TITLE: THE EFFECT OF ETOMIDATE ON TRANSCRANIAL MAGNETIC-INDUCED MOTOR EVOKED POTENTIALS IN PRIMATES

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Initial observations with motor evoked potential (MEP), a modality for testing the efferent motor pathways, have shown that anesthetic agents may adversely interfere with response reproducibility[1]. Etomidate (ET) is known to enhance early cortical somatosensory evoked potential (SSEP)[2]. The present study examined the influence of etomidate (ET) hypnosis on MEPs.

MEPs were elicited in 12 cynomolgus monkeys (5-7 kg) by delivering pulsed extracranial magnetic fields via a circular coil (Novametrix Mag-Stim 200) placed over MEP scalp zone[1,3]. The evoked potentials were recorded from abductor pollicis brevis (APB) and abductor hallucis (AH) muscles contralateral to the site of stimulation using Nicolet Pathfinder unit. Anesthsia was conducted with intravenous ET; an initial intubating dose (0.5mg/kg) followed by 13 repeated doses (0.2mg/kg) every 6 to 11 minutes. Stimulation threshold and response latency/amplitude measurements were studied and compared to the baseline valves (pre-anesthesia) using ANOVA and Tukey's post-hoc test. EKG, BP, core and limb temperatures, arterial oxygen saturation, and end-tidal CO_2 were maintained within the normal range.

Replicable MEPs were consistently recorded under ET hypnosis. The MEP scalp topography was moderately reduced. There were significant ($p<0.05$) threshold elevation ($\geq$1.7mg/kg for ABP and $\geq$0.5mg/kg for AH) and latency delay ($\geq$0.5mg/kg for APB and $\geq$2.5mg/kg for AH) (Fig. 1).

The preservation of MEPs under ET hypnosis perhaps may reflect the drug excitatory action on the motor system as manifested by involuntary skeletal movements and enhanced EMG and epileptic EEG activities[4]. Likewise ketamine, a known motor excitatory agent, was found to maintain reliable MEPs[3]. We conclude that ET can be considered a desirable anesthetic drug to use while monitoring MEPs.

References:
1. Anesthesiology 68:A606, 1988.
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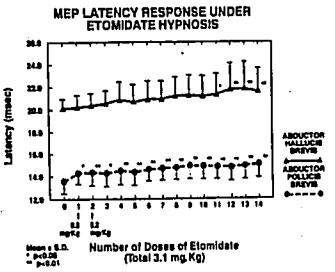

FIGURE 1

TITLE: EFFECTS OF ANESTHETIC INDUCTION DOSE OF MIDAZOLAM ON MOTOR EVOKED POTENTIALS IN PRIMATES

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Intraoperative monitoring of the ascending somatosensory (SEP) and descending motor (MEP) evoked potentials for neurosurgical, orthopedic and vascular operations are advantageous while the sensory and motor tracts are threatened. While midazolam (MDZ) induction dose produced minimal amplitude depression and latency delay of short-latency SEP[1], its effect on MEP is unknown. The present primate study examined the induced MEP alteration during hypnosis and recovery from an anesthetic dose of MDZ.

Extracranial pulsed magnetic fields, applied via a circular coil to the scalp MEP zone[2], was used to excite the motor cortex in 12 cynomolgus monkeys (5-7 kg). The evoked potentials were recorded from abductor pollicis brevis (APB) and anterior tibialis (AT) muscles contralateral to the site of stimulation. Following induction with MDZ (0.5mg/kg i.v.), the trachea was intubated orally and lungs were mechanically ventilated with FIO_2 0.5. EKG, BP, core and limb temperatures, end-tidal CO_2, and arterial oxygen saturation were regularly monitored and maintained within the normal range. MEPs were recorded sequentially during induction (post-intubation), hypnosis, awakening, emergence, and recovery from MDZ. Beside scalp MEP topography zone, the stimulation threshold, and latency/amplitude responses were studied and compared to the baseline values (pre-anesthesia) using ANOVA and Tukey's post-hoc test.

The scalp topography was markedly shrinked and coil demography was altered. MEPs were obtainable with difficulty during MDZ hypnosis. Stimulation threshold was significantly elevated and normalized during recovery while amplitude response was markedly suppressed and did not return to baseline value during apparant recovery ($p<0.01$) (Fig. 1).

In accord to our finding, sedative dose of MDZ (2-6mg) was found to cause marked suppression of MEP amplitude in humans[3]. The detrimental effect of MDZ on MEPs can be based on the central motor inhibitory and muscle relaxant actions of the drug[4]. We conclude that MDZ may produce prolonged and profound effect on MEPs and it's use perhaps should be discouraged while MEP monitoring.

References:
1. Anesth Analg 67:S65, 1988.
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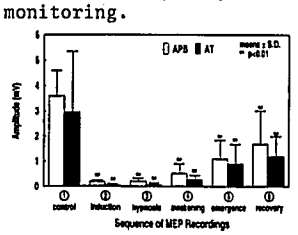

FIGURE 1