TITLE: HYPOTHERMIA TRIGGERS SPONTANEOUS POST-ANESTHETIC TREMOR

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We have previously demonstrated that tremor during recovery from isoflurane anesthesia has two electromyographic (EMG) components, one resembling shivering and the other resembling clonus (5-7 Hz bursting pattern).[1] Both tremor patterns were most common at 0.1-0.2% end-tidal isoflurane. Because all patients in that study were hypothermic, we were unable to determine whether hypothermia was required in addition to "intermediate" isoflurane concentrations. Therefore, we tested the hypothesis that hypothermia triggers spontaneous tremor during recovery from isoflurane anesthesia.

With IRB approval, 9 volunteers each participated in the study on 3 separate days: 1) anesthesia for 2 h with ≈ 0.9% isoflurane/air without change in tympanic temperature; 2) similar anesthesia with 1.5°C reduction in tympanic temperature; and 3) 1.5°C hypothermia without anesthesia, induced by central venous infusion of ≈ 4 L Ringer's at 3°C. Isoflurane concentrations were determined using a Capnomac® (Datex). EMGs from 4 muscles were digitized at 512 Hz: RMS intensity, interchannel correlation, and power spectra were calculated.

Hypothermia without anesthesia produced vigorous shivering with a typical synchronous 4-8 cycle/min "waxing and waning" pattern. Increased muscle tone (tonic stiffening without tremor or "waxing and waning") was observed during the initial recovery period following each anesthetic administration at isoflurane concentrations ≥ 0.4%. No spontaneous tremor was observed when the volunteers were recovering from normothermic anesthesia. Spontaneous tremor was recorded in 7/9 volunteers during recovery from hypothermic anesthesia. Tremor patterns resembled clonus or a mixture of clonus and shivering when end-tidal isoflurane was near 0.3%; at lower concentrations, tremor resembled normal shivering. Clonus could occasionally be induced by plantar flexion when end-tidal isoflurane concentrations were near 0.3% on both normothermic and hypothermic days.

We conclude that tremor during recovery from isoflurane anesthesia is triggered by hypothermia. The initial pattern, at isoflurane concentrations near 0.3%, is clonic or is a mixture of clonus and shivering. Tremor latter in recovery resembles normal shivering.

Reference:

. Sessler DI, *et al*: Anesthesiology 68:843-850, 1988

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TITLE: VECURONIUM 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 cynomologous monkeys (M fascicularis) (3.3-5.4 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 pollicus (elbow ground) using a Biologic Navigator (Filtration 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 ballon sensing device.

Vecuronium (0-0.7 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 +/- sem) at incremental degrees of block (fraction EMG remaining +/- 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 40% EMG depression with reduction below 50% of baseline values not until greater than 80% EMG reduction.

Discussion This study suggests that MMEP monitoring under ketamine anesthesia, as measured by the EMG of the oppens pollicus, is well maintained during neuromuscular block with infusion of vecuronium 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.

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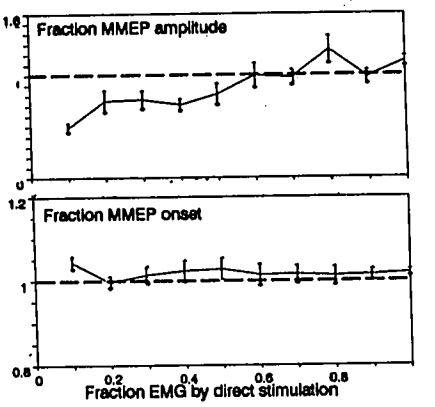

Plot of fraction MMEP amplitude and fraction MMEP onset versus fraction EMG by direct nerve stimulation