

Title: MODULATION OF THE MOTOR-EVOKED POTENTIAL -- INCREASED AMPLITUDE WITH PRIOR CEREBELLAR STIMULATION

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Introduction. Recently, the motor evoked potential (MEP) has been proposed as a monitor of the descending motor systems suitable for intraoperative use (1). Since the MEP is carried in dorso-lateral pathways, it may provide a monitor that complements the somato-sensory evoked potential (SEP), which is carried in the posterior cord.

Clinical use of the MEP is limited by two factors. First, since transcranial currents in excess of 20mA are required in humans, awake patients experience significant dental pain. Second, if SEPs and MEPs are used in the same patient, it is unknown if or how they interact. This is also a possible problem for the cerebellar evoked potential (CEP), another proposed monitor of long tract integrity (2).

One way to surmount these limitations is to modulate the MEP by prior stimulation of cerebellar or somato-sensory pathways, both of which are known to interact with motor systems at several levels (1). This might yield information on the interactions of the various evoked potentials. An increase in MEP amplitude following a conditioning stimulus might also allow lower currents to be used in awake patients yet still yield a recordable MEP. Others have reported increased MEP amplitude in humans following conditioning with SEPs (3). Our initial results of the effect of prior CEP stimulation on the MEP in cats are presented here.

Methods. After approval by our Animal Research Committee, six adult cats (male/female, 3-4 kg) were anesthetized with IM ketamine (100 mg), xylazine (5 mg), and atropine (0.1 mg). Femoral venous and arterial lines were placed. Endotracheal intubation followed IV pancuronium (0.4 mg). Anesthesia was maintained with IV ketamine (35 mg/hr), xylazine (3.5 mg/hr) and pancuronium for relaxation. Arterial pCO₂ was maintained between 28 and 35 mmHg and oral temperature above 36°C. Craniotomies were performed to expose the motor cortex bilaterally and the right cerebellum. Laminectomies were performed to expose the dura in the upper and lower thoracic regions.

MEPs were generated as previously described at a level just sufficient to elicit stable cord signals (Grass needles, anode-motor cortex, cathode-hard

palate, 6-12mA, 200 µsec duration, 4Hz) (1). The resultant signal was recorded from epidurally placed Grass needles (referenced to muscle), amplified (1- to 3000-Hz bandpass), averaged (500 repetitions) and stored using a Neuropack 8 Signal Averager (Nihon-Kohden). CEPs were applied via Grass needles in the right cerebellum with a constant voltage Grass stimulator. Modulated and control MEPs were recorded from both upper and lower cord sites. Before each modulated MEP, a control MEP was recorded. The modulated MEPs were preceded by a CEP stimulus at 4, 5, 6, 7, 8, 9, and 10 msec inter-stimulus intervals (ISI). This was done in a random fashion for a right CEP followed by right and left MEPs at each of these intervals.

The amplitude of the first negative deflection of the MEP was measured at both cord sites and averaged. The control and modulated MEP amplitudes were compared at each ISI for both ipsilateral and contralateral conditioning stimuli using the paired 2-tailed t-test (P<0.05 was considered significant).

Results. Figures 1 and 2 show the percentage increases in the MEP amplitude following a CEP. We observed no reproducible changes in the latencies of the MEP peak following a CEP.

Discussion. We observed a statistically significant increase at 8 ISIs tested. While an explanation for the mechanism of the CEP-MEP interaction will require further investigation, it is clear that the amplitude of the MEP can be increased with appropriate conditioning stimuli.

The facilitated MEP in humans may complement or replace the "wake-up test" in anesthetized patients for assessing motor tract integrity or may be useful for assessing recovery following motor tract injury. Also, since the facilitated MEP involves an afferent signal, trans-synaptic integration, and an efferent volley, it may be more useful as an electro-physiological measure of anesthetic depth than SEPs or EEGs.

References.

1. Levy WJ et al. Neurosurgery 15:287-302, 1984
2. Levy WJ et al. Neurosurgery 19:163-76, 1986
3. Deletis V et al. Neurosurgery 20:195-7, 1987

FIGURE 1. Contralateral Modulation of MEP

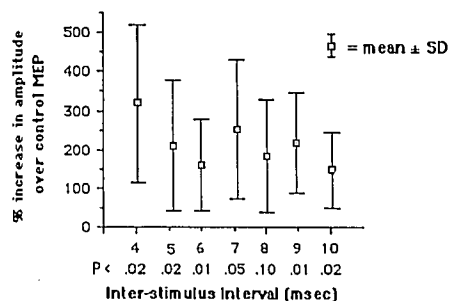


Figure 2. Ipsilateral Modulation of MEP

