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EFFECT OF LOCAL ANESTHETIC ON RODENT BRAINSTEM TOOTH PULP-EVOKED POTENTIALS

Local anesthetic failure is common in those receiving dental treatment for irreversible pulpitis, which produces discomfort and pain during treatment and increases the risk of local/systemic toxicity due to the need for higher anesthetic dosage. We hypothesize that anesthetic failure is due to a change in voltage-gated sodium channel subunits along the length of pulpal afferent axons. The first step in testing this hypothesis is to establish an animal model for measuring anesthetic effect on pulpal afferents of naïve and pulp-inflamed rats, and to use this model to record any changes due to anesthetic administration. A dental drill was used to access the pulp in the right first maxillary molar in the two groups of male Sprague-Dawley rats, into which a bipolar concentric electrode was placed. The brainstem was surgically exposed, and, using a silver-ball electrode, potentials were recorded from the surface of the spinal tract of the trigeminal nerve as the pulp was stimulated (single square wave pulses; 0.25 Hz; 3X threshold). Ten- μ L doses of 2% lidocaine were applied to the infraorbital nerve of each rat at 2-min intervals. In naïve rats ($n=10$), complete knockdown of the tooth pulp evoked potential was seen (as measured by peak-to-peak amplitude), on average, after 2.5 doses (± 0.2). In rats in which the pulp was inflamed by exposing the pulp cavity 72 hours in advance ($n=10$), complete knockdown of the potential was not seen, and an average of 8.5 doses (± 1.2) was needed to achieve a stable, partially reduced potential, supporting the notion that inflammation alters pulp sensitivity to local anesthetics. Future experiments will explore the biophysical properties, relative density, and distribution of sodium channels in pulpal afferent axons. (Supported in part by the NIDCR Training Program DE07334-11, NPS discretionary funds, and NIH grant DE018252).