Evidence That High Voltages of tcs-MEP Stimulation Can Bypass Ischemic Area in Brain: A Case Report

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Patient's Presentation

This 51 year old woman presented with left-sided retro-orbital headaches and 3 weeks of nausea/vomiting. A diagnostic angiogram confirmed an unruptured lobulated 7 x 4 mm left posterior communicating artery aneurysm. She was underwent a left craniotomy for clipping of the aneurysm. She was under general anesthesia using desflurane at 2.4% (0.4mac), propofol at 100µg/kg/min and remifentanil.

Intraoperative Neurophysiological Monitoring

SEP and MEP baselines were reset after craniotomy. Robust MEPs were recorded from right hand at 74 volts (20 V higher than thresholds, not high enough to get response from lower limb), 8 pulses, 75µs pulse width, 2ms ISI. The aneurysm was ruptured during dissecting around the arteries. SEP N20 dropped on right side significantly; MEPs also disappeared from right hand after temporal clip was placed on ICA two and three minutes later. MEPs were recordable from right hand and foot after increasing MEP stimulus from 74 to 112V during clipping on ICA. The SEPs and MEPs recovered to baseline four minutes later after temporary clip was removed. The patient moved all upper and lower limbs on postoperative day one.

Discussion

The brain surgery for clipping of aneurysm may carry the risk of cerebral ischemia during temporary occlusion of parent artery and accidentally clamping the perforating branches. Monitoring of SEPs has been used to detect early cortical ischemia during the surgery. MEPs have been proposed to detect deficits caused by subcortical ischemia by perforating vessel compromise, which may not detect by SEPs. Different parts of the corticospinal tract can be activated depending on the intensity of the MEP stimulus. There are 3 favorable points that are susceptible to depolarization on the corticospinal tract: cortex/subcortex with a low stimulation threshold; internal capsule with a moderate stimulus; and brainstem with a high voltages. If activation occurs at the internal capsule, motor cortex and superficial subcortical motor pathway ischemia, such as corona radiate infarcts can go undetected. Therefore, stimulation close to threshold may prevent excitation of corticospinal tract fibers deep within the white matter.

The presented case confirmed that deeper brain structures can be activated during cortical ischemia when MEP voltages of stimulation are increased to values that are much higher than threshold and can therefore bypass the ischemic area. Furthermore, MEPs may not be able to predict ischemia if the voltages are set up too high, as false negative results may occur.

References

Guo L, Gelb AW. The use of motor evoked potential monitoring during cerebral aneurysm surgery to predict pure motor deficits due to subcortical ischemia.Clinical Neurophysiology 2011;122: 648-655
Guo L, Gelb AW. False negative, muscle relaxants, and motor-evoked potentials. J Neurosurg Anesthesiol. 2012;23:64

SEPs were recorded from sterile needle electrodes placed at scalp sites C3'/Fz, C4'/Fz; Cz/Fz and C3'/C4' to record the primary cortical responses generated in the MCA and ACA territory. Stimulating electrodes were placed over each ulnar nerve, and each posterior tibial nerve. MEPs were recorded to transcranial electrical stimulation delivered to electrodes placed over C3' and C4'. EMG responses were recorded from needle electrodes placed bilaterally in abductor pollicis brevis in hands, and tibia anterior/extensor digitorum brevis in lower limbs.