

[NM-239] Transient loss of motor-evoked potentials associated with intravenous levetiracetam in a patient undergoing craniotomy for resection of astrocytoma

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Levetiracetam is a commonly used anticonvulsant often prescribed in the peri-operative period for patients with seizure activity related to an intracranial mass. The mechanism of action remains unknown, though studies suggest it inhibits pre-synaptic calcium channels (1). Oral levetiracetam has been shown to decrease cortical excitability measured with Transcranial Magnetic Stimulation (TMS), including a decrease in motor-evoked potential amplitude, an increase in resting motor threshold, and an increase in the cortical silent period duration (2). However, little data exists on the effects of IV levetiracetam on cortical excitability. We present a case of a child undergoing craniotomy for tumor with an abrupt decrease in transcranial electric motor evoked potentials (tcMEPs) after intraoperative IV levetiracetam administration.

A 12-year-old, 54-kg, female with fibrillary astrocytoma and persistent seizures presented for right-sided craniotomy and mass resection. After an uneventful induction, the patient was maintained on propofol and remifentanyl infusions to facilitate neurophysiological monitoring.

Baseline tcMEPs were satisfactory. However, ten minutes after the start of a levetiracetam infusion (fig., "Start"), a global amplitude decrease in tcMEPs was observed, despite near-baseline vital signs and minimal intracranial dissection at that point. The levetiracetam infusion was stopped, and after several minutes the tcMEPs returned to baseline levels (fig., "Pause").

TcMEPs remained stable throughout the remainder of surgery and skin closure. After the completion of surgery, the levetiracetam infusion was resumed (fig., "Resume"). A global tcMEP decrease was observed again despite near-baseline vital signs; the tcMEP decrease resolved after infusion cessation (fig., "Stop").

Intraoperative tcMEP monitoring has been utilized to reduce the risk of permanent neurologic deficit during craniotomy procedures. A decrease in tcMEP signals warrants investigation of potential causes including hypotension, hypocapnea, hypothermia, hypoxemia, and direct surgical injury. Anesthetic drugs (e.g. muscle relaxants, volatile agents, dexmedetomidine) can also attenuate tcMEP signals (3).

In summary, this case demonstrates an association between intravenous levetiracetam and subsequent decreases in intraoperative tcMEPs. Therefore, we recommend a multi-disciplinary discussion preoperatively regarding the timing and necessity of intravenous levetiracetam.

1. Fukuyama K, et al. Levetiracetam inhibits neurotransmitter release associated with CICR. *Neuroscience Letters* 2012; 518, 69-74

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3. Mahmoud M, Sadhasivam S, Sestokas AK, Samuels P, McAuliffe J. Loss of transcranial electric motor evoked potentials during pediatric spine surgery with dexmedetomidine. *Anesthesiology*. 2007; 106:393-6.


