Motor evoked potential mapping and monitoring by direct brainstem stimulation

Technical note

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Motor evoked potentials (MEPs) by direct brainstem stimulation were generated during 12 neurosurgical operations performed in five posterior fossa tumors, six vertebrobasilar aneurysms, and an arachnoid cyst. The anterior aspect of the brainstem was exposed using a subtemporal approach (in six cases), a presigmoid approach (one case), or a lateral suboccipital approach (five cases). A train of five monopolar 5 to 25 mA pulses was then applied, and MEPs were recorded from the extremities. Motor evoked potentials were recorded in all patients (four mappings and seven monitorings) except in a 12-year-old child who underwent surgery for a posterior cerebral artery aneurysm. Although he experienced postoperative motor palsy, the aneurysm ruptured before electrodes could be placed. Two patients with postoperative motor palsy, one with a clival meningioma and one with a basilar trunk aneurysm, had shown significant decreases in MEP amplitude and even complete disappearance of MEPs during intraoperative brainstem stimulation. Motor evoked potentials elicited by direct brainstem stimulation seem to be an accurate neurophysiological monitoring method during operations around the anterior and lateral aspects of the brainstem. (DOI: 10.3171/JNS-07/11/1053)

KEY WORDS • brainstem • glioma • intraoperative mapping • motor evoked potential

In posterior fossa surgery, auditory brainstem response monitoring seems essential and is widely used because it is easy to use and highly sensitive. Additionally, facial nerve stimulation, blink reflex testing, and monitoring for extraocular movements are occasionally performed during surgery in the lateral or posterior aspects of the brainstem.

Motor evoked potential monitoring is one of the most important intraoperative monitoring techniques in neurological surgery. There are two ways to conduct MEP monitoring: through direct motor cortex stimulation or transcranial high-voltage stimulation.

Although direct stimulation seems more precise and is safer than transcranial stimulation, it is usually impossible to expose the primary motor cortex during posterior fossa surgery. Transcranial MEPs are useful for monitoring the pyramidal tract during brainstem surgeries in which the motor area does not need to be exposed. Although transcranial MEPs are easy and useful, transcranial stimulation evokes multiple, large descending motor volleys, and the exact stimulated site is unclear. In general, transcranial MEPs are less accurate than direct MEPs and carry a greater likelihood of false-negative results.

Motor evoked potentials are very useful for intraoperative monitoring during posterior fossa surgery, especially in operations at the anterior or lateral aspect of the brainstem such as for gliomas, petroclival meningiomas, and cerebral aneurysms in vertebrobasilar systems. In the present study, we directly stimulated the pyramidal tract in the anterior aspect of the brainstem using direct MEPs and recorded them during posterior fossa surgery.

Materials and Methods

Since July 2002, we have used direct brainstem MEPs during 12 neurosurgical surgeries (Table 1). These included five operations for posterior fossa tumors (three posterior fossa meningiomas and two brainstem gliomas), six for vertebrobasilar aneurysms (two basilar bifurcations, a posterior cerebral artery, a basilar trunk, a vertebral artery union, and a vertebral artery–posterior inferior cerebellar artery), and one for an arachnoid cyst situated just behind the clivus. The anterior aspect of the brainstem was exposed via a subtemporal approach (in six cases), a presigmoid approach (one case), or a lateral suboccipital ap-
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<table>
<thead>
<tr>
<th>Case No.</th>
<th>Age (yrs), Sex</th>
<th>Diagnosis</th>
<th>Approach</th>
<th>Preop Motor Palsy</th>
<th>Stimulation</th>
<th>Latency (msec)</th>
<th>CRAI</th>
<th>Postop Motor Palsy</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>25, M</td>
<td>lt brainstem glioma</td>
<td>lt subtemporal</td>
<td>+</td>
<td>bipolar</td>
<td>25</td>
<td>lt UE, LE</td>
<td>23.2</td>
</tr>
<tr>
<td>2</td>
<td>78, M</td>
<td>SAH, basilar bifurcation aneurysm</td>
<td>rt subtemporal</td>
<td>–</td>
<td>bipolar</td>
<td>10</td>
<td>lt UE, LE</td>
<td>23.1</td>
</tr>
<tr>
<td>3</td>
<td>73, F</td>
<td>clival meningioma</td>
<td>lt subtemporal</td>
<td>+ (mild)</td>
<td>stripe E</td>
<td>5</td>
<td>rt UE, LE</td>
<td>18.4</td>
</tr>
<tr>
<td>4</td>
<td>51, M</td>
<td>clival arachnoid cyst</td>
<td>lt lat suboccip</td>
<td>–</td>
<td>bipolar</td>
<td>10</td>
<td>rt UE, LE</td>
<td>22.1</td>
</tr>
<tr>
<td>5</td>
<td>52, F</td>
<td>rt brainstem glioma</td>
<td>rt presigmoid</td>
<td>–</td>
<td>bipolar</td>
<td>25</td>
<td>lt LE</td>
<td>–</td>
</tr>
<tr>
<td>6</td>
<td>45, F</td>
<td>SAH, VA aneurysm</td>
<td>lt lat suboccip</td>
<td>–</td>
<td>stripe E</td>
<td>5</td>
<td>rt UE, LE</td>
<td>20.5</td>
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<tr>
<td>7</td>
<td>79, F</td>
<td>lt CPA meningioma</td>
<td>lt lat suboccip</td>
<td>–</td>
<td>bipolar</td>
<td>3</td>
<td>rt UE, LE</td>
<td>24.1</td>
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<tr>
<td>8</td>
<td>48, M</td>
<td>lt VA-PICA aneurysm</td>
<td>lt lat suboccip</td>
<td>–</td>
<td>stripe E</td>
<td>7</td>
<td>rt/U/E</td>
<td>22.6</td>
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<td>9</td>
<td>68, F</td>
<td>basilar bifurcation aneurysm</td>
<td>rt subtemporal</td>
<td>–</td>
<td>silver ball</td>
<td>10</td>
<td>lt UE, LE</td>
<td>22.3</td>
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<td>10</td>
<td>80, F</td>
<td>basilar trunk aneurysm</td>
<td>rt subtemporal</td>
<td>–</td>
<td>silver ball</td>
<td>10</td>
<td>lt UE</td>
<td>19.3</td>
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<tr>
<td>11</td>
<td>12, M</td>
<td>rt PCA aneurysm</td>
<td>rt subtemporal</td>
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<td>10</td>
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<td>12</td>
<td>47, F</td>
<td>rt tentorial meningioma</td>
<td>rt lat suboccip</td>
<td>–</td>
<td>silver ball</td>
<td>5</td>
<td>lt LE</td>
<td>–</td>
</tr>
</tbody>
</table>

* CPA = cerebellopontine angle; CRAI = compensated RAI; E = electrode; LE = lower extremity; PCA = posterior cerebral artery; PICA = posterior inferior cerebellar artery; SAH = subarachnoid hemorrhage; suboccip = suboccipital; UE = upper extremity; VA = vertebral artery; + = present; – = absent.

Results

The results of the 12 operations performed with direct brainstem MEPs are summarized in Table 1. Motor evoked potentials were recorded in all patients (four mappings and seven monitorings) regardless of operative approach, except in the 12-year-old boy who underwent surgery for a posterior cerebral artery aneurysm with premature rupture who manifested postoperative motor palsy. In this patient, the aneurysm ruptured before the electrodes could be placed. The mean ± standard deviation of the latency period length after direct brainstem MEP stimulation was 21.7 ± 1.8 msec in the upper extremity and 40.1 ± 4.5 msec in the lower extremity. These values are not significantly different from the previously described latency periods after MEPs elicited by direct motor cortex or transcranial stimulation.

In the seven most recent direct brainstem MEP measurements, the RAI could be calculated before and after the operative procedure and was compensated by the amplitude of the CMAP in eight limbs of seven patients. Two patients—one with a clival meningioma and one with a basilar trunk aneurysm, both with postoperatively worsened motor palsy—had shown a significant decrease and disappearance of the amplitude of direct brainstem MEPs. The compensated RAI was 0.16 and 0 in these patients, respectively. In contrast, in six limbs in five patients with no progression of motor palsy, compensated RAIs were greater than 0.2.

Biopsy sampling of brainstem glioma tissue using MEP mapping by direct peduncular stimulation has been de-
Surgery was performed via a left subtemporal transtentorial approach under MEP mapping by direct peduncular stimulation. The lateral aspect of the midbrain was exposed, and a train of five bipolar, 25 mA pulses was applied and MEPs recorded from the extremities. Motor evoked potentials were only recorded from the left extremities even with left cerebral peduncular stimulation. Partial resection of the tumor was safely performed, with only slight temporary neurological worsening.

A 73-year-old woman (Case 3) had suffered from right hemiparesis due to a clival meningioma that recurred after two operations. On Gd-enhanced T1-weighted magnetic resonance imaging the tumor was noted to have compressed the lower pons (Fig. 2). The operation was performed through the left subtemporal route. Direct brainstem MEP recording was performed at the upper pons distal to the tumor by 5-mA stimulations with a bipolar apparatus for mapping followed by the use of subdural electrodes for monitoring. As resection of the tumor advanced, the MEP amplitude gradually decreased (Fig. 3). Finally, the compensated RAI decreased to less than 0.2, and attempts at total resection were stopped. The patient's right hemiparesis worsened postoperatively.

**Discussion**

During surgeries around the brainstem, facial nerve mapping and brainstem auditory evoked potential monitoring are commonly used, especially in approaches through the floor of the fourth ventricle. Otherwise, in operations in lesions located near the cerebral peduncle or basal pons, and vertebrobasilar arteries, pyramidal tract function is the greatest concern for surgeons. Since it is usually impossible to expose the motor cortex during posterior fossa surgery, transcranial high-voltage stimulation is the only method for MEPs except for the direct brainstem stimulation described in the present study.

Transcranial stimulation seems to evoke multiple large descending motor volleys. It has been reported that the brainstem was stimulated in high-voltage transcranial MEPs based on a comparison of the latencies of transcranial MEPs and direct MEPs. Because the stimulation of transcranial MEPs is much stronger than the stimulation applied in direct MEPs, a comparison of the latencies is not meaningful. A stronger stimulation induces a shorter latency period in MEPs. Although the exact site that is stimulated in transcranial MEPs is unclear, we think that the site is closer to the motor cortex than to the brainstem. In our experience, high-voltage (600 V) stimulation can reach deeper sites, including the brainstem, whereas relatively low-voltage (300 to 400 V) stimulation after craniotomy may be localized near the motor cortex. Transcranial MEP is useful for monitoring the pyramidal tract during brainstem surgery in which the motor area does not need to be exposed.

On the other hand, direct MEPs induced by motor area stimulation are widely used during brain tumor resection and aneurysm surgery. Indeed, transcranial MEPs have become less invasive and more accurate by decreasing the stimulation intensity, but the use of direct MEPs is apparently safer and more accurate than using transcranial MEPs. The pyramidal tract, including the corona radiata and posterior limb of the internal capsule, may also be stimulated through the wall of the tumor cavity. In contrast, little information is available on direct stimulation of the corticospinal tract in brainstem surgery. Authors of a study of intraoperative direct brainstem stimulation after temporal lobectomy for epilepsy found that a monophasic cathodal stimulus at 2 to 10 mA was effective. This first successful recording of MEPs by direct brainstem stimulation was not used for mapping or monitoring for pyramidal tract preservation. Percutaneous electrical stimulation of corticospinal pathways at the level of the pyramidal decussation has also been reported. Our method for direct intraoperative stimulation of the pyramidal tract seems to be the first report of direct brainstem MEPs for intraoperative monitoring or mapping.

Recently, MEP monitoring has been able to be performed more reliably because of progress in train stimulation and the use of propofol anesthesia. However, it is...
still difficult to evaluate changes in wave patterns because MEPS are strongly affected by anesthetic agents, especially muscle relaxants. The disadvantages of transcranial MEPS are body movement with high-voltage stimulation and the previously mentioned uncertainty regarding the exact stimulated site. Our method of relatively low-voltage (300 to 400 V) stimulation in transcranial MEPS limits body movement. Although our method of transcranial MEPS with low-voltage stimulation seems safe and may provide more localized stimulation, it is not possible to compare the amplitudes in contralateral limbs, as we have previously reported. Compound MAP measurements can identify changes in MEPS due to differences in the degree of neuromuscular blockage, and compensation of MEP by CMAPs makes the proper evaluation of transcranial MEPS possible even in relatively low-voltage transcranial MEPS.

Some authors have reported that an RAI of less than 0.5 was significant. As previously reported, we think that the threshold of compensated RAI for motor palsy in our transcranial MEPS compensated by CMAP is 0.2. In direct brainstem MEPS elicited in two limbs in two patients with postoperatively progressive motor paresis, the compensated RAI was less than 0.2. Compensated RAI in the other six limbs of five patients without postoperative motor palsy was greater than 0.2. These results suggest that compensation of MEP monitoring with CMAPs is also useful in direct brainstem MEPS, and that the threshold of the compensated RAI for postoperative motor palsy is 0.2 for direct brainstem MEPS.

Conclusions

Motor-evoked potentials by direct brainstem stimulation is an accurate neurophysiological monitoring method during operations around the anterior and lateral aspects of the brainstem. Compensation of MEP monitoring by CMAPs is also useful in direct brainstem MEPS, and the threshold of compensated RAI for motor palsy is 0.2 for direct brainstem MEPS.

References

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