# The Role of Intraoperative Monitoring of Oculomotor and Trochlear Nuclei – Safe Entry Zone to Tegmental Lesions

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Original Article



## Abstract

**Objective:** A safe entry zone to tegmental lesions was identified based on intraoperative electrophysiological findings, the compound muscle action potentials (CMAP) from the extraocular muscles, and anatomic considerations. This entry zone is bordered caudally by the intramesencephalic path of the trochlear, laterally by the spinothalamic tract, and rostrally by the caudal margin of the brachium of the superior colliculus. **Methods:** Four intrinsic midbrain lesions were operated upon via the safe entry zone using the infratentorial paramedian supracerebellar approach. All lesions involved the tegmentum and included an anaplastic astrocytoma, a metastatic brain tumor, a radiation necrosis, and a cavernous angioma. CMAP were bilaterally monitored from the inferior recti (for oculomotor function) and superior oblique (for trochlear nerve function) muscles. **Results:** In three of four cases, CMAP related to the oculomotor nerve were obtained upon stimulation at the cavity wall after removal of the tumor. Stimulation at the surface of the quadrigeminal plate, however, did not cause any CMAP response. Using this monitoring as an indicator, the lesions were totally removed. **Conclusions:** In the surgery of tegmental lesions, CMAP monitoring from extraocular muscles is particularly helpful to prevent damage to crucial neural structures during removal of intrinsic lesions, but less so to select the site of the medullary incision. The approach via the lateral part of the colliculi is considered to be a safe route to approach the tegmental lesions.

## Key words

Intraoperative monitoring · oculomotor complex · trochlear nerve

## Introduction

Intraoperative monitoring of cranial nerves has become indispensable to preserve cranial nerve functions in skull base surgery [1,2]. This technique has also been applied to the direct surgery of pontine lesions. In combination with precise anatomic exploration, this technique has minimized operative morbidity, especially at the floor of the 4th ventricle [3–6]. The use of intraoperative monitoring during midbrain surgery, however, is still under debate. In this publication, we present our experience of electrophysiological monitoring during midbrain surgery, and describe a safe entry zone to approach dorsal tegmental lesions.

## Patients and Methods

### Anesthesia and placement of the recording electrodes

General anesthesia was induced with thiopental sodium, and the fast-acting muscle relaxant pancronium bromide was administered for orotrachial intubation. Isoflurane (0.1–1.0 vol%) was used to maintain balanced anesthesia, while patients breathed 30% oxygen and 70% nitrous oxide without further muscle relaxation. The recording platinum needle electrodes were placed percutaneously in the inferior recti and superior oblique muscles on both sides. The reference electrode was placed on the ipsilateral shoulder, and the ground electrode was placed on the sternum.

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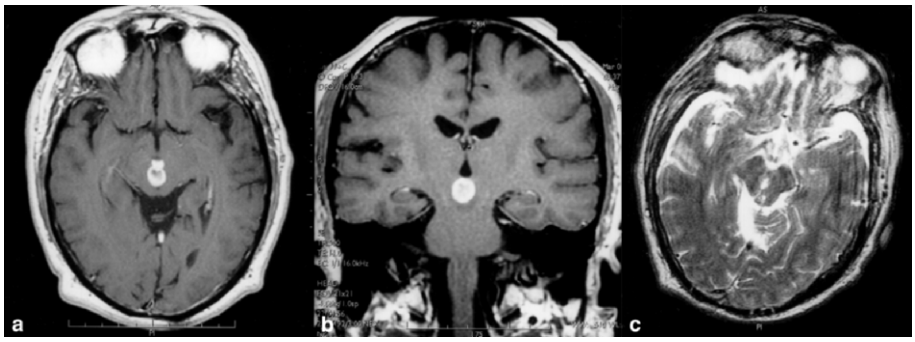


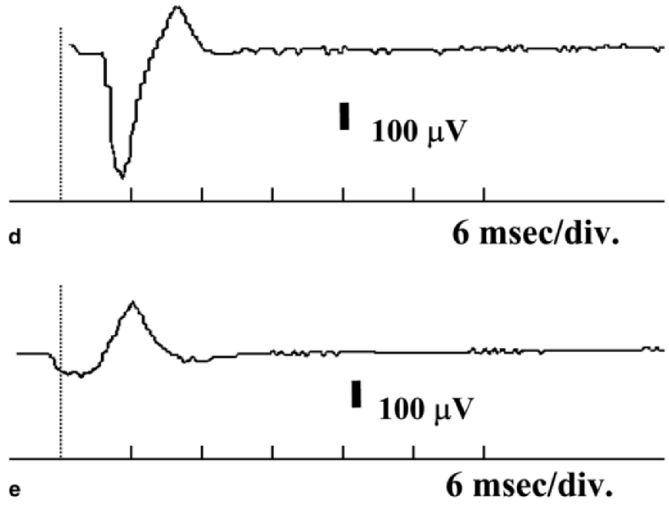


Fig. **1** Case 1: a 31-year-old male. Preoperative axial (**a**) and coronal (**b**) enhanced MRI scans revealing a cystic enhanced mass 12 mm in diameter in the tegmentum. Postoperative axial $T_2$-weighted image (**c**) showing the surgical corridor. CMAP was obtained from the right inferior rectus muscle (**d**) when the right rostral border of the tumor was stimulated with 0.30 mA intensity. CMAP from the left superior oblique muscles (**e**) was elicited by stimulation with 0.45 mA intensity on the right caudal border of the tumor.





**Electrical stimulation and recording**

A monopolar stimulation electrode (bare tip) was used to elicit responses at the brainstem. Electrical stimulation was at constant current with rectangular pulses of 0.2 msec duration and a repetition rate of 3 Hz. The intensity of the stimulus varied from 0.15 up to 0.5 mA. The apparatus for both electrical stimulation and analysis of CMAP was a Nerve Integrity Monitor type 2 (NIM-2, manufactured by XOMED, Florida, USA). Initial electrical stimulations were performed at the surface of the tectal plate. During surgical removal of the lesions, especially during manipulation at the boundaries between tumor and neural tissue, electrical stimulations were repeated.

**Illustrative cases**

*Case 1:* A 51-year-old man presented with a 5-month history of a progressive oculomotor paresis on the right side including an anisocoria (wider pupil on the right side) and intermittent headache. Neurological examination at admission revealed, in addition to the right-sided external and internal oculomotor palsy, bradydiadochokinesis of the left hand. MRI showed a cystic gadolinium-enhanced mass lesion (diameter: 12 mm) in the tegmentum of the midbrain (Fig. **1a** and **b**). The operation was performed through a right paramedian infratentorial supracerebellar approach. A 4 mm longitudinal medullary incision was made 5 mm laterally from midline in the recess between superior and inferior colliculus, after confirming that no CMAP could be elicited by surface stimulation. The capsule of the tumor was recognized 3 mm below the surface. Xanthochromic liquid was aspirated and solid material was removed. During the removal of the mass, electrical stimulation was repeatedly applied at the cavity wall, since the border between tumorous and normal tissue was not distinct. When the neural tissue adjacent to the right rostral border of the tumor was stimulated with 0.30 mA intensity, CMAP was recorded from the right inferior rectus muscle (Fig. **1d**). Similarly, CMAP was recorded from the left inferior rectus muscle by the stimulation of neural tissue at the left rostral border of the tumor (Fig. **1e**). Then, the lower portion of the tumor was removed. Its caudal border was detected by eliciting CMAP from the superior oblique muscles on both sides. Finally, the tumor was completely removed by avoiding areas where CMAP could be evoked (Fig. **1c**). The pathological diagnosis was adenocarcinoma. Postoperatively, the patient developed transient tetraparesis, which was completely reversible within 4 weeks, but required a ventriculo-peritoneal shunt. The right-sided ptosis had improved, but there was an additional slight internal oculomotor palsy on the left side. Further radiological examination failed to detect the primary lesion. The patient went back home 7 weeks after the operation.

*Case 2:* A 7-year-old boy developed sudden headache, right ptosis and left hemiparesis, and was admitted our hospital. He had already been operated on twice for a right thalamic hemorrhage in 1991 and a midbrain hemorrhage in 1993. Both times the diagnosis was cavernous angioma. After the second operation, the patient had recovered well, and only a slight right oculomotor palsy and left upper limb dominant hemiparesis (Weber's syndrome) had remained. A neurological examination at admission revealed an increase of the known right-sided oculomotor palsy. Consciousness was not affected. MRI showed two hemorrhagic lesions, one in the right tegmentum of midbrain with low inten-

sity rim in $T_1$- and $T_2$-weighted images (diameter: 16 mm), the other in left frontal lobe (Fig. **2a** and **b**). Following removal of the left frontal lobe lesion, the right dorsal part of the midbrain was exposed through a paramedian, infratentorial-supracerebellar approach. First, electrical stimulations were applied to the surface of the quadrigeminal plate, but no CMAPs were recorded. A 4 mm longitudinal medullary incision was made 5 mm laterally from midline in the recess between superior and inferior colliculus. The capsule of a hematoma was recognized 1 mm below the surface. The small vessels in the medial wall of the hematoma were not removed although no CMAP was recorded with electrical stimulation in this part. The hematoma was totally removed (Fig. **2c**). Stimulation of the wall of the hematoma did not elicit CMAP in the recorded eye muscles. The pathological diagnosis of both lesions was cavernous angioma. Postoperatively, the right oculomotor palsy was slightly improved. The patient was discharged from the hospital 10 days after the operation.



## Discussion

Intraoperative electrophysiological monitoring of the ocular motor neurons in the midbrain (nuclei and axons of the IIIrd and IVth nerves) was successfully performed in four patients who underwent surgery for midbrain lesions. Stimulation using a monopolar electrode with a current intensity up to 0.5 mA was safely carried out, and proved to be helpful in avoiding postoperative morbidity. CMAP from extraocular muscles could be obtained on the stimulation of the cavity wall after the removal of the lesion, except in case 2 where the wall probably consisted of small vessels. Stimulation from the surface of the quadrigeminal plate, the posterior commissure and the floor of the posterior third ventricle did not elicit any eye muscle response. Our results indicate that the role of electrophysiological monitoring in surgery for midbrain lesions is slightly different from that for pontine lesions. In the context of surgery on midbrain lesions, CMAP does not play a role in identifying the medullary incision, but is useful for the removal of the tumor. The safe entry zone, however, has to be identified based on the precise anatomic considerations and preoperative evaluation of the tumor's location.

### Anatomic considerations for safe entry zone

The midbrain is the shortest segment of the brainstem; its longitudinal dimension is less than 2 cm. Like in other parts of the brainstem, important neural structures are densely contained. The dorsal surface of the midbrain consists of the four colliculi situated caudally to the posterior commissure and rostrally to the superior medullary velum. The brachium of the inferior colliculus ascends to reach the medial geniculate body. The brachium of superior colliculus runs underneath the brachium of inferior colliculus to the lateral geniculate body. The dorsal part, the tectum, and central part, the tegmentum, contain important relay nuclei of the auditory and the ocular motor systems. The trochlear and oculomotor nuclei lie in the dorsomedial part of the tegmentum. When tegmental lesions are surgically approached from the dorsal surface of the midbrain, these important structures should be given thorough consideration.

*Caudal midbrain:* The inferior colliculi receive input from every nuclear group within the auditory brain stem. They are the secondary relay structures of the ascending auditory system, and are densely connected with each other by the commissure of the inferior colliculus. The auditory nuclei, including cochlear nuclei, superior olivary complex and nuclei of the lateral lemniscus, have bilateral ascending projections. This might be the reason why Bognar et al. and Kaku et al. could resect the inferior colliculus on one side without adverse effects on hearing [7, 8].

The trochlear nuclei lie in the ventral region of the central gray matter, just below the rostral part of the inferior colliculus. The respective fibers descend dorsolaterally around the central gray matter at the level of the caudal part of the inferior colliculus, and reach the superior medullary velum, where they decussate to emerge at the lateral side of the frenulum veli [9]. Therefore, a unilateral lesion in the area of the caudal part of the inferior colliculus produces a contralateral trochlear nerve palsy. In case 1, electric stimulation of the caudal cavity wall in this area elicited CMAP of the superior oblique muscle, which was probably due to the activation of intrinsic trochlear nerve fibers. When the inferior colliculus is chosen for an approach to a tegmental lesion, one should restrict possible damage to the lateral and rostral parts of inferior colliculus to spare the commissure of the inferior colliculus and the trochlear nerve fibers.

*Rostral midbrain:* The superior colliculus (SC) is an important structure in the control of visual fixation and the generation of saccadic eye movements. This is reflected in the anatomic connections to other structures with oculomotor functions. The deeper layers of the SC project mainly to premotor structures of the ocular motor system, such as the paramedian pontine reticular formation (PPRF) and the rostral interstitial nucleus of the medial longitudinal fasciculus (riMLF). By superficial electrical stimulation, however, no eye movements can be evoked [10], because the superficial layers of SC connect to the visual system by

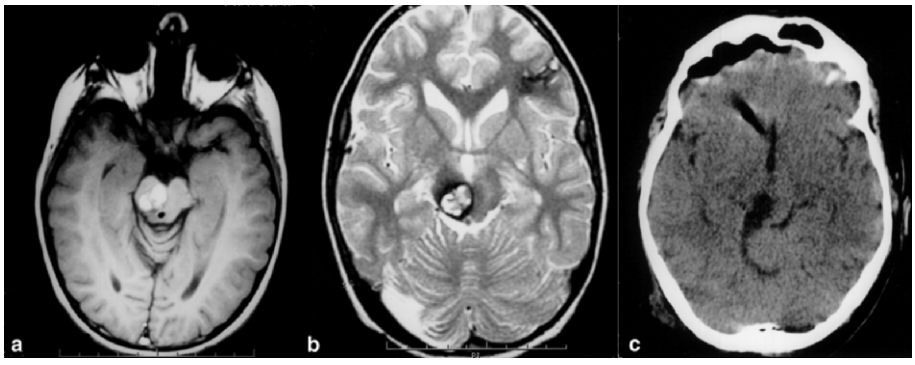


Fig. **2** Case 2: a 7-year-old boy. Preoperative axial $T_1$-weighted image (**a**) and $T_2$-weighted image (**b**), revealing hematomas with low intensity rim in the right tegmentum and left frontal lobe. Postoperative CT (**c**) showing the surgical corridor.

projection to the thalamus and the lateral geniculate nuclei. This lack of ocular motor responses is in agreement with our findings. More recent studies have revealed that the superior colliculus fixation neurons which are located in the rostral pole of the SC control saccades by suppressing or activating omnipause neurons which are located in the nucleus raphe interpositous [11,12].

The rostral interstitial nucleus of the MLF (riMLF) and the accessory oculomotor nuclei are closely connected with the oculomotor complex. The accessory oculomotor nuclei comprise the interstitial nucleus of Cajal (INC), the nucleus of Darkschewitsch and the nuclei of the posterior commissure. The INC is situated ventrolaterally to the MLF at the mesodiencephalic junction, and plays an important role in the integration of eye-velocity signals into eye-position signals and in eye-head coordination [13,14]. The riMLF lies adjacent to the rostral border of the INC and is wing-shaped. This nucleus contains neurons with vertical (upward, downward) and torsional (ipsitorsional) eye movement on-directions. In the monkey, unilateral lesions are characterized by a loss of all rapid eye movements with an ipsitorsional component, and downward movements are slowed. In bilateral lesions all vertical and torsional rapid eye movements are abolished [15]. Similar deficits can be seen after a lesion of the INC [16]. This anatomic location of these crucial structures related to vertical eye movements suggests that dorsal tegmental lesions should not be approached from the rostral part of the SC, but rather from the caudal and lateral parts.

*Safe entry zone for tegmental lesion:* In order to minimize the postoperative morbidity, we identified a safe entry zone from the dorsolateral mesencephalon on the basis of our electrophysiological findings and anatomic considerations. This "inferior brachial triangle" (Fig. **3**) is bordered caudally by the trochlear nerve fibers inside the brainstem, laterally by the spinothalamic tract, and rostrally by the caudal margin of the brachium of the superior colliculus. Although a medullary incision in this triangle compromises unilateral ascending projections from the inferior colliculus, this approach preserves the trochlear tract, the connection between the inferior colliculi on both sides, the superior colliculus, the accessory oculomotor nuclei and the oculomotor complex.

The paramedian infratentorial supracerebellar approach has advantages to access lateral parts of the quadrigeminal plate [17–19], and therefore is a suitable approach for the "inferior brachial triangle".

**Midbrain monitoring**

Intraoperative monitoring of motor nuclei has been applied and has established its importance in surgery through the rhomboid fossa [6,20]. The facial colliculus is the main target in the mapping of the 4th ventricle floor, because the abducens nuclei and facial nerve tracts lie just beneath. In the midbrain, however, it is difficult to identify the oculomotor complex and the trochlear nuclei from the surface of the midbrain, since they are embedded in the ventral border of the periaqueductal gray matter. Therefore, electrophysiological monitoring in the midbrain should mainly be applied during the removal of lesions rather than to decide on the location of the medullary incision.

There are two reasons why we used lower current for stimulation. One is for the purpose of avoiding neural damage by the electrical stimulation itself. Neuronal damages due to electrical stimulation were shown in animal models. Asanuma and Arnold showed that currents above 0.04 mA (at 0.2 ms duration) and up to 0.08 mA transiently damaged pyramidal tract neurons [21,22]. It is considered that a higher current can damage neural tissue, and also can give false negative results, although the character of the current is surely important.

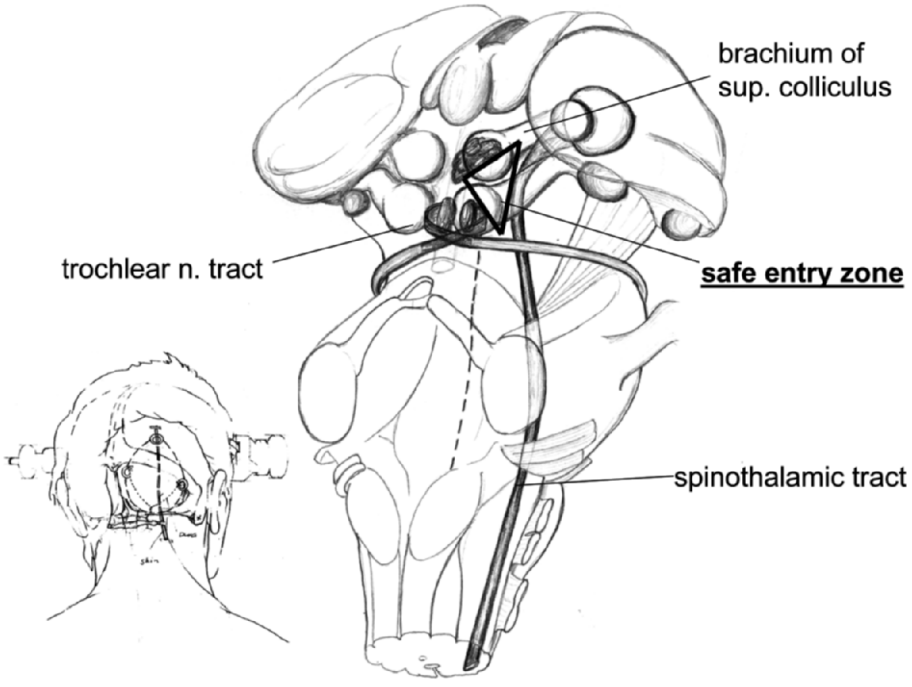


Fig. **3** Schematic drawing of the dorsolateral midbrain anatomy and the safe entry zone.

The other reason is for the purpose of reducing the false positive results of monitoring. At the brainstem, the threshold intensity to obtain the CMAPs required is only 0.05 to 0.20 mA when the stimulation probe is directly applied to motor nuclei or tracts [20,23]. The current-distance estimates of several neurons have been shown in animal models [24,25]. According to these experiments, the relation between the current intensity that evoked an action potential from neurons and the distance from stimulation probe is: current = K (distance)$^2$ where K is the current-distant constant, and it can range from 0.1 to 4.0 mA/mm$^2$ depending on the neural elements. The current intensity necessary to activate a neuron 1 mm away from the electrode tip would be 0.1 mA for a low and 4.0 mA for a high threshold neuron. For example, when a 1 mA current is applied to neural tissue, a low threshold neuron more than 3 mm away from the electrode would be activated. We therefore prefer low currents repeatedly for the electrical stimulation in order to reduce false positive results of the monitoring.

Recently, Sekiya et al. reported the usefulness of oculomotor nuclei monitoring to avoid surgical injury to ocular motor functions during midbrain surgery [26]. This study corresponds well with our results. Our aim of ocular motor monitoring in the midbrain, however, is not only to preserve ocular motor function but also to support surgical orientation. The midbrain is tightly packed with many important nuclei and neural tracts, for instance, the central tegmental tract which connects the reticular formation to the cerebral cortex is crucial for consciousness. This tract is located just lateral to the part of the oculomotor nucleus that contains neurons innervating the inferior rectus muscle. To confirm the location of the oculomotor nucleus is hence important to avert surgical damages to other crucial structures.



## Conclusion

In conclusion, anatomic considerations and our results indicate that the lateral part of the tectal plate, the “inferior brachial triangle”, is a safer entry zone to tegmental lesions. Furthermore, the intraoperative monitoring of CMAP from extraocular muscles using lower current stimulation is useful to preserve ocular motor function as well as the function of other neural structures.

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