Electrical stimulation of neural tissue involves the transfer of charge to tissue via electrodes. Safe charge transfer can be achieved using biphasic current pulses designed to reduce the generation of direct current (DC) or the production of electrochemical products. However, neural stimulators must also use capacitors in series with electrodes, or electrode shorting between current pulses, to further minimize DC due to electrode polarization. We have recently shown that high rate electrical stimulation, using stimulus intensities above clinical levels, can induce a significant decrement in the excitability of the auditory nerve. While these changes have been attributed to neuronal hyperactivity, DC levels of up to 2.8 μA were also reported in this study. The present investigation was established to establish the extent to which this DC contributed to the decrement in auditory nerve excitability. Twenty adult guinea pigs were anaesthetized with ketamine (40 mg/kg) and xylazine (4 mg/kg), bilaterally implanted and unilaterally stimulated for two hours using charge balanced biphasic current pulses. Animals were stimulated using rates of 200, 400 or 1000 pulses/s (pps) at a high stimulus intensity (0.34 μC/phase). Two techniques were used to minimize DC: i) electrode shorting, and ii) capacitive coupling. Electrically evoked auditory brainstem responses (EABR’s) were recorded before and periodically following the acute stimulation. Significant post-stimulus reductions in the EABR amplitude were observed following both stimulus regimes. Furthermore, only partial EABR recovery was observed during the three hour post-stimulus monitoring period. Stimulation using capacitive coupled electrodes, which eliminated all DC, showed a highly significant reduction in the response amplitude of wave III as a function of stimulus rate; 200 pps, 85% of prestimulus amplitude (p < 0.01); 400 pps, 30% (p < 0.001); and 1000 pps, 10% (p < 0.001) at EABR probe currents approximately 16 dB below the intensity of the acute stimulus. Significantly, these reductions in amplitude were similar to the changes observed following stimulation using electrode shorting techniques. The present findings indicate that the majority of the stimulus induced changes observed are associated with prolonged neuronal hyperactivity and are not related to DC.


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**Poster 218**

**THE AUDITORY BRAINSTEM RESPONSE (ABR) AND RESPONSES FROM BRAINSTEM NUCLEI DURING DEVELOPMENT IN MACROPUS EUGENII.**

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The acoustic click-evoked auditory brainstem response (ABR) and the evoked potentials in several nuclei in the brainstem were recorded at stages of development in the tammar wallaby. Pouch young (PY) animals, aged between 90 and 250 days, were anaesthetised with urethane (20%, 1ml/100g, i.p. initially; a quarter of the initial dose at intervals subsequently to maintain anaesthesia). Acoustic clicks at up to 71 dB SPL (at the left ear) were presented from a distance of 820mm and about 45" left of the animal's mid-line. ABRs, being the average of scalp-recorded responses to 500-1000 stimulus presentations, were obtained prior to craniotomy, which exposed the right side of the brain for recording more focal, evoked potentials from auditory nuclei. Electrolytic lesions marked the electrode positions of maximal evoked response amplitudes. Following urethane overdose and perfusion of the preparation, recording sites were verified histologically. Auditory evoked potentials were recorded from the superior colliculus (SC), inferior colliculus (IC), superior olive (SO), and auditory nerve root (ANR). The earliest ages at which responses occurred at these sites were found to be 101 days for the ANR, 112 days for the SO, 116 days for the IC, and 182 days for the SC. The first auditory response from the IC appeared at its rostral pole. With further development, more caudal parts of the IC showed an evoked auditory response. At 125 days, all of the IC was responsive to sound. From one PY at age 101 days, a click at 71dB SPL elicited an ABR, which was a small positive-negative deflection with a latency of the positive peak of 5.82ms. In the same animal, the only focal response was recorded at the ANR, with the latencies of its positive and negative peaks being 4.3 and 5.79ms respectively. At 112 days, the ABR consisted of two or three positive components. The response from the SO had two positive components which occurred in approximate correspondence with the latter two components of the ABR. After 140 days, the ABR consisted of four to six components and the response from the SO (at this age still two positive peaks) retained temporal correspondence with waves I-III of the ABR. In PY less than age 150 days, the IC response (latency >13.5ms) occurred beyond the range of the ABR (3-10ms); in animals aged over 150 days, the IC response had reduced latency (7-9ms for its early components) that overlapped the long latency components of the ABR (waves IV-VI). After age 182 days, the early component of the SC response (negative, latency 5-7ms) overlapped the ABR. Although the time course of the anatomical establishment of the auditory pathway is unknown, it appears that functionally, development in the auditory system follows an ascending sequence.
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