stimulation of the auditory nerve: stimulus induced reductions in neural excitability [Abstract]. In Proceedings of the Australian Physiological and Pharmacological Society.

**Persistent Link:**

<http://hdl.handle.net/11343/26830>

**File Description:**

Electrical stimulation of the auditory nerve: stimulus induced reductions in neural excitability  
[Abstract]