

Review Article

Intraoperative neurophysiological monitoring during microvascular decompression for trigeminal neuralgia

Byung-Euk Joo*

Department of Neurology, Soonchunhyang University Seoul Hospital, Soonchunhyang University College of Medicine, Seoul, Korea

ABSTRACT

Trigeminal neuralgia (TN) is a severe neuropathic disorder marked by sudden, shock-like episodes of facial pain caused by pathological irritation of the trigeminal nerve. Microvascular decompression (MVD) remains the only intervention that directly alleviates the neurovascular conflict underlying classical TN. Because the procedure is performed near cranial nerves VII and VIII as well as the brainstem, intraoperative neurophysiological monitoring (IONM) has become an essential component for minimizing surgical morbidity. Among existing modalities, brainstem auditory evoked potentials serve as the most dependable technique for protecting auditory pathway function. Additional methods—such as trigeminal somatosensory evoked potentials and free-running/triggered electromyography—provide complementary information that enables early detection of nerve stress or impending injury. This review synthesizes current physiologic concepts, monitoring strategies, and clinical evidence supporting the application of IONM during MVD for TN.

Keywords: electromyography; evoked potentials, auditory, brain stem; intraoperative neurophysiological monitoring; microvascular decompression surgery; trigeminal neuralgia

Introduction

Trigeminal neuralgia (TN) is characterized by episodes of intense, electric shock-like facial pain involving one or more divisions of the trigeminal nerve. In most patients with classical TN, the disorder results from neurovascular compression at the trigeminal root entry zone, which leads to focal demyelination and aberrant neural transmission [1-5]. Since Jannetta first introduced microvascular decompression (MVD), the procedure has provided durable pain relief for a majority of affected patients [1,2,6-8]. Despite its effectiveness, MVD requires cerebellar retraction and manipulation of vessels near cranial nerves VII and VIII, exposing these structures to potential traction, ischemic damage, or thermal injury [9,10]. Consequently, intraoperative neurophysiological monitoring (IONM) has become a routine and essential component of posterior fossa surgery, offering real-time

feedback on neural integrity during critical steps of the operation [11-13]. The monitoring techniques most frequently employed in MVD for TN include brainstem auditory evoked potentials (BAEPs), trigeminal somatosensory evoked potentials (TSEPs), and free-running/triggered electromyography (EMG) [14-16]. Each modality provides different, complementary insights, and their combined use significantly enhances the safety profile of the procedure. This review integrates current knowledge on the physiologic basis, technical implementation, and clinical value of IONM in the context of MVD for TN [17,18].

Intraoperative Neurophysiological Monitoring

1. Brainstem auditory evoked potentials

BAEPs represent the most widely adopted and clinically validated IONM technique during MVD for TN. Although MVD is highly effective for treating

Received Dec 19, 2025; Revised Dec 23, 2025; Accepted Dec 24, 2025

*Corresponding author: Byung-Euk Joo, Department of Neurology, Soonchunhyang University Seoul Hospital, Soonchunhyang University College of Medicine, Seoul 04401, Korea

Tel: +82-2-709-9224, Fax: +82-2-710-3098, E-mail: faithjoo17@gmail.com

© 2025 Korean Society of Intraoperative Neurophysiological monitoring (KSION)

This is an Open Access article distributed under the terms of the Creative Commons Attribution Non-Commercial License (<http://creativecommons.org/licenses/by-nc/4.0/>) which permits unrestricted non-commercial use, distribution, and reproduction in any medium, provided the original work is properly cited.

classical TN, the surgical approach requires cerebellar retraction and dissection around the cranial nerve VII/VIII complex within the cerebellopontine angle. Because the vestibulocochlear nerve lies adjacent to the operative field, even minor traction, thermal exposure, or vascular compromise can result in temporary or permanent postoperative hearing loss. BAEPs provide continuous, real-time assessment of cochlear nerve function, allowing the surgical team to identify early signs of auditory pathway stress before irreversible injury occurs [19]. These potentials consist of short-latency waveforms generated along the auditory pathway from the cochlea to the midbrain. Among these components, wave V is considered the most robust under anesthesia and serves as the primary warning marker. Meaningful intraoperative alerts include a latency increase greater than 0.5 ms, a reduction in amplitude exceeding 50%, or the complete loss of wave V [20,21]. Such electrophysiologic changes frequently precede visible structural deformation, making BAEPs a sensitive early indicator of neural compromise. When significant deterioration is observed, immediate interventions—such as decreasing cerebellar retraction, adjusting the surgical trajectory, pausing vessel manipulation, or irrigating with warm saline—may restore the waveform and prevent lasting cochlear nerve injury.

Clinical investigations have shown that BAEP-guided MVD reduces the likelihood of postoperative hearing deficits. Persistent loss of wave V is strongly correlated with postoperative sensorineural hearing loss [22], whereas transient waveform changes that return to baseline intraoperatively typically do not result in long-term morbidity [23-25]. Because patients with TN generally have normal preoperative hearing, preservation of wave V is a key determinant of postoperative quality of life [26]. Despite its advantages, BAEP monitoring has limitations: signal quality may be affected by drilling, electrocautery, irrigation, or variations in anesthetic depth; and the averaging time required to generate stable waveforms may delay detection of rapid physiologic changes. Nonetheless, BAEPs remain the gold standard modality for auditory preservation during MVD.

2. Trigeminal somatosensory evoked potentials

Trigeminal SSEPs are obtained by stimulating peripheral branches of the trigeminal nerve—most commonly the supraorbital (V1), infraorbital (V2), or mental nerve (V3). The resulting responses are recorded from brainstem and cortical sites. Compared with limb SSEPs, trigeminal responses tend to be lower in amplitude and more susceptible to noise; however, with appropriate adjustments in anesthetic regimen, stimulation intensity, and recording techniques, reproducible waveforms can be obtained [27-29]. During MVD, TSEPs offer a method for detecting early sensory pathway disturbances caused by traction, manipulation near the root entry zone, or transient ischemia. Latency prolongation or amplitude reduction may serve as an early indicator of impending neural compromise, prompting technique modification to reduce postoperative sensory deficits. In some cases, stabilization or improvement of the waveform after decompression may signal effective relief of the neurovascular conflict.

Optimal use of trigeminal SSEPs requires careful attention to electrode placement, stimulation frequency, filtering, and artifact management. Although standardized guidelines are not yet fully established, TSEPs are increasingly recognized as a valuable adjunct for improving safety during MVD.

3. Free-running/triggered electromyography

Free-running EMG is widely used during MVD for TN. The trigeminal motor root innervates the muscles of mastication; spontaneous EMG activity from the masseter, temporalis, or lateral pterygoid therefore provides continuous feedback on mechanical or thermal irritation affecting the nerve. Free-running EMG does not require electrical stimulation but instead monitors spontaneous muscular activity throughout the procedure. When the trigeminal motor root is stretched, compressed, or manipulated, characteristic EMG patterns—bursts, spikes, or neurotonic discharges—may appear [30,31]. These signals often precede visible structural injury, allowing the surgeon to reduce retraction, adjust dissection, or reposition instruments. The use of free-running EMG has been

associated with reduced postoperative complications, including masticatory weakness, jaw deviation, and chewing difficulty. EMG also complements other modalities such as TSEPs and BAEPs by providing direct information about the motor pathways of the trigeminal nerve. Facial nerve monitoring is also commonly performed by recording spontaneous EMG activity from orbicularis oculi and oris, reflecting the function of the temporal and zygomatic branches. When traction or irritation occurs, characteristic EMG discharges may be detected, allowing immediate corrective actions. Triggered EMG is sometimes incorporated to identify the facial nerve in cases with distorted anatomy, such as revision surgeries or narrow corridors, reducing the risk of postoperative facial dysfunction [18,32]. Although EMG does not directly influence pain outcomes, it plays a central role in preventing avoidable complications and preserving facial nerve integrity.

Conclusions

MVD remains the most definitive treatment for classical TN. The integration of IONM significantly enhances surgical safety by providing real-time assessment of auditory, sensory, and motor pathway integrity. BAEPs continue to serve as the most reliable method for protecting cochlear nerve function, while trigeminal SSEPs and free-running/triggered EMG add complementary layers of protection. Although technical limitations persist—particularly for trigeminal SSEPs—ongoing technological advancements are expected to improve their reliability and clinical utility. The combined use of these modalities strengthens the safety profile of MVD and supports improved functional outcomes for patients with TN.

Ethical approval

Not applicable.

Conflicts of interest

No potential conflict of interest relevant to this article was reported.

ORCID

Byung-Euk Joo, <https://orcid.org/0000-0003-3566-1194>

References

1. Jannetta PJ. Arterial compression of the trigeminal nerve at the pons in patients with trigeminal neuralgia. *J Neurosurg.* 1967;26(1part2):159-62.
2. Barker FG, Jannetta PJ, Bissonette DJ, Larkins MV, Jho HD. The long-term outcome of microvascular decompression for trigeminal neuralgia. *N Engl J Med.* 1996;334(17):1077-84.
3. Love S, Coakham HB. Trigeminal neuralgia: pathology and pathogenesis. *Brain.* 2001;124(12):2347-60.
4. Devor M, Amir R, Rappaport ZH. Pathophysiology of trigeminal neuralgia: the ignition hypothesis. *Clin J Pain.* 2002;18(1):4-13.
5. Burchiel KJ. A new classification for facial pain. *Neurosurgery.* 2003;53(5):1164-7.
6. Lovely TJ, Jannetta PJ. Microvascular decompression for trigeminal neuralgia: surgical technique and long-term results. *Neurosurg Clin N Am.* 1997;8(1):11-30.
7. Jiao L, Ye H, Lv J, Xie Y, Sun W, Ding G, et al. A systematic review of repeat microvascular decompression for recurrent or persistent trigeminal neuralgia. *World Neurosurg.* 2022;158:226-33.
8. Kalkanis SN, Eskandar EN, Carter BS, Barker FG. Microvascular decompression surgery in the United States, 1996 to 2000: mortality rates, morbidity rates, and the effects of hospital and surgeon volumes. *Neurosurgery.* 2003;52(6):1251-62.
9. Sekula RF Jr, Frederickson AM, Jannetta PJ, Quigley MR, Aziz KM, Arnone GD. Microvascular decompression for elderly patients with trigeminal neuralgia: a prospective study and systematic review with meta-analysis. *J Neurosurg.* 2011;114(1):172-9.
10. Jung NY, Lee SW, Park CK, Chang WS, Jung HH, Chang JW. Hearing outcome following microvascular decompression for hemifacial spasm: series of 1434 cases. *World Neurosurg.* 2017;108:566-71.
11. Watanabe E, Schramm J, Strauss C, Fahlbusch R. Neurophysiologic monitoring in posterior fossa surgery: II. BAEP-waves I and V and preservation of

- hearing. *Acta Neurochir.* 1989;98(3):118-28.
12. Morisaki Y, Takatani T, Kotsugi M, Yamazaki S, Yokoyama S, Nakase K, et al. Intraoperative monitoring of trigeminal neuralgia: a technical note. *Acta Neurochir.* 2024;166(1):238.
 13. Nuwer MR. Intraoperative neurophysiologic monitoring. *J Clin Neurophysiol.* 1998;15(3):181-2.
 14. Soustiel JF, Hafner H, Chistyakov AV, Guilburd JN, Zaaroor M, Yussim E, et al. Monitoring of brainstem trigeminal evoked potentials. Clinical applications in posterior fossa surgery. *Electroencephalogr Clin Neurophysiol.* 1993;88(4):255-60.
 15. Singh N, Sachdev KK, Brisman R. Trigeminal nerve stimulation: short latency somatosensory evoked potentials. *Neurology.* 1982;32(1):97.
 16. Ferreira CA, Thirumala P, Crammond DJ, Sekula RF Jr, Balzer JR. Intraoperative neurophysiological monitoring during microvascular decompression of cranial nerves. In: Deletis V, Shils JL, Sala F, Seidel K, editors. *Neurophysiology in neurosurgery.* 2nd ed. Cambridge, MA: Academic Press; 2020. p. 457-72.
 17. Grundy BL. Monitoring of sensory evoked potentials during neurosurgical operations: methods and applications. *Neurosurgery.* 1982;11(4):556-75.
 18. Guntinas-Lichius O, Eisele DW. Facial nerve monitoring. *Adv Oto-Rhino-Laryngol.* 2016;78:46-52.
 19. Chiappa KH, Ropper AH. Evoked potentials in clinical medicine. *N Engl J Med.* 1982;306(19):1140-50.
 20. Thirumala PD, Carnovale G, Loke Y, Habeych ME, Crammond DJ, Balzer JR, et al. Brainstem auditory evoked potentials' diagnostic accuracy for hearing loss: systematic review and meta-analysis. *J Neurol Surg B Skull Base.* 2017;78(01):043-051.
 21. Watanabe N, Ishii T, Fujitsu K, Kaku S, Ichikawa T, Miyahara K, et al. Intraoperative cochlear nerve mapping with the mobile cochlear nerve compound action potential tracer in vestibular schwannoma surgery. *J Neurosurg.* 2018;130(5):1568-75.
 22. Romstöck J, Strauss C, Fahlbusch R. Continuous electromyography monitoring of motor cranial nerves during cerebellopontine angle surgery. *J Neurosurg.* 2000;93(4):586-93.
 23. El Damaty A, Rosenstengel C, Matthes M, Baldauf J, Dziemba O, Hosemann W, et al. A new score to predict the risk of hearing impairment after microvascular decompression for hemifacial spasm. *Neurosurgery.* 2017;81(5):834-43.
 24. James ML, Husain AM. Brainstem auditory evoked potential monitoring: when is change in wave V significant? *Neurology.* 2005;65(10):1551-5.
 25. Bartindale M, Kircher M, Adams W, Balasubramanian N, Liles J, Bell J, et al. Hearing loss following posterior fossa microvascular decompression: a systematic review. *Otolaryngol Head Neck Surg.* 2018;158(1):62-75.
 26. Sloan TB. Anesthesia and motor evoked potential monitoring. In: Deletis V, Shils JL, editors. *Neurophysiology in neurosurgery.* Cambridge, MA: Academic Press; 2002. p. 451-74.
 27. Bromm B, Chen ACN. Brain electrical source analysis of laser-evoked potentials in response to painful trigeminal nerve stimulation. *Electroencephalogr Clin Neurophysiol.* 1995;95(1):14-26.
 28. Zhao YX, Miao SH, Tang YZ, He LL, Yang LQ, Ma Y, et al. Trigeminal somatosensory-evoked potential: a neurophysiological tool to monitor the extent of lesion of ganglion radiofrequency thermocoagulation in idiopathic trigeminal neuralgia: a case-control study. *Medicine.* 2017;96(3):e5872.
 29. Sandel T, Eide PK. Long-term results of microvascular decompression for trigeminal neuralgia and hemifacial spasms according to preoperative symptomatology. *Acta Neurochir.* 2013;155(9):1681-92.
 30. Sutter M, Eggspuehler A, Grob D, Jeszenszky D, Benini A, Porchet F, et al. The validity of multimodal intraoperative monitoring (MIOM) in surgery of 109 spine and spinal cord tumors. *Eur Spine J.* 2007;16(Suppl 2):S197-208.
 31. Redmond MD, Rivner MH. False positive electrodiagnostic tests in carpal tunnel syndrome. *Muscle Nerve.* 1988;11(5):511-8.
 32. Kartush JM, Rice KS, Minahan RE, Balzer GK, Yingling CD, Seubert CN. Best practices in facial nerve monitoring. *Laryngoscope.* 2021;131(S4):S1-42.