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Intraoperative neurophysiological mapping of trigeminal nerve: A surgical advancement in neurovascular decompression

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1. Introduction

Trigeminal neuralgia (TN) is a paroxysmal pain disorder distinguished by sudden, severe, and unilateral facial pain affecting one or more branches of the trigeminal nerve. The pain is commonly characterized as electric-shock-like, stabbing, shooting in quality, and can be triggered by innocuous stimuli such as light touch or vibration.[1,2] In the majority of cases, the etiology of TN is attributed to neurovascular compression of the trigeminal nerve at the root entry zone (REZ), caused by adjacent blood vessels, resulting in demyelination and hyperexcitability of the affected nerve fibers. Additionally, other potential causative factors encompass structural anomalies such as tumors or arteriovenous malformations, as well as demyelinating diseases like multiple sclerosis.[3–5]

The trigeminal nerve REZ represents a critical anatomical structure situated at the lateral aspect of the pons, where the trigeminal nerve fibers enter the brainstem, encompassing layers of connective tissue and clusters of glial cells, such as astrocytes and oligodendrocytes. These glial cells play pivotal roles in preserving the structural and functional integrity of the trigeminal nerve fibers. Adjacent to the REZ, a small area known as the transition zone (TZ) emerges, typically located at a distance of 1–2.5 mm from the REZ, depending upon its medial or lateral side. This region is characterized by the transition from the central myelinated fibers to the peripheral unmyelinated fibers.[6] Importantly, within this zone, the trigeminal nerve closely interfaces with adjacent blood vessels, with particular emphasis on the superior cerebellar artery, which is the most common offending vessel in trigeminal neuralgia.[7]

The collective evidence derived from systematic reviews consistently supports the use of anticonvulsant drugs as the most extensively studied and preferred therapy for both the initial and long-term management of classical TN.[8–10] Nevertheless, the incidence of medically refractory TN exhibits variability across studies but it is estimated to affect at least 20%–30% of all patients with TN, with approximately 8% developing intolerance to treatment.[11] It is crucial to recognize that microvascular decompression, while standing as the foremost surgical procedure for the management of classical trigeminal neuralgia, may not be universally suitable for all patients. A pooled analysis involving 5149 individuals demonstrated a notably high efficacy associated with this surgical intervention, with approximately 62–89% of patients reporting complete freedom from pain during long-term follow-up.[12]

Intraoperative electrophysiological mapping has been employed for several decades to assist in microvascular decompression and rhizotomy surgeries, particularly in cases of trigeminal neuralgia. This technique involves the application of electrical stimulation to evoke antidromic responses in compound nerve action potentials (CNAP), thereby enabling precise identification and recording from the three trigeminal branches.[13] Electrophysiological mapping has also played an important role in corroborating anatomical descriptions of trigeminal root divisions.[14] Nonetheless, the precise localization of trigeminal nerve territories at the REZ exhibits variation across distinct studies and assumes utmost significance in discerning the most susceptible region in each specific surgical case. To the best of our knowledge, this is the first study

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to investigate the neurophysiological mapping of the REZ at the neurovascular compression site, and its relations with clinical findings and long-term outcome.

2. Patients and methods

This retrospective study was conducted at the Neurological Institute of Curitiba Hospital over the period from 2017 to 2022. This series included 23 cases of electrophysiological mapping of the trigeminal nerve during microvascular decompression for classic trigeminal neuralgia treatment. A comprehensive dataset was extracted from each patient's medical records, encompassing demographic variables (age, sex), time interval from symptom onset to treatment, symptomatic trigeminal division, side of pain, pre-operative Barrow Neurological Index (BNI) classification, stimulation zone, clinical-neurophysiologic correspondence, vascular compression type, and post-operative BNI outcome. Based on insights from anatomical and physiological studies, we designated 3 distinct zones at the trigeminal nerve REZ for the purposes of antidromic stimulation and compound nerve action potential (CNAP) recording: Zone 1 (superior/rostral), Zone 2 (intermediate), and Zone 3 (inferior/caudal).[13,15] Categorical variables were subjected to statistical analysis using Fisher's exact test, with a significance level set at 0.05. SPSS version 28 software was used as a tool to analyze the data.

Surgery was performed under Total Intravenous Anesthesia (remifentanil and propofol), with the patient positioned in dorsal decubitus and the head held in a Mayfield headholder rotated contralaterally to facilitate retrosigmoid craniotomy. After positioning, for the recording of antidromic responses, subdermal needle electrodes (bipolar Sunderland needles twister pair) were placed near the supraorbital, infraorbital, and mental foramina, and the ground electrode on the shoulder (Fig. 1). Following retrosigmoid craniotomy and duramater opening, the cerebelomedullary cistern was drained, and any small tentorial veins under tension are coagulated. Trigeminal nerve arachnoid dissection is meticulously performed from the tentorial to the caudal region, preferably with avoidance of unnecessary dissection involving the VII-VIII complex and coagulation of petrous veins. After trigeminal nerve complete dissection, stimulation was carried out using a monopolar handheld probe at the three distinct zones, both prior to and following microvascular decompression. Stimulus intensity was progressively increased up to 2 mA until a clear response is obtained, duration was 0.3 ms, and frequency was 7 Hz. The resulting compound nerve action potentials were captured using a band-pass filter of 200–2000 Hz in single wave mode. Each trace presented was generated from the averaging of 100 individual responses. If the compression site and stimulation response were not congruent to symptomatic nerve root and additional dissection of TN is done, mainly in the cisternal and more proximal portion at the pons, looking for any minor artery or vein compression. We used a polytetrafluoroethylene (Teflon) felt as interposed material to ensure lasting separation between the vessel and the nerve.

3. Results

The average age of the patients was 55 ± 8,14 years, with 14 females (60,8%) and 9 males (39,2%). The time from the onset of trigeminal neuralgia to treatment averaged 94 months (4–264 months). The most symptomatic trigeminal nerve division was V2 + V3 (29%) followed by V1 + V2 (25%) and V3 (21%). When considering isolated trigeminal nerve divisions, the V2 branch was the most frequently affected, causing pain in 45% of the patients. Thirteen patients (54%) had an pre-operative BNI of IV (indicating pain not adequately controlled by medications) and 11 patients (46%) presented a BNI of V (severe pain with no relief). Patient data is summarized in Table 1, the distribution of pain according to site of compression was found to be significantly different as shown in Table 2 (Fisher's exact test $p = 0{,}008$).

The responses obtained through the stimulation of the 3 zones are resumed in Fig. 2. Due to technical limitations or surgical considerations, we were able to obtain 18 clear responses in Zone 1, 14 in Zone 2 and 19 in Zone 3 for analysis. When stimulating the superior aspect of the trigeminal nerve REZ (Zone 1), the most common response was V1 alone (33%), however, we also recorded responses from V1 + V2 (17%), V2 alone (17%) and V3 alone (17%). In the middle portion of the trigeminal nerve (Zone 2) stimulation primarily elicited responses from V2 (36%) but also from V3 (29%). Finally, the stimulation of the inferolateral border of the trigeminal nerve resulted in responses from V3 (69%) and V2+V3 (16%). When considering isolated trigeminal nerve division responses following stimulation, we observed that after recruiting Z1 fibers, responses were evoked from V1 in 40%, V2 in 36% and V3 in 14%. Following stimulation of Z2 fibers, responses were evoked from V2 in 47%, V3 in 35% and V1 in 17%. Finally, stimulation of Z3 lead to responses from V3 in 70%, V2 in 22% and V1 in 8%. No complications were reported in association with the use of this technique.

We analyzed the responses obtained from areas where the neurovascular compression was identified: 10 cases (Zone 3), 7 cases (Zone 1) and 6 cases (Zone 2). In Zone 1, responses were evoked mainly in V1 (29%) and also in V1 + V2 (29%). When comparing with microvascular compressions in Z2, V2 was found in 50% followed by V2 – V3 (33%). Ultimately, in Zone 3, mainly V3 responses were observed in 75% of the patients. We evaluated the clinical-neurophysiologic correspondence between the pain division and the stimulated response at the neurovascular conflict zone. This correspondence yielded three categories: total correspondence ($n = 11$, 47%) was considered when all clinical divisions of the trigeminal nerve were obtained after stimulation, partial correspondence ($n = 9$, 39%) when not all clinical relevant trigeminal nerve divisions were evoked after stimulation (e.g., V3 evoked stimulation in a patient with V2 and V3 pain) and no correspondence ($n = 3$, 14%) (Fisher's exact test, $p = 0{,}417$). Only patients with total correspondence obtained a favorable outcome with a BNI of I or II in 90% of the cases ($p = 0{,}41$).

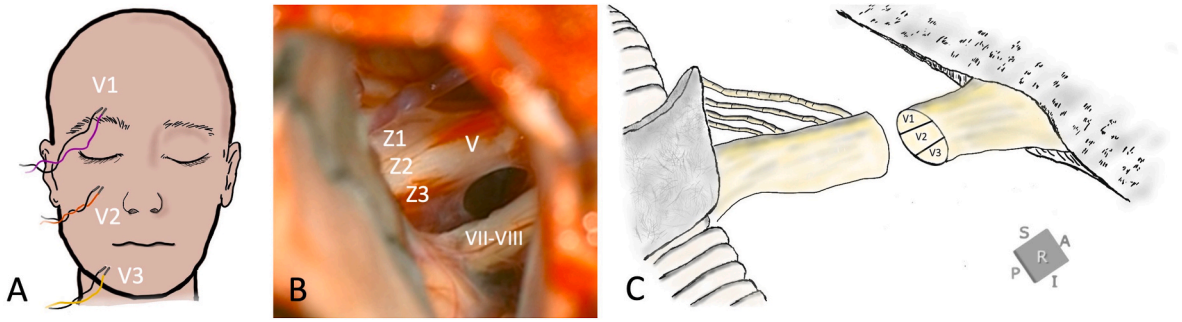

Fig. 1. A. Subdermal needle electrodes positioning schematic drawing for monitoring ophthalmic, maxillary and mandibular divisions of the trigeminal nerve. B. Intraoperative right trigeminal nerve view depicting stimulating zones (Z1, Z2 and Z3). C. Schematic representation of a right trigeminal nerve cross section with the three trigeminal nerve division topography. Trigeminal nerve (V), facial-vestibulocochlear nerve complex (VII-VIII).

Table 1
Summary of patient data.

N	Sex	Age	Side	Pain division	Disease time in months	BNI PRE	NV compression zone	Clinical and Neurophysiological correspondence	Anatomical distortion	Vessel compression	BNI POS (follow-up in months)
1	M	50	R	V3	132	IV	Z1	No	No	A	III (17m)
2	F	54	R	V3	36	IV	Z2	Yes	No	V	I (67m)
3	F	55	R	V2	72	V	Z2	Yes	Yes	V	II (64m)
4	M	63	R	V2	96	IV	Z2	Yes	Yes	A	I (63m)
5	M	44	R	V2	9	IV	Z2	Yes	No	V	I (57m)
6	M	60	R	V1	96	V	Z1	No	Yes	A	I (59)
7	F	44	R	V1 V2	240	IV	Z3	Partial	No	A	II (59m)
8	F	42	R	V3	120	IV	Z1	Yes	No	A	II (48m)
9	F	75	L	V2 V3	60	IV	Z3	Partial	Yes	A	I (36m)
10	F	24	R	V1	46	V	Z1	Yes	No	A	I (45m)
11	M	68	L	V1 V2	36	V	Z3	Partial	Yes	V	II (42m)
12	F	41	L	V2 V3	36	IV	Z1	Yes	No	A	I (43m)
13	M	63	L	V2	120	IV	Z2	Yes	No	A	II (31m)
14	F	60	R	V2 V3	17	V	Z2	Yes	Yes	A	I (29m)
15	F	48	R	V1 V2	96	V	Z1	Partial	No	A	IV (26m)
16	M	66	L	V2 V3	72	IV	Z3	Partial	Yes	A	II (22m)
17	F	51	L	V2 V3	12	V	Z1	Partial	No	A	I (22m)
18	M	63	L	V1 V2	60	V	Z3	Yes	No	A	I (17m)
19	F	57	L	V3	240	V	Z3	Yes	No	A	III (15m)
20	F	39	R	V2 V3	24	V	Z3	Partial	No	V	I (14m)
21	F	61	R	V2 V3	264	IV	Z3	Partial	Yes	A	I (11m)
22	F	65	R	V1 V2	10	IV	Z3	No	No	V	II (10m)
23	M	54	L	V1 V2	4	V	Z3	Partial	No	A	IV (7m)

BNI, Barrow Neurological Index; NV, neurovascular; A, arterial; V, venous; V1, trigeminal nerve ophthalmic division; V2, trigeminal nerve maxillary division; V3, trigeminal nerve mandibular division; Z1, trigeminal nerve zone 1 (superior/rostral); Z2, trigeminal nerve zone 2 (intermediate); Z3, trigeminal nerve zone 3 (inferior/caudal).

Table 2
Distribution of pain according to compression site.

	Neurovascular compression site			Total (%)
Pain territory	Z1	Z2	Z3	
V1	2	0	0	2 (8,7%)
V2	0	4	0	4 (17,5%)
V3	2	1	1	4 (17,5%)
V1 V2	1	0	5	6 (26%)
V2 V3	2	1	4	7 (30%)
Total (%)	7(30%)	6 (26%)	10 (44%)	23 (100%)

$p = 0{,}008$.
V1, trigeminal nerve ophthalmic division; V2, trigeminal nerve maxillary division; V3, trigeminal nerve mandibular division; Z1, trigeminal nerve zone 1 (superior/rostral); Z2, trigeminal nerve zone 2 (intermediate); Z3, trigeminal nerve zone 3 (inferior/caudal).

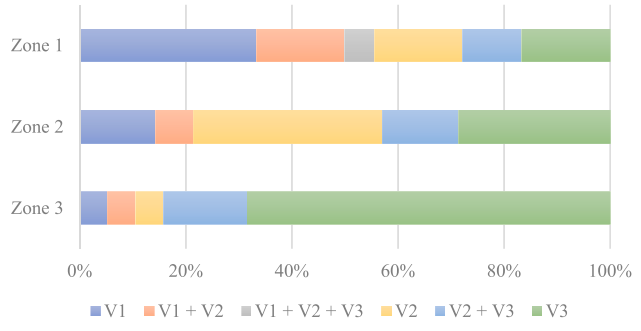

Fig. 2. Topographical mapping proportions after neurophysiological stimulation.

Furthermore, we identified 8 patients with anatomical distortion during surgery. In the assessment of vessel compression, 18 cases were attributed to arterial compression (75%), 3 to venous (12,5%) and 3 to a combination of arterial and venous compression. Patients were followed for an average duration of 34,95 months (7–67 months), with reported BNI scores distributed as follows: BNI I (57%), BNI II (30%), BNI III (9%) and BNI IV (4%).

4. Discussion

The findings of this study suggest the potential utility of intraoperative monitoring guiding surgical decisions in trigeminal nerve decompression. This is underscored by finding variations in the patterns of pain distribution in trigeminal neuralgia based on the location of the neurovascular conflict, which are consistent with descriptions provided in previous studies.[16] Additionally, the study establishes a correlation between the location of the neurovascular conflict and neurophysiological stimulation, indicating that the site of compression has a significant influence on the territory of pain experienced by patients.

A cross-section study of the REZ in animals have revealed a remarkably clear somatotopic organization with the ophthalmic fibers located ventrally and mandibular fibers dorsally with minimal intermingling.[17] An anatomical investigation conducted by Sindou et al involving 579 patients described the disposition of the trigeminal nerve REZ based on neurovascular compression as follows: supero-lateral in 53,9%, supero-medial in 31,6% and inferior in 14,5%.[18] Gudmundsson and Rhoton further delineated an anatomical division that corresponded to the typical triangular plexus division observed in radiofrequency procedures. They found that the rostral fibers most frequently entered the first division, those arising more caudally entered the third division, with the maxillary division situated between and occupying a larger proportion of the medial surface of the trigeminal sensory root as compared to the lateral surface. Notably, this disposition could vary depending on the angle between the longest diameter of the cross section and the long axis of the body, with most common angle falling between 40 and 50°.[15] These findings could explain the variations observed in our patients when stimulating the same zones but obtaining different responses. For example, the antidromic response of V3 branch was obtained in 3 patients by stimulating Zone 1. Juxtapontine rhizotomies performed by Dandy also supports this somatotopic arrange,

where complete V1 and partial V2 preservation are achieved during inferolateral rhizotomy.[19] He also described a bundle of small sensory fibers between the major sensory and motor roots in the pons and hypothesized that might be concerned with perception of light touch. In 1965, Janetta reviewed this concept and stated that it would help to explain the variable sensory loss, frequent preservation of light touch and absence of corneal complications in patients operated upon by the suboccipital approach for trigeminal neuralgia.[20] Moreover some studies have reported that only 48% experience complete sensory loss following total sensory root (portio major) section and stimulating these fibers evokes responses exclusively from the lower face.[13,21]

Compared to those experiencing pain in other territories, we found that patients with isolated V1 or V2 pain are more likely to have neurovascular compressions located superiorly at Zone 1 or intermediately at Zone 2, respectively (Fisher's exact test, $p = 0.008$) (Table 2). However, for isolated V3 pain, the compression sites tend to be more variable. When considering the opposite situation, we did not observe a direct correlation between patients experiencing compression in a specific zone and the type of pain (without considering stimulation). This finding contrast with publications from the Sindou group, which describes an odds ratio of 2.7 with superomedial compression in cases presenting V1 pain and an odds ratio of 2.56 for inferior compression in cases presenting V3 pain. One possible explanation for this inconsistency, where compressing fibers in the ventral part may cause pain in the upper face for example, is that it could be due to a distortion phenomenon that tents up and may disrupt the nerve on the opposite side of the compression.[13]

Electrophysiological confirmation was initially used as refinement tool in selective rhizotomy with the aim of eliminating all responses from the symptomatic branches. Despite some variations could occur in latency, morphology and duration, generally a typical triphasic CNAP response is obtained after trigeminal nerve stimulation.[13] As depicted in Figs. 2 and 3 stimulation of Zone 1 regions of the trigeminal REZ predominantly elicited activity in the upper face, while stimulation of Zone 3, led to activity in the lower face. Activation of the middle face was most consistently observed when stimulating the Zone 2 of the nerve root on its intermediate lateral surface. The higher response of Zone 3 and 2 could be attributed to the greater number of myelinated fibers in maxillary and mandibular division, as evidenced in histometric studies of the trigeminal nerve.[22] These findings are in agreement with previous anatomical investigations, thereby validating the results achieved through this technique.[13,14,18]

A limitation of the current study is the absence of recorded data related to accessory fiber stimulation. In certain instances, the observed outcomes involving multiple root responses may solely reflect electrical propagation rather than the "entanglement" of root fibers within a specific stimulated zone. In order to address this maybe a thinner probe should be used trying to minimize the current stimulation to avoid losing specificity. Future studies are required in order to further elucidate the role of the portio minor in TN. Besides total correspondence between clinical pain and neurophysiologic stimulation did not achieve statistical significance regarding surgical outcome, the sample size was small and it would be important to explore this clinical association in a larger cohort of patients. Such an expanded study could contribute to a more comprehensive understanding of the electrophysiological and topographical relationships within the trigeminal nerve. Ultimately, this could facilitate intraoperative decision-making to ensure accurate microvascular decompression or the identification of other vascular factors contributing to trigeminal neuralgia.

5. Conclusions

Intraoperative neurophysiological mapping of the trigeminal nerve root entry zone is a safe and practical technique that can be seamlessly integrated into microvascular decompression surgeries for trigeminal neuralgia. This approach, which incurs no significant morbidity or notable extension of surgical duration, has proven its utility in elucidating the complex relationship between neurovascular compression, pain distribution, and topographical anatomy within the trigeminal nerve. By employing this mapping technique, surgeons can gain a deeper understanding of the individualized aspects of each patient's trigeminal neuralgia, enabling more precise identification and isolation of the offending vessel responsible for the compression. This level of precision holds the potential to significantly enhance surgical outcomes, ultimately providing patients with effective and lasting relief from the debilitating pain associated with trigeminal neuralgia. Overall, the application of neurophysiological mapping in the context of microvascular decompression for trigeminal neuralgia holds great promise for improving the quality of care and outcomes for affected individuals.

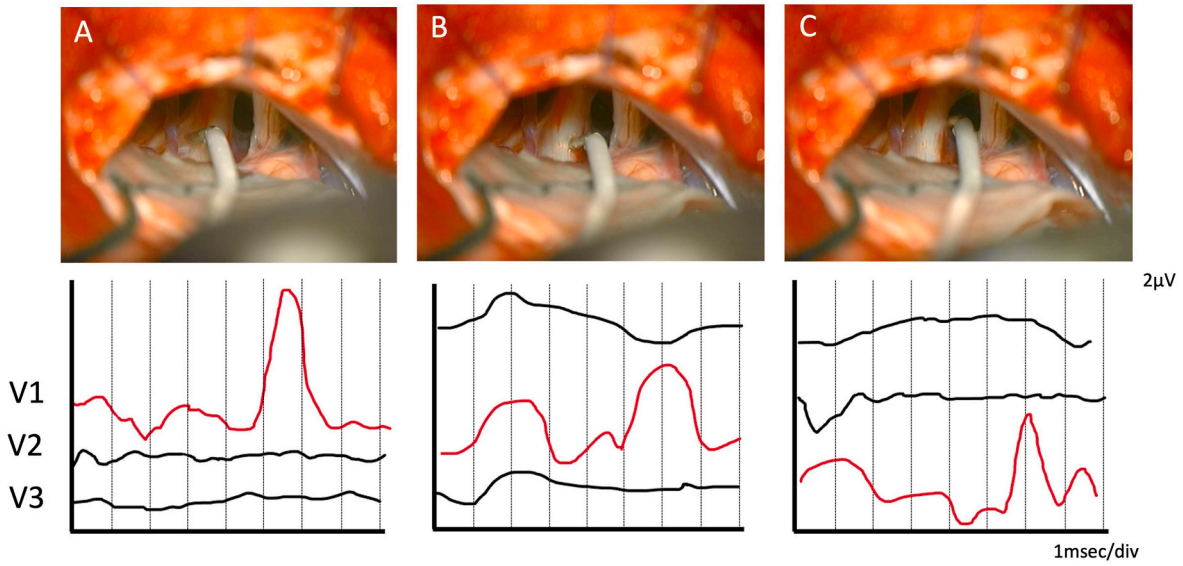

Fig. 3. Right trigeminal neuralgia microvascular decompression showing intraoperative neurophysiological mapping and responses after stimulating Zone 1 (A), Zone 2 (B) and Zone 3 (C).

CRediT authorship contribution statement

Joel Sanabria Duarte: Writing – review & editing, Writing – original draft, Investigation, Formal analysis, Data curation, Conceptualization. **Daniel Benzecry de Almeida:** Writing – review & editing, Validation, Supervision, Investigation. **Gabriella Mara Arcie:** Resources, Project administration, Formal analysis, Data curation, Conceptualization. **Mauricio Coelho Neto:** Validation, Supervision, Methodology. **Murilo Sousa de Meneses:** Visualization, Validation. **Ricardo Ramina:** Visualization, Validation.

Declaration of competing interest

None.

References

1. Zakrzewska JM, Linskey ME. Trigeminal neuralgia. *BMJ Clin Evid*. 2014;2014. http://www.ncbi.nlm.nih.gov/pubmed/25299564.
2. Cruccu G, Finnerup NB, Jensen TS, et al. Trigeminal neuralgia. *Neurology*. 2016;87(2):220–228. https://doi.org/10.1212/WNL.0000000000002840.
3. Love S. Trigeminal neuralgia: pathology and pathogenesis. *Brain*. 2001;124(12):2347–2360. https://doi.org/10.1093/brain/124.12.2347.
4. Maarbjerg S, Di Stefano G, Bendtsen L, Cruccu G. Trigeminal neuralgia – diagnosis and treatment. *Cephalalgia*. 2017;37(7):648–657. https://doi.org/10.1177/0333102416687280.
5. Obermann M, Katsarava Z. Update on trigeminal neuralgia. *Expert Rev Neurother*. 2009;9(3):323–329. https://doi.org/10.1586/14737175.9.3.323.
6. Peker S, Kurtkaya Ö, Üzün İ, Pamir MN. Microanatomy of the central myelin-peripheral myelin transition zone of the trigeminal nerve. *Neurosurgery*. 2006;59(2):354–359. https://doi.org/10.1227/01.NEU.0000223501.27220.69.
7. Joo W, Yoshioka F, Funaki T, Mizokami K, Rhoton AL. Microsurgical anatomy of the trigeminal nerve. *Clin Anat*. 2014;27(1):61–88. https://doi.org/10.1002/ca.22330.
8. Wiffen PJ, Collins S, McQuay HJ, Carroll D, Jadad A, Moore RA. Anticonvulsant drugs for acute and chronic pain. In: Wiffen PJ, ed. *Cochrane Database of Systematic Reviews*. John Wiley & Sons, Ltd; 2005. https://doi.org/10.1002/14651858.CD001133.pub2.
9. Wiffen PJ, McQuay HJ, Edwards J, Moore RA. Gabapentin for acute and chronic pain. In: Wiffen PJ, ed. *Cochrane Database of Systematic Reviews*. John Wiley & Sons, Ltd; 2005. https://doi.org/10.1002/14651858.CD005452.
10. Cruccu G, Gronseth G, Alksne J, et al. AAN-EFNS guidelines on trigeminal neuralgia management. *Eur J Neurol*. 2008;15(10):1013–1028. https://doi.org/10.1111/j.1468-1331.2008.02185.x.
11. Haridas A, Mathewson C, Eljamel S. Long-term results of 405 refractory trigeminal neuralgia surgeries in 256 patients. *Zentralblatt für Neurochir - Cent Eur Neurosurg*. 2008;69(4):170–174. https://doi.org/10.1055/s-2008-1077076.
12. Bendtsen L, Zakrzewska JM, Abbott J, et al. European Academy of Neurology guideline on trigeminal neuralgia. *Eur J Neurol*. 2019;26(6):831–849. https://doi.org/10.1111/ene.13950.
13. Stechison MT, Møller A, Lovely TJ. Intraoperative mapping of the trigeminal nerve root: technique and application in the surgical management of facial pain. *Neurosurgery*. 1996;38(1):76–82. https://doi.org/10.1097/00006123-199601000-00018.
14. Hatayama T. Intraoperative mapping of the trigeminal nerve root during MVD. *Int Congr Ser*. 2004;1259:347–352. https://doi.org/10.1016/S0531-5131(03)01407-9.
15. Gudmundsson K, Rhoton AL, Rushton JG. Detailed anatomy of the intracranial portion of the trigeminal nerve. *J Neurosurg*. 1971;35(5):592–600. https://doi.org/10.3171/jns.1971.35.5.0592.
16. Sindou M, Brinzeu A. Topography of the pain in classical trigeminal neuralgia: insights into somatotopic organization. *Brain*. 2020;143(2):531–540. https://doi.org/10.1093/brain/awz407.
17. Kerr FWL. The divisional organization of afferent fibres of the trigeminal nerve. *Brain*. 1963;86(4):721–732. https://doi.org/10.1093/brain/86.4.721.
18. Sindou M, Howeidy T, Acevedo G. Anatomical observations during microvascular decompression for idiopathic trigeminal neuralgia (with correlations between topography of pain and site of the neurovascular conflict). Prospective study in a series of 579 patients. *Acta Neurochir (Wien)*. 2002;144(1):1–13. https://doi.org/10.1007/s701-002-8269-4.
19. Dandy WE. AN operation for the cure of tic douloureux. *Arch Surg*. 1929;18(2):687. https://doi.org/10.1001/archsurg.1929.04420030081005.
20. Jannetta PJ. Gross (mesoscopic) description of the human trigeminal nerve and ganglion. *J Neurosurg*. 1967;26(1part2):109–111. https://doi.org/10.3171/jns.1967.26.1part2.0109.
21. Ley A, Guitart JM. Clinical observations on sensory effects of trigeminal dorsal root section. *J Neurol Neurosurg Psychiatry*. 1971;34(3):260–264. https://doi.org/10.1136/jnnp.34.3.260.
22. Pennisi E, Cruccu G, Manfredi M, Palladini G. Histometric study of myelinated fibers in the human trigeminal nerve. *J Neurol Sci*. 1991;105(1):22–28. https://doi.org/10.1016/0022-510X(91)90113-L.

Abbreviations

TN: Trigeminal Neuralgia
REZ: Root Entry Zone
CNAP: Compound Nerve Action Potential
BNI: Barrow Neurological Index