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Trigeminal root dislocation subtends focal demyelination of primary trigeminal afferents in patients with trigeminal neuralgia

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ABSTRACT

Objective: Trigeminal neuralgia (TN) is a neuropathic pain condition associated with focal demyelination of primary trigeminal afferents. Although MRI is routinely used in TN, the specific anatomical abnormality of the trigeminal root that reflects focal demyelination remains unclear. In this combined clinical, neurophysiological and neuroimaging study, we aimed to identify whether specific clinical features and trigeminal root abnormalities predict trigeminal reflex asymmetry, which may reflect unilateral focal demyelination of primary trigeminal afferents.

Methods: We retrospectively analysed clinical, neurophysiological and neuroimaging data from 78 patients with primary TN (67 classical, 11 idiopathic). Logistic regression was used to assess whether trigeminal root abnormalities and clinical features predicted trigeminal reflex asymmetry. Reflex asymmetry was defined as latency differences exceeding 0.1 ms between affected and unaffected sides in the early component of the blink or masseter inhibitory reflex, selected according to the trigeminal division involved. Trigeminal root volumes and dislocation were independently assessed by blinded neuroradiologists.

Results: Severe trigeminal root dislocation predicted trigeminal reflex asymmetry ($p < 0.05$). Conversely, trigeminal root atrophy was not associated with asymmetry.

Conclusions: This retrospective, clinical, neurophysiological, and neuroimaging study in patients with TN showed that a severe trigeminal root dislocation predicted trigeminal reflex asymmetry.

Significance: This suggests that root dislocation may underlie focal demyelination in TN.

1. Introduction

Trigeminal neuralgia (TN) is a neuropathic pain condition characterized by sudden, unilateral, paroxysmal pain in the trigeminal nerve territory, evoked by tactile innocuous stimulation of trigger zones. Focal demyelination of primary trigeminal afferents plays a central role in the pathophysiology of these pain paroxysms, as it promotes hyperexcitability and facilitates the ectopic generation of high-frequency discharges (Cruccu et al., 2020).

TN is classified based on its aetiology into secondary TN, related to major neurological diseases, classical TN, associated with neurovascular compression causing anatomical changes of the trigeminal root, and idiopathic TN, of unknown aetiology (Cruccu et al., 2016; Bendtsen et al., 2019; Bendtsen et al., 2020). In the diagnostic process, magnetic resonance imaging (MRI) is essential to identify dislocation and atrophy of the trigeminal root, the morphologic changes required to distinguish between simple contact and compression (Antonini et al. 2014). Which of these anatomical abnormalities is directly related to the focal demyelination of primary trigeminal afferents, however, remains an open question.

Trigeminal reflex testing, the established neurophysiological assessment of the trigeminal nerve, investigates large myelinated Aβ fibres and is commonly used in patients with TN. This neurophysiological technique, by reflecting trigeminal afferents damage or alterations in the interneurons and relay nuclei in the reflex pathway, in patients with TN can reveal the focal demyelination of primary trigeminal

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afferents (Truini et al, 2023).

This clinical, neurophysiological, and neuroimaging study was designed to identify a neuroimaging marker subtending the focal demyelination of primary trigeminal afferents in patients with TN. The aim is to enhance our understanding of morphological changes of the trigeminal root associated with focal demyelination. To achieve this, we retrospectively analysed trigeminal reflex and neuroimaging data in consecutive patients with classical and idiopathic TN. Assuming that trigeminal reflex latencies may reflect the demyelinating process affecting trigeminal afferents at the site of neurovascular compression, we implemented a logistic regression model to determine whether latency asymmetry of the trigeminal reflex was predicted by neuroimaging findings, specifically atrophy and dislocation of the trigeminal nerve root. Clinical characteristics were systematically collected to identify possible associations with trigeminal reflex asymmetry.

2. Methods

2.1. Study cohort and design

We retrospectively reviewed clinical records of consecutive patients with a definite diagnosis of TN referred to the Peripheral Neuropathy and Neuropathic Pain Unit at the Department of Human Neuroscience, Sapienza University of Rome between December 2019 and December 2023. The diagnosis of TN was independently confirmed by two clinicians (AT, GDiS) in accordance with accepted criteria (ICHD, 2018; ICOP, 2020) defining classical and idiopathic TN. Exclusion criteria includes secondary TN, history of previous surgical procedures for TN (including peripheral nerve blocks), other orofacial pain conditions, cognitive impairments, and communication barriers. Written informed consent was obtained prospectively from participants as part of a standardized institutional protocol for clinical data use (0162/22).

A total of 118 patients with a definite diagnosis of TN were screened. We excluded 32 patients with TN related to multiple sclerosis, six with a benign tumour of the posterior fossa, and two with bilateral abnormal trigeminal reflex testing suggesting trigeminal neuropathy. The remaining 78 patients with primary TN were categorized into classical and idiopathic groups. Classical TN was defined by neurovascular compression and significant anatomical abnormalities of the trigeminal nerve root, such as dislocation and/or atrophy. Idiopathic TN was defined by the absence of neurovascular compression or by minor morphological changes on the trigeminal nerve root caused by neurovascular conflict (Maarbjerg et al., 2015; Hughes et al., 2016).

2.2. Clinical and neurophysiological tests

Each patient underwent a neurological examination including precise sensory profiling using bedside tools (Cruccu et al, 2004) and evaluation of topographical distribution of pain. Clinical information was systematically collected through a dedicated questionnaire (Di Stefano et al., 2020a) focused on demographics, pain distribution, quality of pain, occurrence of remission periods, familial occurrence, association with autonomic symptoms, and comorbidities.

In all patients, large myelinated Aß fibre function was assessed through trigeminal reflexes (Cruccu et al., 2005; Bendtsen et al., 2019), including the blink reflex after electrical stimulation of the supraorbital nerve and the masseter inhibitory reflex after electrical stimulation of the infraorbital and mental nerves (EMG Sierra Summit, Cadwell Industries Inc). Stimulation and recording adhered to the recommendations for clinical practice of the International Federation of Clinical Neurophysiology (Deuschl et al, 1999).

For the blink reflex, we measured the latency of R1, the direct and crossed R2 responses, as well as the difference between sides. For the masseter inhibitory reflex, we measured the onset latency of the silent period corresponding to the beginning of the EMG suppression (Deuschl and Eisen, 1999) and duration of SP1 and SP2 responses after stimulation of each side, as well as the difference between sides. We subsequently restricted our analysis to the early reflex components (R1 and SP1), as these are less influenced by central modulation and are therefore considered to more accurately reflect the function of primary trigeminal afferents (Cruccu and Deuschl, 2000). For the blink reflex, four non-averaged trials were collected, to measure the latency of R1 component as well as its difference between sides. For the masseter inhibitory reflex eight, non-averaged trials per side were recorded to measure the onset latency of SP1 component after stimulation of each side and their difference between sides (Deuschl and Eisen, 1999). In patients with pain in V1, the onset latency of the R1 was selected as the main neurophysiological measure. In patients with pain in V2 or V3, the onset latency of the SP1 after electrical stimulation of the infraorbital or mental nerve respectively were selected as the main neurophysiological measures. In patients with more than one division affected, we calculated accordingly the mean of the latencies of the neurophysiological measures of interest. The latency difference between the two sides was calculated by subtracting the latency of the healthy side from the latency of the affected side. Values of asymmetry between the affected and not affected side greater than 0.1 ms were considered abnormal. Although this asymmetry is far smaller than the asymmetry considered abnormal for clinical purposes (Cruccu and Deuschl, 2000), we chose this value to guarantee the maximum sensitivity in the analysis of association with radiological variables. We applied this same cutoff to both the blink reflex and the masseter inhibitory reflex to obtain a binary outcome parameter reflecting the latency asymmetry of affected divisions.

2.3. Neuroimaging protocol

Patients underwent magnetic resonance imaging (MRI) performed using a Siemens Verio 3 Tesla scanner (Erlangen, Germany) with a dedicated 12-channel head coil. The imaging protocol was designed to identify neurovascular conflict and morphological alterations of the trigeminal nerve roots. Specifically, we acquired three-dimensional constructive interference in steady-state (CISS) sequences with the following parameters: repetition time (TR) 1000 ms, echo time (TE) 132 ms, field of view (FOV) 200 × 200 mm, 0.5 mm slice thickness, 56 slices, and a matrix size of 384 × 384. These axial-oriented images encompassed bilateral trigeminal nerve roots, ensuring clear differentiation between hypointense cranial nerves and vessels against the hyperintense cerebrospinal fluid background (Chavhan et al., 2008).

To enhance differentiation between vascular structures and the trigeminal nerve root, we also obtained a three-dimensional time-of-flight magnetic resonance angiography (TOF-MRA), characterized by TR 22 ms, TE 3.60 ms, FOV 181 × 200 mm, slice thickness 0.5 mm, and a matrix of 696 × 768, focused on posterior fossa structures. Neurovascular compression was diagnosed if direct vessel-nerve root contact was observed without intervening cerebrospinal fluid, accompanied by morphological nerve root alterations.

Quantitative assessment of trigeminal nerve root atrophy was conducted through volumetric analysis using Medical Image Processing, Analysis, and Visualization (MIPAV) software (https://mipav.cit.nih.gov/), as previously described (Di Stefano et al., 2020b). Two independent neuroradiologists, blinded to clinical details, separately measured trigeminal nerve root volumes bilaterally for each patient. The mean of their measurements was used for analysis. Consistent with earlier findings indicating that healthy individuals typically have nerve asymmetry below 5% (Di Stefano et al., 2020b), we classified atrophy severity into four groups: none (<5% asymmetry), mild (5–10%), moderate (10–20%), and severe (>20%), an approach used in previous literature (De Stefano et al., 2023).Trigeminal root dislocation is a distinctive finding of TN, being reported only in a minority (9%) of healthy subjects (Di Stefano et al., 2020b). Dislocation was assessed on axial imaging by tracing a line from the nerve's exit at Meckel's cave to its entry into the pons. Dislocation was defined as significant deviation from this baseline, quantified as the greatest perpendicular distance

from the nerve margin to the reference line. Patients exhibiting dislocation were categorized for analytical purposes into moderate (<3 mm) or severe (>3 mm), aligning with criteria previously established in the literature (Antonini et al., 2014).

2.4. Statistical analysis

Descriptive summaries were reported as frequencies and percentages for categorical variables and as means ± standard deviations for numerical variables. Preliminary comparisons between patients with and without trigeminal reflex asymmetry were performed using Student's t or Mann-Whitney test for numerical variables, and Chi- squared or Fisher's exact tests for categorical variables. A logistic regression model was then used to assess the association between reflex asymmetry (defined as a binary outcome based on R1 blink reflex and SP1 masseter inhibitory reflex latency asymmetry) and potential predictors, including gender, pain quality (paroxysmal pain only or with concomitant continuous pain), disease duration, trigeminal root volume on the affected side, and the degree of atrophy and dislocation. Model performance in predicting asymmetry was evaluated using the receiver operating characteristic (ROC) curve and the area under the curve (AUC). All statistical tests were two-tailed and a p-value < 0.05 was deemed to be indicative of significant results. The statistical analysis was conducted in the R environment (R Core Team 2025).

3. Results

We included 78 patients in the analysis (25 men, 53 women; mean age 66.4 ± 12.1 years; mean disease duration 7.3 ± 6.4 years; 57 with classical and 21 with idiopathic TN.

Pain was more often located on the right (63%) than left side (37%), and in the second (68%) and the third (56%) trigeminal divisions than the first trigeminal division (29%). Concomitant continuous pain was reported by 32 patients (41%).

Of the 78 patients included in this study, 25 (32%) had a trigeminal reflex asymmetry, characterized by a longer latency of the R1 blink reflex and the SP1 masseter inhibitory reflex on the affected side compared to the healthy side.

The only variable distinguishing patients with and without trigeminal reflex asymmetry was the presence of severe dislocation (greater than 3 mm) ($p < 0.05$) (Table 1).

The logistic regression model confirmed that a severe dislocation of the trigeminal root was predictive of trigeminal reflex asymmetry ($p < 0.05$) (Table 2). Conversely, trigeminal root volume on the affected side and the level of atrophy were not associated with trigeminal reflex asymmetry. Clinical variables including disease duration and presence of concomitant continuous pain were not predictive of trigeminal reflex asymmetry. The ROC curve analysis, used to assess the model's performance in predicting asymmetry, yielded an area under the curve (AUC) of 0.73. (Fig. 1).

Table 1
Clinical and neuroimaging characteristics of the full sample and by the presence of trigeminal reflex asymmetry. Significant p-values for tests comparing the two groups are in bold.

	Full sample N = 78	Without trigeminal reflex asymmetry n = 53 (67.9%)	With trigeminal reflex asymmetry n = 25 (32.1%)	*p*-value
Female, N (%)	53 (67.9)	38 (71.7)	15 (60.0)	0.439
Male, N (%)	25 (32.1)	15 (28.3)	10 (40.0)	
With purely paroxysmal pain, N (%)	46 (59.0)	32 (60.4)	14 (56.0)	0.904
With concomitant continuous pain, N (%)	32 (41.0)	21 (39.6)	11 (44.0)	
Duration of disease, years, mean ± sd	7.3 ± 6.4	7.4 ± 6.5	7.2 ± 6.4	0.919
Absence of atrophy of trigeminal root, N (%)	24 (30.8)	18 (34.0)	6 (24.0)	0.274
Mild or moderate atrophy, N (%)	22 (28.2)	12 (22.6)	10 (40.0)	
Severe atrophy, N (%)	32 (41.0)	23 (43.4)	9 (36.0)	
Trigeminal root volume of affected side, mm^3, mean ± sd	43.1 ± 15.9	41.4 ± 15.6	46.8 ± 16.4	0.165
Without severe dislocation of trigeminal root, N (%)	63 (80.8)	47 (88.7)	16 (64.0)	**0.015**
With severe dislocation, N (%)	15 (19.2)	6 (11.3)	9 (36.0)	

For categorical variables: *p*-values refer to the Chi-squared or exact Fisher's test, as appropriate.

Table 2
Odds ratios and 95% confidence intervals of the estimated logistic regression model for prediction of asymmetry. Significant p-values are in bold.

	Odds ratio	95% CI	*p*-value
Male *(vs female)*	1.06	[0.30, 3.54]	0.923
With concomitant continuous pain *(vs with purely paroxysmal pain)*	1.21	[0.39, 3.76]	0.740
Duration of disease	1.01	[0.93, 1.10]	0.758
With severe trigeminal root dislocation *(vs without)*	4.69	[1.36, 17.58]	**0.016**
Mild or moderate trigeminal root atrophy *(vs absence)*	2.10	[0.55, 8.46]	0.281
Severe atrophy *(vs absence)*	1.13	[0.27, 4.89]	0.864
Trigeminal root volume of affected side	1.02	[0.98, 1.07]	0.225

For numerical variables: *p*-value refers to the *t*-test or Mann-Whitney test, as appropriate.

4. Discussion

In this clinical, neurophysiological, and neuroimaging study, we found that in a sample of 78 patients with primary TN, a severe dislocation of trigeminal root predicted trigeminal reflex asymmetry. The other neuroimaging variables, including trigeminal root volume on the affected side and atrophy of the nerve, were not predictive of trigeminal reflex asymmetry. Similarly, no clinical data were associated with the asymmetry of neurophysiological responses.

Trigeminal reflexes, including the blink reflex and the masseter inhibitory reflex, are the recommended techniques for the assessment of trigeminal afferent damage (Truini et al, 2023). In patients with postherpetic neuralgia, the blink reflex delay correlated with the intensity of paroxysmal pain, related to large, myelinated Aß fibres damage (Truini et al., 2008). By testing the function of this set of fibres, trigeminal reflex testing in trigeminal neuralgia detects the damage related to focal demyelination of primary trigeminal afferents (Bendtsen et al, 2019). In a recent study, latency asymmetry in trigeminal reflexes was found to be predictive of classical trigeminal neuralgia (TN) (De Stefano et al., 2025). This finding supports the hypothesis that trigeminal reflex asymmetry may indirectly subtend the focal demyelination of primary trigeminal afferents.

Our findings show a significant association between trigeminal reflex

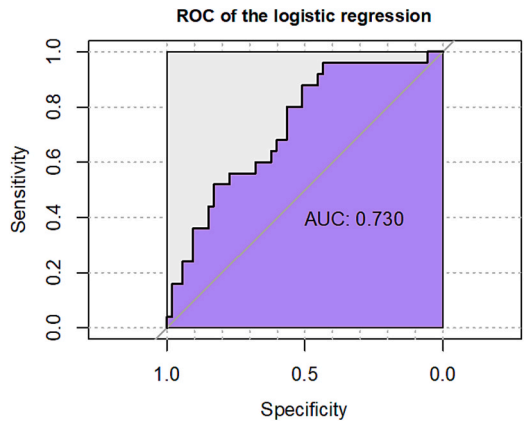

Fig. 1. ROC curve and AUC (0.73, 95%CI [0.64,0.83]) of the logistic regression for prediction of trigeminal reflex asymmetry.

asymmetry and severe trigeminal root dislocation. This morphological abnormality may indirectly subtend the focal demyelination of the trigeminal afferents. The demyelination of primary trigeminal afferents is considered able to increase the susceptibility of the primary trigeminal afferent to ectopic excitation, ephaptic transmission, and high-frequency discharges, key pathophysiological hallmarks of trigeminal neuralgia (Cruccu et al, 2020).

This finding aligns with existing literature demonstrating an association between severe neurovascular compression and the long-term success of surgical interventions. Notably, the presence of severe vascular compression has been shown to predict a success rate exceeding 90% for microvascular decompression (Sindou et al., 2007; Hughes et al., 2019). Therefore, according to the evidence in literature, the combined identification of trigeminal reflex asymmetry and severe trigeminal root dislocation may aid in selecting optimal candidates for microvascular decompression.

While we found a significant association between trigeminal reflex asymmetry and root dislocation, no such relationship was observed between reflex asymmetry and trigeminal root atrophy. Previous literature reported an association between trigeminal root atrophy on the affected side and the development of concomitant continuous pain in patients with trigeminal neuralgia (Di Stefano et al., 2020b). Furthermore, a neurophysiological study demonstrated that concomitant continuous pain in trigeminal neuralgia is associated with unmyelinated C-fibre damage, as assessed by laser-evoked potentials, suggesting that this pain phenotype may be related to axonal degeneration and aberrant activity in denervated second-order trigeminal neurons (De Stefano et al., 2022). In line with this interpretation, previous studies have shown that trigeminal root atrophy is associated with a significantly poorer outcome following microvascular decompression (Duan et al., 2015). We found that trigeminal reflex asymmetry was not associated with clinical variables, including disease duration. This finding aligns with the view that trigeminal neuralgia typically plateaus in terms of structural nerve damage, as it is driven by focal, non-progressive dysfunction—commonly resulting from neurovascular compression—rather than by a degenerative disease process. Clinical evidence supports this interpretation: patients often exhibit stable symptoms over many years, MRI findings remain unchanged over time, and surgical interventions, such as microvascular decompression, can effectively relieve pain without repairing the nerve itself. These observations reinforce the concept that compression-induced dysfunction, rather than progressive nerve injury, underlies the pathophysiology of the condition.

The relatively small, single-center sample size may restrict generalizability. Retrospective data collection may represent a limitation of this study, mitigated by the application of established diagnostic criteria (ICHD, 2018; ICOP, 2020), the use of our structured interview approach (Di Stefano et al, 2020a) and utilization of standardized neuroimaging and neurophysiological methods. However, we cannot exclude that the retrospective design may limit causal inference and may introduce selection bias. Another limit concerns the assumption of focal demyelination, based on neurophysiological findings rather than direct histopathological or advanced myelin MRI evidence. Although we considered the asymmetry of trigeminal reflex latencies as marker of demyelination, reasonably reflecting the focal demyelination of the primary trigeminal afferent, we cannot exclude the impact of alterations in the interneurons and relay nuclei in the reflex pathway. However, the absence of efferent or crossed abnormalities supports our assumption.

We measured reflex asymmetry in the latency only since this outcome variable is the reliable neurophysiological parameter routinely measured in clinical practice. However, we cannot exclude that this analysis may have overlooked the effect of phase cancellation or response size changes related to neuronal integration of asynchronous input volleys in the context of transsynaptic reflex responses.

5. Conclusions

Among the morphological changes of the trigeminal root, a severe dislocation is predictive of trigeminal reflex asymmetry—a neurophysiological marker compatible with focal demyelination of primary trigeminal afferents. This association suggests that trigeminal root dislocation may indirectly subtend focal demyelination in patients with TN. Conversely, trigeminal root atrophy is not associated with trigeminal reflex asymmetry.

7. Ethical board approval

The study was approved by the local Ethics Committee (Ref. 0162/22).

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Declaration of competing interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

References

Antonini, G., Di Pasquale, A., Cruccu, G., Truini, A., Morino, S., Saltelli, G., et al., 2014. Magnetic resonance imaging contribution for diagnosing symptomatic neurovascular contact in classical trigeminal neuralgia: a blinded case-control study and meta-analysis. Pain 155, 1464–1471. https://doi.org/10.1016/j.pain.2014.04.020.

Bendtsen, L., Zakrzewska, J.M., Abbott, J., Braschinsky, M., Di Stefano, G., Donnet, A., et al., 2019. European Academy of Neurology guideline on trigeminal neuralgia. Eur. J. Neurol. 26, 831–849. https://doi.org/10.1111/ene.13950.

Bendtsen, L., Zakrzewska, J.M., Heinskou, T.B., Hodaie, M., Leal, P.R.L., Nurmikko, T., et al., 2020. Advances in diagnosis, classification, pathophysiology, and management of trigeminal neuralgia. Lancet Neurol. 19, 784–796. https://doi.org/10.1016/S1474-4422(20)30233-7.

Chavhan, G.B., Babyn, P.S., Jankharia, B.G., et al., 2008. Steadystate MR imaging sequences: physics, classification, and clinical applications. Radiographics 28, 1147–1160. https://doi.org/10.1148/rg.284075031.

Cruccu, G., Deuschl, G., 2000 Mar. The clinical use of brainstem reflexes and hand-muscle reflexes. Clin. Neurophysiol. 111 (3), 371–387.

Cruccu, G., Anand, P., Attal, N., et al., 2004. EFNS guidelines on neuropathic pain assessment. Eur. J. Neurol. 11, 153–162. https://doi.org/10.1111/j.1468-1331.2004.00791.x.

Cruccu, G., Iannetti, G.D., Marx, J.J., Thoemke, F., Truini, A., Fitzek, S., Galeotti, F., Urban, P.P., Romaniello, A., Stoeter, P., Manfredi, M., Hopf, H.C., 2005. Brainstem reflex circuits revisited. Brain 128, 386–394. https://doi.org/10.1093/brain/awh366.

Cruccu, G., Finnerup, N.B., Jensen, T.S., Scholz, J., Sindou, M., Svensson, P., et al., 2016. Trigeminal neuralgia: new classification and diagnostic grading for practice and

research. Neurology 87, 220–228. https://doi.org/10.1212/WNL.0000000000002840.
Cruccu, G., Di Stefano, G., Truini, A., 2020. Trigeminal Neuralgia. N. Engl. J. Med. 20 (383), 754–762. https://doi.org/10.1056/NEJMra1914484.
De Stefano, G., Leone, C., Di Pietro, G., Esposito, N., Falco, P., Galosi, E., Litewczuk, D., Mollica, C., Truini, A., Di Stefano, G., 2022 Oct. Unravelling the role of unmyelinated nerve fibres in trigeminal neuralgia with concomitant continuous pain. Clin. Neurophysiol. 142, 52–58. https://doi.org/10.1016/j.clinph.2022.07.502. Epub 2022 Aug 5.
De Stefano, G., Litewczuk, D., Mollica, C., Di Pietro, G., Galosi, E., Leone, C., Falco, P., Tullo, M.G., Caramia, F., Truini, A., Di Stefano, G., 2023 Dec. Sex differences in trigeminal neuralgia: a focus on radiological and clinical characteristics. Neurol. Sci. 44 (12), 4465–4472. https://doi.org/10.1007/s10072-023-06923-5. Epub 2023 Jul 12.
De Stefano, G., Mollica, C., Leone, C., Galosi, E., Di Pietro, G., Falco, P., Esposito, N., Litewczuk, D., Evangelisti, E., Caramia, F., Truini, A., Di Stefano, G., 2025 Mar. Trigeminal reflex testing abnormalities as a predictive model for distinguishing classical and idiopathic trigeminal neuralgia. Clin. Neurophysiol. 171, 61–66. https://doi.org/10.1016/j.clinph.2024.12.025.
Deuschl, G., Eisen, A., 1999. Long-latency reflexes following electrical nerve stimulation. the International Federation of Clinical Neurophysiology. Electroencephalogr. Clin. Neurophysiol. Suppl. 52, 263–268.
Di Stefano, G., Yuan, J.H., Cruccu, G., Waxman, S.G., Dib-Hajj, S.D., Truini, A., 2020a. Familial trigeminal neuralgia - a systematic clinical study with a genomic screen of the neuronal electrogenisome. Cephalalgia 40 (8), 767–777. https://doi.org/10.1177/0333102419897623.
Di Stefano, G., De Stefano, G., Leone, C., Cruccu, G., Tardioli, S., Cartocci, G., et al., 2020b. Concomitant continuous pain in patients with trigeminal neuralgia is associated with trigeminal nerve root atrophy. Cephalalgia 40, 1502–1510. https://doi.org/10.1177/0333102420949206.
Duan, Y., Sweet, J., Munyon, C., Miller, J., 2015 Dec. Degree of distal trigeminal nerve atrophy predicts outcome after microvascular decompression for Type 1a trigeminal neuralgia. J. Neurosurg. 123 (6), 1512–1518. https://doi.org/10.3171/2014.12.JNS142086.
Headache Classification Committee of the International Headache Society (IHS). The International Classification of Headache Disorders (ICHD), 3rd edition. Cephalalgia 2018;38:1–211. doi:10.1177/0333102417738202.
International Classification of Orofacial Pain, 2020. 1st edition (ICOP). Cephalalgia 40, 129–221. https://doi.org/10.1177/0333102419893823.
Hughes, M.A., Frederickson, A.M., Branstetter, B.F., Zhu, X., Sekula Jr., R.F., 2016. MRI of the trigeminal nerve in patients with trigeminal neuralgia secondary to vascular compression. AJR Am. J. Roentgenol. 206 (3), 595–600. https://doi.org/10.2214/AJR.14.14156.
Hughes MA, Jani RH, Fakhran S, et al. Significance of degree of neurovascular compression in surgery for trigeminal neuralgia. J Neurosurg. 2019;133(2):411-416. 2019. doi:10.3171/2019.3.JNS183174.
Maarbjerg, S., Wolfram, F., Gozalov, A., Olesen, J., Bendtsen, L., 2015. Significance of neurovascular contact in classical trigeminal neuralgia. Brain 138 (Pt 2), 311–319. https://doi.org/10.1093/brain/awu349.
R Core Team, 2025. R: a language and environment for statistical computing. R Foundation for Statistical Computing, Vienna, Austria https://www.R-project.org/.
Sindou, M., Leston, J., Decullier, E., Chapuis, F., 2007 Dec. Microvascular decompression for primary trigeminal neuralgia: long-term effectiveness and prognostic factors in a series of 362 consecutive patients with clear-cut neurovascular conflicts who underwent pure decompression. J. Neurosurg. 107 (6), 1144–1153. https://doi.org/10.3171/JNS-07/12/1144.
Truini A, Galeotti F, Haanpaa M, Zucchi R, Albanesi A, Biasiotta A, Gatti A, Cruccu G. Pathophysiology of pain in postherpetic neuralgia: a clinical and neurophysiological study. Pain. 2008 Dec;140(3):405-410. doi: 10.1016/j.pain.2008.08.018.
Truini, A., Aleksovska, K., Anderson, C.C., Attal, N., Baron, R., Bennett, D.L., et al., 2023. Joint European Academy of Neurology-European Pain Federation-Neuropathic Pain special Interest Group of the International Association for the Study of Pain guidelines on neuropathic pain assessment. Eur. J. Neurol. 30, 2177–2196. https://doi.org/10.1111/ene.15831.