

TITLE: ATRACURIUM ALTERS CORTICAL MAGNETIC MOTOR EVOKED POTENTIALS

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Introduction Magnetically induced cortical motor evoked potentials (MMEP) allow assessment of the spinal cord motor tracts. Interest in surgical monitoring prompted us to conduct an institutionally approved study to assess the effect of vecuronium muscle blockage on the EMG recordings of MMEP.

Methods Five adult cynomolgus monkeys (M fascicularis) (3-4.6 Kg) were anesthetized with ketamine (15-20 mg/kg IM followed by continuous infusion 10-15 mg/kg/hr). The trachea was intubated and ventilation supported. MMEP was elicited by a Cadwell MES-10 at supra-maximal intensity (70-80%) with the coil placed optimally over the scalp. Direct nerve stimulation was applied to the median nerve at the wrist using supramaximal constant current 0.3 ms stimulation. The EMG response to MMEP or single stimulation was recorded from the opponens pollicis (elbow ground) using a Biologic Navigator (Filteration 10-3000 Hz, 1000 or 3000 amplification, respectively). Mechanical twitch height was measured for the single or train of four stimulation (2 Hz) using a balloon sensing device.

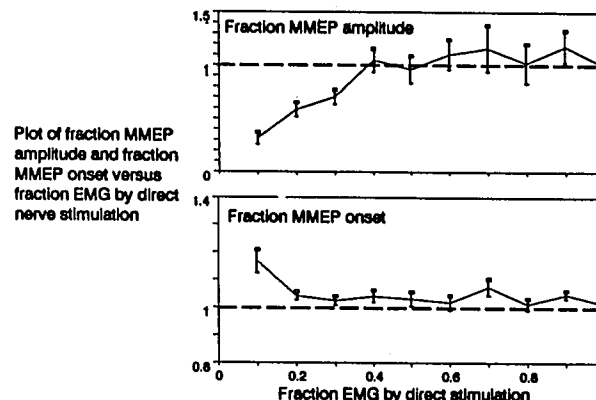
Atracurium (0-5.25 mcg/kg/min) was infused after baseline recordings to produce varying degrees of neuromuscular paralysis until obliteration of EMG and mechanical response and then gradual resolution. The above electrical and mechanical responses were recorded and stored during the block in a paradigm that allowed 10 sec pause between stimulations and additional 50 sec prior to MMEP. Extracted from responses were response amplitude (for the EMG it was voltage from minimum to maximum peak) and MMEP onset (time from stimulation to first deflection from baseline of the complex). Data were analyzed by comparing the data with the measured mechanical change and degree of EMG reduction from five averaged baseline recordings.

Results Shown at the right are plots of the MMEP onset and amplitude (mean \pm sem) at incremental degrees of block (fraction EMG remaining \pm 0.05). As shown,

onset was essentially unaltered until greater than 80% EMG depression, beyond which an increase was seen. Amplitude was essentially unchanged until greater than 60% EMG depression with reduction below 50% of baseline values occurring at greater than 80% EMG reduction.

Discussion This study suggests that MMEP monitoring under ketamine anesthesia, as measured by the EMG of the opponens pollicis, is well maintained during neuromuscular block with infusion of atracurium until the EMG from direct stimulation of the median nerve is depressed greater than 80% at which time onset increase and amplitude decreases are significant. The differences in depression between the EMG from direct nerve stimulation and cortical stimulation may result from differing muscle group sensitivities and differences in the type of neural signal to the muscle.

This study was funded by an institutional grant from the University of Texas.



TITLE: EFFECTS OF HYPERGLYCEMIA ON SEIZURE INDUCED BRAIN DAMAGE IN THE RAT

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Objectives. Hyperglycemia is deleterious in the presence of global brain ischemia (1). Even without energy failure, the substantia nigra pars reticulata (SNPR) in rat brain has been shown to be particularly susceptible to seizure (SZ)- and ischemia-mediated brain damage, and is known to be sensitive to lactic acidosis (2). We tested the hypothesis that hyperglycemia exacerbates seizure-induced damage to the SNPR.

Methods. Fed male Sprague-Dawley rats 300-400 g were used. Rats were anesthetized with halothane, intubated, and mechanically ventilated. Vascular cannule, rectal temperature probe, and EEG screws were placed. Halothane was discontinued for 1 hour. Three treatment groups (N=20 each) were subdivided by duration of SZ and recovery period, as follows (n=5 each subgroup): Controls, LR 3 ml/kg loading dose (LD) followed by 4 ml/kg/hr 40% mannitol LD 3.4 ml/kg with every 5 min .56 ml/kg; 50% glc LD

2.8 ml/kg followed by 40% glc 6.25 ml/kg/h. Infusions were started 1 h preSZ. SZ were induced with flurothyl 3% inhaled for 45 or 75 min. Acute animals underwent cerebral perfusion fixation at the end of SZ and the recovery group underwent 2 hr mechanical ventilation prior to perfusion fixation. At end SZ thiopental 15 mg/kg IV was administered. Brains were assessed for presence of eosinophilic neurons (EN) in neocortical areas and substantia nigra with severity of damage graded from 0-5 (0=no damage, 5=100% EN). SNPR was also graded (0-5) for degree of vacuolation (vac). Riddit analysis was used to test statistical significance.

Results. Blood glc was 59-107 mg% in control and mannitol groups and was 409 and 452 mg% in the glc treated groups. Glc administration decreased the grade of SNPR damage following 75 min flurothyl SZ plus recovery (SNPR vac = 2.8 vs control = 4.0, SNPR EN = 3.3 vs control = 4.0; P < .05). No effect was evident for mannitol infusion or acute groups.

Conclusions. Hyperglycemia can be protective versus SZ-induced SNPR damage.

References

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