Preliminary results of fully endoscopic microvascular decompression with intraoperative neurophysiological monitoring

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Abstract

Introduction: Endoscopic microvascular decompression (MVD) with intraoperative neurophysiological monitoring (IONM) is increasingly being adopted as the standard approach for treating neurovascular compression syndromes in the cerebellopontine angle. This study aims to evaluate the initial outcomes of the first five cases of endoscopic MVD with IONM.

Materials and Methods: This prospective study included the first five cases of endoscopic MVD with IONM performed for trigeminal neuralgia and hemifacial spasm at the Department of Neurosurgery – Spine surgery, Hanoi Medical University Hospital between September 2024 and February 2025. The neurophysiological monitoring techniques used included brainstem auditory evoked potentials (BAEP), brainstem trigeminal evoked potentials (BTEP), lateral spread response (LSR), and Zhong-Lee response (ZLR). Treatment outcomes for trigeminal neuralgia were assessed using the Barrow Neurological Institute (BNI) pain intensity scale, while hemifacial spasm outcomes were evaluated using the Hemifacial Spasm (HFS) score.

Results: Three cases of hemifacial spasm had a preoperative mean HFS clinical score of 12.67, which improved to 0 postoperatively, with a preoperative quality of life (QoL) of 73.3%, improving to 0% postoperatively. All three cases involved compression by the anterior inferior cerebellar artery (AICA) and exhibited intraoperative LSR, which disappeared after decompression, accompanied by a positive Zhong-Lee response. Two cases of medically refractory trigeminal neuralgia were classified as BNI grade IV and V preoperatively, with immediate postoperative improvement to BNI grade I. The compressing structures were the superior cerebellar artery (SCA) and Dandy's vein. In both cases, BTEP waves in the V2 and V3 branches emerged following decompression. Although all five cases demonstrated intraoperative BAEP amplitude reduction, postoperative hearing function was preserved.

Conclusions: Endoscopic MVD with IONM enhances surgical success rates and reduces postoperative hearing loss.

Keywords: trigeminal neuralgia, hemifacial spasm, endoscopic microvascular decompression, Jannetta procedure, intraoperative neurophysiological monitoring.

Introduction

Trigeminal neuralgia (TN) and hemifacial spasm (HFS) are neurological disorders caused by vascular compression of cranial nerves. In trigeminal neuralgia, vascular compression of the trigeminal nerve (cranial nerve V) leads to severe, paroxysmal facial pain. In contrast, hemifacial spasm results from compression of the facial nerve (cranial nerve VII), causing involuntary, unilateral facial muscle contractions. Microvascular decompression (MVD), introduced by Peter Jannetta in 1967, marked a breakthrough in the treatment of both conditions. This surgical technique aims to relieve nerve compression by repositioning or insulating the offending vessel, thereby alleviating symptoms and restoring normal nerve function. MVD is considered the gold standard treatment due to its high success rate and low recurrence rates, with studies reporting an 80–90% success rate for both TN and HFS.

Intraoperative neurophysiological monitoring (IONM) is a vital adjunct in neurosurgery, designed to enhance patient safety by providing real-time feedback on the functional integrity of neural structures during surgical procedures. Its primary indications include surgeries with a significant risk of neurological injury, such as spinal deformity corrections, tumor resections near eloquent brain areas, and vascular surgeries involving the brainstem or spinal cord. However, IONM is not universally applicable. Contraindications may include patients with certain implanted electronic devices, severe pre-existing neurological deficits that preclude baseline monitoring, or situations where neuromonitoring may not provide additional benefit. In MVD, however, to optimize surgical outcomes and minimize complications, intraoperative neurophysiological monitoring (IONM) has gained increasing attention. Recent research has focused on refining IONM techniques to enhance the accuracy of real-time nerve function assessment during MVD. Montano et al. (2024) developed an intraoperative method for stimulating the compressing artery to evaluate trigeminal nerve responses, demonstrating that this technique could serve as a diagnostic tool for assessing decompression effectiveness. Additionally, IONM plays a crucial role in monitoring the function of the vestibulocochlear nerve (cranial nerve VIII) to prevent postoperative hearing loss. Polo et al. (2004) demonstrated that the use of brainstem auditory evoked potentials (BAEPs) significantly reduces the risk of hearing impairment following MVD. Furthermore, Wang et al. (2019) proposed integrating multiple monitoring techniques, including electromyography (EMG) and direct nerve stimulation, to optimize surgical outcomes and reduce complications.

In Vietnam, the Jannetta procedure has been widely implemented across neurosurgical centers, yielding promising results. With the advancement of minimally invasive surgical techniques, the incorporation of endoscopy in MVD has become increasingly prevalent in recent

years, progressing from endoscopic-assisted approaches to fully endoscopic procedures. Given this trend, we conducted this study to evaluate the initial outcomes of fully endoscopic microvascular decompression with intraoperative neurophysiological monitoring in the treatment of trigeminal neuralgia and hemifacial spasm at Hanoi Medical University Hospital.

Materials and methods

Study Subjects

This study reports the outcomes of the first five cases of fully endoscopic microvascular decompression (MVD) with intraoperative neurophysiological monitoring (IONM) performed for the treatment of trigeminal neuralgia and hemifacial spasm at the Department of Neurosurgery – Spine Surgery, Hanoi Medical University Hospital, from September 2024 to February 2025.

Study Design

This was a prospective clinical trial.

Inclusion Criteria

Patients were eligible for the study if they met the following criteria:

Diagnosed with trigeminal neuralgia or hemifacial spasm with a surgical indication.

Preoperative magnetic resonance imaging (MRI) excluded secondary causes of trigeminal neuralgia or hemifacial spasm, such as brain tumors, epidermoid cysts, cranial nerve schwannomas, arteriovenous malformations (AVMs), or dural AVMs.

Underwent fully endoscopic MVD with IONM.

Exclusion Criteria

Patients were excluded from the study if they met any of the following criteria:

Presence of severe underlying medical conditions posing a high surgical risk.

Secondary trigeminal neuralgia or secondary hemifacial spasm caused by multiple sclerosis.

Hemifacial spasm occurring after peripheral facial nerve palsy caused by other medical conditions.

Surgical Technique: Fully Endoscopic Microvascular Decompression with Intraoperative Neurophysiological Monitoring

Anesthesia Method: Intravenous general anesthesia.

S**urgical Positioning:** Park bench position.

General Setup Procedure for Intraoperative Neurophysiological Monitoring

Hemifacial spasm case:

Electrode placement: Head: Cz, Fpz, A1, A2. Facial Nerve (CN VII): Frontalis muscle, Orbicularis oculi, Orbicularis oris, Mentalis muscle. Hand: Abductor pollicis brevis muscle. Ear: Auditory stimulation

Intraoperative neurophysiological monitoring techniques: **EEG** (Electroencephalography), **EMG** (Electromyography), **LSR** (Lateral spread response), **ZLR** (Zhong-Lee response), **BAEP** (Brainstem auditory evoked potentials), **TOF** (Train-of-Four for neuromuscular blockade)

Trigeminal neuralgia case:

Electrode placement: Head: C5, C6, Fz, A1, A2. Trigeminal Nerve (CN V): Supraorbital nerve, Infraorbital nerve, Mental nerve. Ear: Auditory stimulation

Intraoperative neurophysiological monitoring techniques: **EEG** (Electroencephalography), **EMG** (Electromyography), **BTEP** (Brainstem trigeminal evoked potentials), **BAEP** (Brainstem auditory evoked potentials), **TOF** (Train-of-Four for neuromuscular blockade)

Lateral spread response (LSR): Lateral Spread Response (LSR) is an abnormal neurophysiological phenomenon that occurs due to unintended nerve stimulation, often observed when the facial nerve is compressed or subjected to vascular pressure. LSR is recorded by stimulating the facial nerve at one site and detecting abnormal signal propagation at different regions, reflecting atypical neural transmission. Following decompression of the facial nerve, LSR disappears or significantly decreases.

Electrode placement: Subdermal electrodes are positioned at the intersection of the facial nerve (n.

facialis) midpoint between the ipsilateral lesion site and the outer side of the eyeball.

Electrical stimulation: Two electrodes (3 cm apart) are placed on the temporal branch or zygomatic branch of the facial nerve. Stimulation is directed toward the brainstem using a pulse duration of 0.2–0.3 ms and an intensity of 5–25 mA. Stimulation of the buccal or mandibular branch is also effective, similar to upper branch stimulation, to assess LSR at the upper branch.

Stimulation riming: Performed at six key intraoperative stages:

1. Before dural opening
2. After dural opening
3. After arachnoid dissection
4. After placement of the decompression patch
5. After dural closure
6. At the end of surgery

Zhong-Lee response (ZLR): The Zhong-Lee response (ZLR) is measured at the same site as LSR, with electrical stimulation applied before nerve decompression and within 5 mm of the nerve. After decompression, stimulation is repeated. Stimulation Method: Bipolar stimulator with pulse duration: 0.2 ms. Intensity: 2 mA. Simultaneous monitoring of LSR and ZLR provides valuable intraoperative neurophysiological guidance for microvascular decompression (MVD), offering superior surgical feedback compared to LSR alone, regardless of whether the facial nerve compression is simple or complex.

Brainstem auditory evoked potentials (BAEPs): Brainstem auditory evoked potentials (BAEPs) are used to assess auditory nerve function and prevent hearing loss during surgery.

Electrode placement: Surface disc electrodes are positioned at both mastoid processes and the vertex (Cz).Two-channel montage configuration: Cz–Ai (Vertex – Ipsilateral mastoid). Cz–Ac (Vertex – Contralateral mastoid)

Auditory stimulation: Broadband click stimuli delivered via tubal insert earphones. Stimulus intensity: Surgical side: 85 dB normal hearing level (nHL). Contralateral masking stimulus: 55 dB nHL or 120 dB sound pressure level (SPL) or 80 dB SPL to minimize cross-stimulation effects

Stimulation frequency and averaging time: Based on Damaty's research: Stimulation frequency: 11.1 Hz. Averaging trials: 1000 repetitions. BAEPs measurement duration: typically >1 minute, but results should be analyzed within 15 seconds when using a higher 31.1 Hz frequency with 1000–4000 averaging trials.

Brainstem trigeminal evoked potential (BTEP) [10]

Stimulation sites: The trigeminal nerve (cranial nerve V) is stimulated using subdermal needle electrodes placed at: V1 (Ophthalmic branch): Supraorbital foramen. V2 (Maxillary branch): Infraorbital foramen. V3 (Mandibular branch): Mental foramen

Stimulation intensity and frequency: Each BTEP dataset consists of 300 stimulus repetitions to confirm the reproducibility of cortical responses. Stimulation frequency: 4.7 Hz. Pulse duration: 0.1 ms. Polarity: Alternating polarity to prevent baseline shift. Stimulation intensity: Ranges from 5 to 11 mA per test set.

Surgical Procedure

Step 1: Skin Incision and Craniotomy

Skin Incision: A 3–5 cm vertical incision is made 5 mm medial to the mastoid notch of the temporal bone. The muscle and fascia are carefully dissected.

Burr Hole Placement: A burr hole is drilled 1 cm inferior and medial to the Asterion.

Craniotomy: The lateral limit of the craniotomy is the sigmoid sinus, with a diameter of 2–3 cm.

First stimulation is performed to identify LSR/ BTEP before dural opening.

Step 2: Dural Opening and Lesion Management

The dura is opened in an C-shaped fashion along the transverse sinus and sigmoid sinus.

30 degree 2.75 mm endoscope is introduced. Arrange the surgical instrument entry points into an isosceles triangle configuration to minimize

instrument collision during the procedure: Position A, located at the 12 o'clock position, is designated for the endoscope. Position B, at the 5 o'clock position, is for the dissecting instrument. Position C, at the 7 o'clock position, is for the suction cannula (Figure 1)

Second stimulation is performed to LSR/BTEP after dural opening

The cerebellum is minimally retracted or not at all. Instead, a small cottonoid is gently placed at the cerebellopontine angle, allowing cerebrospinal fluid (CSF) drainage to reduce brain tension.

The arachnoid membrane is carefully dissected to explore the entire course of the facial nerve (cranial nerve VII) from its exit at the pontomedullary junction to the internal auditory canal in cases of hemifacial spasm, and the trigeminal nerve (cranial nerve V) from its anterolateral emergence at the pons to the Meckel's cave in trigeminal neuralgia, to identify neurovascular conflicts.

Third stimulation is performed to identify LSR/BTEP and ZLR after arachnoid dissection

Perform microsurgical dissection to decompress the offending artery or vein. Subsequently, place a Teflon® pledget or GORE-TEX® implant to insulate and separate all identified neurovascular conflict sites (Figure 1).

Fourth stimulation is performed to confirm the absence of LSR/BTEP and ZLR after neurovascular decompression.

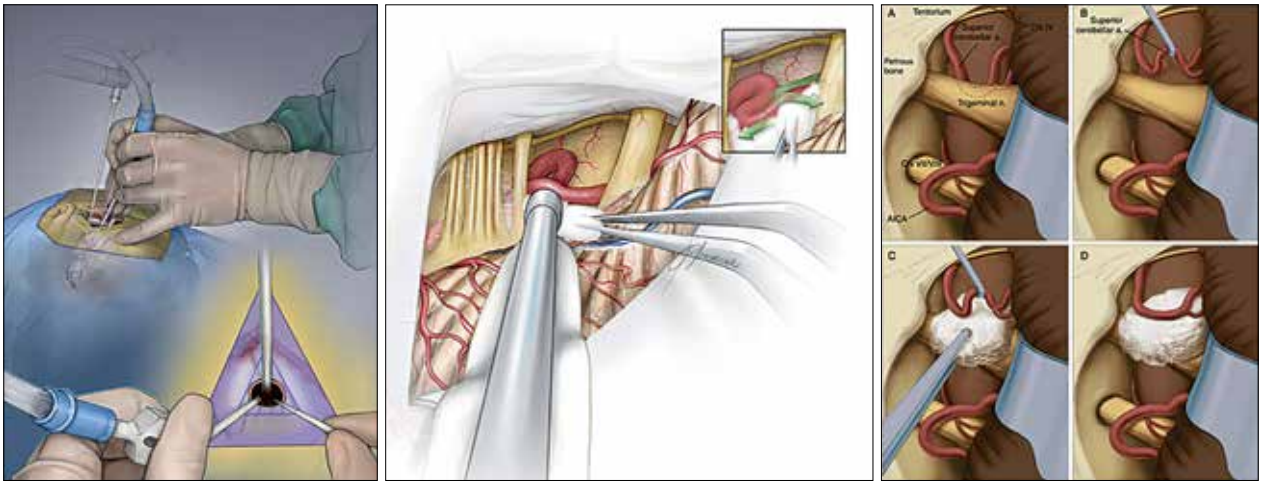

Figure 1: Position of surgical instruments and decompression Techniques for Hemifacial Spasm (left) and Trigeminal Neuralgia (right)

Step 3: Dural and Wound Closure

Dural Closure: The surgical field is gently irrigated with warm saline to prevent injury to the cranial nerves. High-pressure irrigation is avoided.

Fifth stimulation is performed to identify LSR/BTEP after dural closure

Valsalva Maneuver is performed to check for CSF leakage before watertight dural closure.

Bone Replacement and Soft Tissue Closure: The bone flap is repositioned, and the fascia and muscle are sutured before skin closure.

Sixth stimulation is performed to identify LSR/BTEP after skin closure.

Data Analysis and Processing

Data entry was performed using EpiData software (version 4.6.0.6).

Statistical analysis was conducted using STATA software (version 14.0).

Ethical Considerations

The study was conducted with informed consent from all patients.

Patient confidentiality was strictly maintained, and all personal information was used exclusively for research purposes.

Results

Table 1. Clinical features and surgical outcomes

No.	Age/Gender	Clinical manifestation	Medical history	Preoperative grading	Postoperative grading
1	33/Male	Typical right hemifacial spasm, poor response to botulinum toxin injections	Botox injections 4 times in 1 year, only effective for ~2 months	HFS score Clinical: 13 pts QoL: 80%	HFS score Clinical: 0 pts QoL: 0%
2	62/Male	Atypical right trigeminal neuralgia affecting V2, V3 branches, refractory to Tegretol®	Previous MVD (16 months prior), alcohol injection for ganglion ablation (10 months prior)	BNI grade V	BNI grade I
3	51/Male	Typical right hemifacial spasm, poor response to botulinum toxin injections	Botox injections twice in 10 months, previous right epidural hematoma evacuation (TBI in 2015)	HFS score Clinical: 13 pts QoL: 70%	HFS score Clinical: 0 pts QoL: 0%
4	47/Male	Classic left trigeminal neuralgia for 10 years, affecting V2, V3 branches, poor response to Tegretol®	Depression	BNI grade IV	BNI grade I
5	56/Female	Typical right hemifacial spasm	None	HFS score Clinical: 12 pts QoL: 70%	HFS score Clinical: 0 pts QoL: 10%

Comments:

All three hemifacial spasm (HFS) cases showed a complete resolution of clinical symptoms (HFS score: 0 points postoperatively) and significant improvement in quality of life (QoL).

Both trigeminal neuralgia (TN) cases demonstrated a marked reduction in pain severity, improving from BNI grades IV–V to BNI grade I, indicating complete pain relief.

Table 2. Intraoperative findings and postoperative complications

No.	Age/Gender /Diagnosis	Offending vessels	Surgical duration	Compression Site	Postoperative complications
1	33/Male Right HFS	AICA	125 mins	Root exit zone (REZ)	None
2	62/Male Right TN	SCA, arterial branch of AICA and granulation tissue of the previous patch	135 mins	Root entry zone (REZ) and CPA portion	Mild numbness in the right upper lip
3	51/Male Right HFS	AICA	105 mins	Root exit zone (REZ)	Temporary facial paresis (House-Brackmann Grade II), fully recovered after 4 weeks
4	47/Nam Left TN	Two Dandy's vein branches	180 mins	Root entry zone (REZ) and CPA portion	None
5	56/Female Right HFS	AICA	95 mins	Root exit zone (REZ)	Temporary facial paresis (House-Brackmann Grade II), fully recovered after 4 weeks

Comments: The anterior inferior cerebellar artery (AICA) was the primary compressing vessel in all hemifacial spasm (HFS) cases (3/3 cases, 100%). Trigeminal neuralgia (TN) cases showed more diverse compression sources, including the superior cerebellar artery (SCA), an AICA branch, and even granulation tissue from a previous patch (Case #2), as well as Dandy's veins (Case #4).

All hemifacial spasm (HFS) cases presented compression at the root exit zone (REZ) of the facial nerve. Trigeminal neuralgia (TN) cases had compression at both the root entry zone (REZ) and cerebellopontine angle (CPA).

Temporary facial paresis (House-Brackmann Grade II) occurred in two HFS cases (Cases #3 and #5) but fully recovered in Case #3 within 4 weeks, which is reassuring for postoperative recovery. Case #2 (TN with prior MVD and alcohol injection) developed mild numbness in the upper lip, likely due to prior nerve injury or surgical manipulation. No major complications (such as hearing loss, CSF leaks, or severe neurological deficits) were reported.

Table 3. Intraoperative neurophysiological monitoring findings

No.	Age/Gender/ Diagnosis	Intraoperative Neurophysiological Monitoring Configuration	Pre-Decompression Electrophysiological Signals	Post-Decompression Electrophysiological Signals
1	33/Male Right HFS	EEG, EMG, LSR, ZLR, BAEP, TOF	Presence of LSR from orbicularis oris and mentalis muscle w/ stimulation of zygomatic branch of facial nerve	Loss of LSR from orbicularis oris and mentalis muscle Zhong-Lee test (+) No intraoperative changes in BAEP
2	62/Male Right TN	EEG, EMG, BTEP, BAEP, TOF	No detectable BTEP waves in V2, V3 branches	Appearance of BTEP waves in the V3 branch
3	51/Male Right HFS	EEG, EMG, LSR, ZLR, BAEP, TOF	Presence of LSR from orbicularis oris and mentalis muscle w/ stimulation of zygomatic branch of facial nerve	Loss of LSR from orbicularis oris and mentalis muscle Zhong-Lee test (+) No intraoperative changes in BAEP
4	47/Nam Left TN	EEG, EMG, BTEP, BAEP, TOF	No detectable BTEP waves in V2, V3 branchesx`	Appearance of BTEP waves in V2, V3 branches No intraoperative changes in BAEP
5	56/Female Right HFS	EEG, EMG, LSR, ZLR, BAEP, TOF	Presence of LSR from frontalis muscle w/ stimulation of madibular branch of facial nerve	Loss of LSR LSR from frontalis muscle Zhong-Lee test (+) No intraoperative changes in BAEP

Comments:

LSR disappearance after decompression was observed in all hemifacial spasm cases, confirming effective neurovascular decompression.

Positive Zhong-Lee response in all HFS cases, indicating adequate nerve decompression.

BTEP wave reappearance post-decompression in trigeminal neuralgia cases, suggesting restored trigeminal nerve conduction.

BAEP remained stable intraoperatively, indicating preserved auditory function.

Discussion

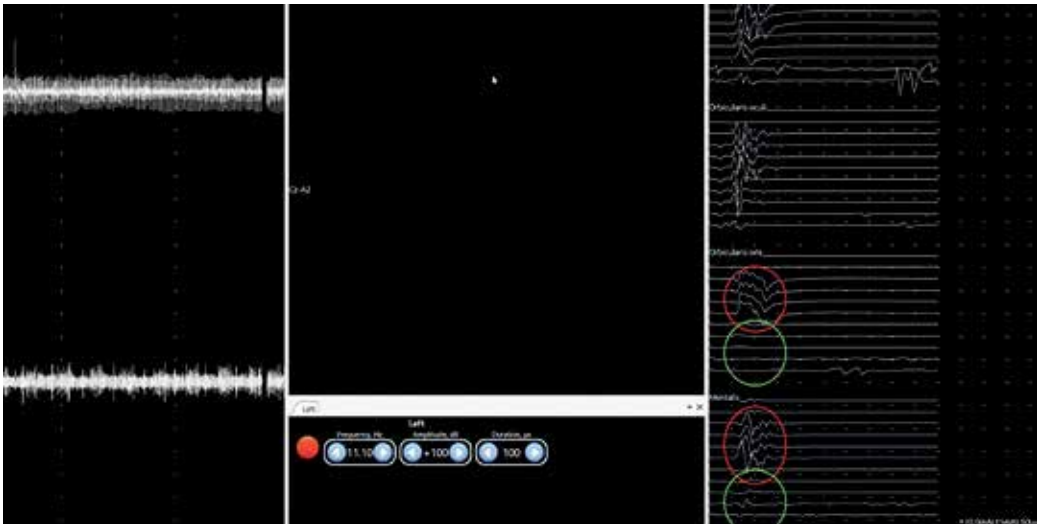

Figure 2. Presence of LSR from orbicularis oris and mentalis muscle (red circle) disappearance of LSR after decompression and Zhong-Lee test (+) (green circle)

Role of lateral spread respond and Zhong-Lee response: All three HFS cases (#1, #3, #5) demonstrated complete loss of LSR post-decompression. The Zhong-Lee test was positive in all HFS cases. This result is consistent with recent previous studies, further supporting the role of LSR disappearance as an intraoperative marker of successful neurovascular decompression. Lee et al. (2017) conducted a study evaluating the prognostic ability of intraoperative electromyographic monitoring during MVD for HFS. They found that patients whose LSR disappeared intraoperatively were more likely to experience complete symptom relief (89.4%) compared to those with persistent LSR (81.9%) [11]. A more recent study by Cho et al. (2023) explored the optimal methods for reliable LSR monitoring during MVD. Their findings indicated that LSR disappearance was associated with higher rates of immediate and sustained postoperative relief from HFS symptoms, reinforcing its role as a key intraoperative marker (Adj. OR 1.41, 95% CI 0.07-27.6) [12]. Additionally, Thirumala et al. (2020) conducted a meta-analysis examining the utility of intraoperative LSR recordings. Their study suggested that LSR disappearance was associated with better short-term symptom relief but was not an absolute predictor of long-term surgical success. The final intraoperative LSR status predicted clinical outcomes after MVD with the following specificities and sensitivities: 89% (0.83-0.93) and 40% (0.30- 0.51) at discharge, 90% (0.84-0.94) and 41% (0.29-0.53) at 3 mo, 89% (0.83-0.93) and 40% (0.30-0.51) at 1 yr. When LSR persisted after MVD, the probability (95% CI) of persistent HFS was 47.8% (0.33-0.63) at discharge, 40.8% (0.23-0.61) at 3 mo, and 24.4% (0.13-0.41) at 1 yr. However, when LSR resolved, the probability for HFS persistence dropped to 7.3% at discharge, 4.2% at 3mo, and 4.0% at 1 yr [13]. Recent studies highlight the clinical significance of the Zhong-Lee response in improving surgical accuracy. Park (2020) in a review on intraoperative monitoring

for MVD highlighted bed the ZL response as a reliable tool to confirm adequate decompression, especially in cases where lateral spread response (LSR) persistence raises concerns about residual compression. Montano et al. (2024) provide the first evidence supporting ZLR as an intraoperative tool for TN MVD. These findings suggest that ZLR could serve as a real-time intraoperative biomarker for MVD success, helping neurosurgeons to verify successful decompression s and potentially reduce recurrence rates [4].

Role of Brainstem trigeminal evoked potentials. Preoperatively, no BTEP waves were detected in the V2 and V3 branches of TN cases (#2, #4), indicating impaired conduction. Postoperatively, BTEP waves reappeared in V3 (Case #2) and in both V2 and V3 (Case #4), suggesting restored sensory pathway function after neurovascular decompression. This supports the hypothesis that successful decompression leads to reactivation of trigeminal sensory conduction, potentially correlating with postoperative pain relief. BTEP is an electrophysiological signal generated in response to stimulation of the trigeminal nerve, allowing surgeons to assess nerve integrity and functional recovery intraoperatively. Compared to brainstem auditory evoked potentials (BAEPs), which primarily focus on auditory pathways, BTEP specifically targets trigeminal nerve conduction, making it a more relevant biomarker for TN surgeries. A recent study by Yun et al. (2024) evaluated the role of brainstem auditory and trigeminal evoked potentials in fully endoscopic MVD for both TN and hemifacial spasm. The study demonstrated that BTEP changes correlated with successful decompression, and a sustained improvement in evoked potentials predicted better long-term pain relief [14]. Zhu et al. (2017) reported that among 29 of 33 patients who achieved complete pain relief without medication, only one patient showed no change in BTEP waveform across the ophthalmic, maxillary and mandibular branches. The remaining 28 patients demonstrated improvement in at least one branch post- decompression. This highlights the potential predictive value of BTEP in ensuring complete nerve decompression during MVD. While the disappearance of lateral spread response (LSR) is a well-accepted success marker for MVD in hemifacial spasm, an equivalent biomarker for TN has been lacking. BTEP has emerged as a candidate for intraoperative success prediction. A prospective study by Lee et al. (2025) analyzed 682 MVD cases and found that real-time changes in BTEP corresponded with better surgical outcomes. The study reported that patients with BTEP normalization post-MVD experienced superior pain relief and fewer relapses. Furthermore, studies have indicated that BTEP monitoring may reduce iatrogenic nerve damage, as persistent abnormalities post-decompression may suggest inadequate vascular separation or additional pathology. This ability to assess nerve recovery in real time helps guide surgeons in optimizing decompression techniques, thereby reducing complications such as facial numbness and residual pain.

Role of brainstem auditory evoked potentials. BAEP remained stable throughout the procedure in all cases, suggesting no intraoperative auditory nerve damage or ischemic events affecting the cochlear nucleus. This is a key finding in microvascular decompression, as postoperative hearing loss is a known risk, particularly in cases with AICA involvement BAEP is an electrophysiological response elicited by auditory stimuli to assess cochlear nerve function and brainstem integrity. Since MVD requires microsurgical manipulation near the brainstem and auditory pathways, intraoperative BAEP monitoring provides real-time feedback on auditory nerve function, allowing surgeons to adjust their approach and minimize damage. A preliminary study by Yun et al. (2024) investigated the use of BAEP in fully endoscopic MVD and found that BAEP changes correlated with intraoperative nerve handling and postoperative auditory function.

Patients who experienced persistent BAEP deterioration intraoperatively were more likely to develop postoperative hearing deficits, reinforcing BAEP's predictive value for hearing preservation [5], [14]. BAEP monitoring has been extensively studied in MVD for HFS, where its role extends beyond auditory protection to predicting surgical success. Unlike trigeminal neuralgia, where intraoperative monitoring options remain limited, HFS surgeries benefit from multiple neurophysiological markers, including lateral spread response (LSR) and BAEPs. A large-scale study by Lee et al. (2025) analyzed 682 MVD cases and confirmed that real-time BAEP changes were associated with a higher risk of postoperative hearing impairment. Interestingly, the study found that MVD performed with a retro-mastoid suboccipital approach had a lower incidence of BAEP changes, correlating with reduced hearing loss. This was 1.0% of all MVD patients and 1.1% among patients with hemifacial spasm. Partial hearing loss defined as a decrease in hearing of 20 db or more from preoperative levels, occurred in 37 patients: 35 with HFS and 2 with TN. Of the 2 TN patients, one recovered about a month after surgery, while the other developed delayed low-frequency hearing loss about one month later. Of 35 patients who developed partial hearing loss after MVD surgery for HFS, 5 patients developed conductive hearing loss that improved. Among the remaining 30 patients, 8 had a high-frequency hearing loss, 14 had a low-frequency hearing loss, and the remaining 8 had a full-range partial hearing loss. These findings suggest that both surgical technique and intraoperative monitoring strategies impact auditory preservation outcomes [15]

Conclusion

Based on our analysis of five cases undergoing fully endoscopic microvascular decompression (MVD) with intraoperative neurophysiological monitoring (IONM) for the treatment of trigeminal neuralgia and hemifacial spasm, we conclude that IONM is a reliable and effective method for predicting surgical outcomes and minimizing the risk of intraoperative nerve injury.

References

1. McLaughlin MR, Jannetta PJ, Clyde BL, Subach BR, Comey CH, Resnick DK. Microvascular decompression of cranial nerves: lessons learned after 4400 operations. J Neurosurg. 1999;90(1):1-8. doi:10.3171/JNS.1999.90.1.0001
2. Zhao Z, Chai S, Xiao D, et al. Microscopic versus endoscopic microvascular decompression for the treatment of hemifacial spasm in China: A meta-analysis and systematic review. Journal of Clinical Neuroscience. 2021;91:23-31. doi:10.1016/j.jocn.2021.06.034
3. Zheng X, Zhang B, Shao D, et al. Fully endoscopic microvascular decompression for hemifacial spasm: a clinical study and analysis. Neurosurg Rev. 2024;47(1). doi:10.1007/S10143-024-02311-5
4. Montano N, D'Alessandris QG, Grilli F, et al. Abnormal electromyographical trigeminal activation through stimulation of the offending artery (Z-L response): An intraoperative tool during microvascular decompression for trigeminal neuralgia. Cephalalgia. 2024;44(11). doi:10.1177/03331024241273913
5. Polo G, Fischer C, Sindou MP, et al. Brainstem auditory evoked potential monitoring during microvascular decompression for hemifacial spasm: intraoperative brainstem auditory evoked potential changes and warning values to prevent hearing loss--prospective study in a consecutive series of 84 patients. Neurosurgery. 2004;54(1):97-106. doi:10.1227/01.NEU.0000097268.90620.07
6. Pham Hoang Anh, Duong Dai Ha, Chu Thanh Hung et al. Surgical outcomes of fully endoscopic microvascular decompression for hemifacial spasm at VietDuc University Hospital. Journal of Medical Research. 2023; 162(1):37-45
7. Bui Huy Manh. Application of microvascular decompression for trigeminal neuralgia. PhD dissertation in Medicine. 2015.
8. Guo X, Zhang C, Li Y, Li X, Ma X, Li W. Fully Endoscopic Microvascular Decompression for Hemifacial Spasm Using Improved Retrosigmoid Infrafloccular Approach: Clinical Analysis of 81 Cases. Operative Neurosurgery. 2022;23(1):40-45. doi:10.1227/ONS.0000000000000221
9. Jiang H, Wang P, Zhou D, Zeng L, Lin B, Wu N. Fully endoscopic microvascular decompression

for hemifacial spasm. Exp Ther Med. 2022;24(1). doi:10.3892/ETM.2022.11410

10. Zhu J, Zhang X, Zhao H, Tang Y Da, Ying TT, Li ST. Utility of Brainstem Trigeminal Evoked Potentials in Patients With Primary Trigeminal Neuralgia Treated by Microvascular Decompression. J Craniofac Surg. 2017;28(6):e571-e577. doi:10.1097/SCS.0000000000003882
11. Lee SH, Park BJ, Shin HS, Park CK, Rhee BA, Lim YJ. Prognostic ability of intraoperative electromyographic monitoring during microvascular decompression for hemifacial spasm to predict lateral spread response outcome. J Neurosurg. 2017;126(2):391-396. doi:10.3171/2016.1.JNS151782
12. Cho KR, Lee HS, Kim M, Park SK, Park K. Optimal method for reliable lateral spread response monitoring during microvascular decompression surgery for hemifacial spasm. Scientific Reports 2023 13:1. 2023;13(1):1-13. doi:10.1038/s41598-023-49008-1
13. Thirumala PD, Altibi AM, Chang R, et al. The utility of intraoperative lateral spread recording in microvascular decompression for hemifacial spasm: A systematic review and meta-analysis. Neurosurgery. 2020;87(4):E473-E484. doi:10.1093/NEUROS/NYAA069
14. Yun GY, Ahn JM, Park JH, Oh HJ, Shim JJ, Yoon SM. Preliminary Report of Fully Endoscopic Microvascular Decompression. J Korean Neurosurg Soc. 2024;67(6):646-653. doi:10.3340/JKNS.2024.0003
15. Lee H, Park K, Park SK, Lee DH. Hearing Loss after Microvascular Decompression Surgery Performed with the Retro-Mastoid Suboccipital Approach: A Single-Center Study, Analysis of 682 Cases. J Neurol Surg B Skull Base. 2025;86(S 01):S357. doi:10.1055/S-0045-1803370.