DECREMENT IN AUDITORY NERVE FUNCTION FOLLOWING ACUTE HIGH RATE STIMULATION IN GUINEA PIGS

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Cochlear implants have been shown to sucessfully provide profoundly deaf patients with auditory cues for speech discrimination. Psychophysical studies suggested that speech processing strategies based on stimulus rates of up to 1000 pulses per second (pps) may lead to an improvement in speech perception, due to a better representation of the rapid variations in the amplitude of speech. However, "neural fatigue" has been known to occur following brief periods of electrical stimulation at rates high enough to ensure that stimuli occur within the neurons relative refractory period, and has been shown to depend on stimulus duration and rate of the evoked neural activity. Prolonged electrical stimulation at these high stimulus rates could, therefore, have an adverse effect on the neurons metabolism and result in cellular energy depletion.

In the present study we examined the effects of acute high rate electrical stimulation using various stimulus paradigms on auditory nerve excitability. Charge balanced, biphasic current pulses, delivered to a bipolar electrode pair, at stimulus rates of 400 or 1000 pps and a stimulus intensity of 0.34 μC/phase were used in an animal model. This stimulus intensity falls within the range of the Nucleus Cochlear implant. Post-stimulus recovery of neural populations close to the stimulating electrodes was monitored using the electrically evoked auditory brainstem response (EABR). A continuous and a 50% duty cycle stimulus paradigm were used, both featuring fixed-amplitude pulses. Additionally, an amplitude-modulated stimulus paradigm, using a 4-talker babble sequence, was used. This stimulus injected the same total charge over the stimulation period compared with 50% duty cycle stimulation. Continuous stimulation at 400 pps produced a significant reduction in auditory nerve excitability, followed by a slow and incomplete recovery of the EABR. Following both 50% duty cycle and 4-talker babble stimulation for two and four hours, EABR decrement was reduced, although it recovered rapidly to pre-stimulus levels. Immediately following continuous stimulation at 1000 pps no EABR's could be recorded. Introducing a 50% duty cycle resulted in a statistically significant increase in post-stimulus EABR recovery, even if total charge was kept constant. An initial decrement in the EABR was also observed following 4-talker babble stimulation, however, the response amplitude underwent rapid and complete recovery. While these results show the importance of both duty cycle and amplitude modulation in minimizing post-stimulus neural fatigue, they also indicate that the post-stimulus reduction in auditory nerve excitability depends on stimulus rate. These short term changes in excitability may have important consequences for speech processing strategies based on high stimulus rates.

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