

EXCITATION AND INHIBITION IN RESPONSES OF COCHLEAR NUCLEUS SINGLE UNITS TO ELECTRICAL STIMULATION OF THE AUDITORY NERVE

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We have investigated the extracellular response of cochlear nucleus single units to electrical pulse train stimulation of the auditory nerve via an electrode array implanted into the feline scala tympani. The findings of these studies provide the physiological basis for the future design of the implanted electrode array and electronics and external speech processor of a cochlear implant prosthesis system.

Eleven adult domestic cats were studied. A premedication of 0.3 mg atropine sulphate intramuscularly was given, anaesthesia was induced with 50mg of ketamine and 10mg xylazine intraperitoneally, and maintained with 30 mg of sodium pentobarbitone intravenously every 2 hours as required. After the experiment the animal was sacrificed with an overdose of sodium pentobarbitone intravenously and the cochlear nucleus removed for histological sectioning. The surgery, the signal generation and recording systems have been described previously (Shepherd et al, 1988). Hearing thresholds were monitored using auditory brainstem responses. In some cats a loss of acuity of up to 10dB was found above 10 kHz after cochlear implantation.

For each unit isolated a tuning curve was derived and responses to 100 msec acoustic stimuli recorded. The acoustic stimuli were routinely tones at best frequency and other frequencies (500-40000Hz) and noise. The standard electrical stimulus was a 100 msec burst of biphasic current pulses, 100 or 200 usec per phase and with repetition rates of 100, 200 and 400 pulses per second. All stimuli were repeated every 400 msec with 50 repetitions. For nonspontaneous units inhibitory phenomena were identified by the method of Shofner and Young (1985), namely the suppression of electrically driven responses by acoustic tones outside the excitatory responses area of the unit.

Single unit responses to both acoustic and electric stimuli were recorded from 74 units throughout the cochlear nucleus. Units showing no sign of inhibitory responses to acoustic stimuli responded to electrical pulse trains by an action potential 1.1-3.7 msec poststimulus after each pulse in the stimulus. Except for the longer latency of response this result was the same as that observed in the auditory nerve (Javel et al, 1987) and primary like responders of the VCN (Maffi et al, 1987). Units exhibiting inhibitory sidebands to acoustic stimuli demonstrated a variety of responses to electrical stimulation. These electrical responses included the a reduction in the spike rate of spontaneous activity or an excitatory tone, a response to early pulses in the pulse train followed by a reduction in spontaneous activity or a response to pulses late in the train. Often a unit would exhibit several of these patterns as stimulus current increased. Spontaneous activity of units exhibiting primarily inhibitory responses to tones was reduced by the electrical stimulus. We suggest that the complex patterns of response to the electrical pulse trains observed in the cochlear nucleus units are due to the interplay of excitatory and inhibitory drives which are dependent on the locus of the primary input and may exhibit different time courses (van Gisbergen et al, 1975). We propose that the temporal response patterns for electrical stimulation differ from those of best frequency tones because the locus of the primary auditory input excited by the electrical stimulus differs from that of the tonal stimulus.

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