



“PATHOPHYSIOLOGICAL AND CLINICAL ASPECTS OF TRIGEMINAL NEURALGIA OF PREDOMINANTLY CENTRAL ORIGIN”

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Annotation: Among the most frequent etiological factors contributing to the development of trigeminal neuralgia of predominantly central origin are vascular disorders—most notably hypertension—rheumatism, traumatic brain injury, and infectious diseases. Lesions of the central nervous system, including arachnoiditis and encephalitis, as well as acute cerebrovascular accidents, also play a significant role. Additional predisposing factors include the climacteric period, chronic bacterial (such as tonsillitis) and viral infections (influenza, ARVI), intoxication with various toxic substances (lead, copper, arsenic), endogenous intoxications, and endocrine pathologies. The condition most commonly affects individuals between 40 and 60 years of age.

Keywords: Trigeminal neuralgia, central origin, vascular disorders, cerebrovascular diseases, pathogenesis, neural pain, cranial nerves, hypertension, neuroinflammation, middle age.

Introduction: Clinical Manifestations of Trigeminal Neuralgia of Predominantly Central Origin
The condition is characterized by the occurrence of acute, sharp, paroxysmal facial pain, often described by patients as a sensation resembling an "electric shock." Pain paroxysms last from several seconds to a few minutes, with variable frequency. The attacks may occur spontaneously, but are more frequently provoked by facial muscle movement, such as during speaking, eating, washing, or shaving. During an episode, patients typically freeze in a particular posture, avoid movement, and may hold their breath or breathe shallowly, compressing or rubbing the affected area with their fingers. Some individuals exhibit involuntary chewing or smacking movements. The pain is localized within specific regions of the face innervated by one of the branches of the trigeminal nerve—most commonly the second (maxillary) or third (mandibular) branches, and less frequently the first (ophthalmic). The attacks are often accompanied by autonomic symptoms such as facial flushing, lacrimation, rhinorrhea, and hypersalivation (or, rarely, xerostomia). Hyperkinesis of facial muscles—such as twitching of the chin, eyelids, or other muscles—may also occur. The pain ceases abruptly.

In 84% of cases, trigeminal neuralgia of central origin is characterized by the presence of trigger (or "pain-initiating") zones—small areas of the skin or oral mucosa, irritation of which provokes a pain attack. On the facial skin, these trigger zones are typically localized near the nasolabial region, particularly around the lips and nose. Intraoral trigger zones may also be present on the palate, alveolar processes of the maxilla and mandible, or other mucosal sites, always on the affected side of the nerve. Ballé's pain points correspond to the anatomical locations where the branches of the trigeminal nerve emerge through bone foramina into the soft tissues of the face. Some patients experience a preceding autonomic aura—such as localized hyperthermia or lacrimation—before the onset of pain. Others report paresthesia, described as tingling, itching, or "crawling sensations."

According to Kränzle (1977), a correlation exists between fluctuations in blood pressure and pain paroxysms, with attack frequency increasing alongside atmospheric pressure elevations.

Between attacks, patients are typically symptom-free; however, prolonged disease progression may lead to trophic changes on the affected side, including dry skin, eyelash loss, seborrheic



eczema, hyperpigmentation, and even atrophy of masticatory or, less commonly, facial muscles. Occasionally, patients experience persistent dull, stabbing, or pressing pain between paroxysms. Bilateral trigeminal neuralgia of central origin may occur in some cases, with symptoms mirroring unilateral involvement. Pain can initially appear on one side and later on the other, or simultaneously on both sides. The second and third branches are most commonly affected. The disease course may range from several months to many years, sometimes lasting decades.

Treatment of Trigeminal Neuralgia of Central Origin is carried out by neurologists.

First of all, to relieve pain, antiepileptic drugs are prescribed, such as **Tegretol (Finlepsin), Ethosuximide, Morpholep, Trimetin, and Clonazepam.**

Tegretol (Carbamazepine, Finlepsin) is prescribed on the first day at a dose of **0.1–0.2 g twice daily**. The daily dose is gradually increased by 0.1 g until reaching a maximum of **0.6–0.8 g per day** (in 3–4 doses). The therapeutic effect usually occurs on the **2nd–3rd day** of treatment. After pain subsides, the dose is gradually reduced by 0.1 g per day to a maintenance dose of **0.1–0.2 g daily**. The course of treatment lasts **3–4 weeks**. Before discharge, the dose is reduced to the minimum that prevents pain recurrence.

Ethosuximide (Suxilep, Ronton) is prescribed at an initial dose of **0.25 g per day**, gradually increasing to **0.5–1.0 g daily** (in 3–4 doses), maintaining this level for several days, and then slowly decreasing back to **0.25 g daily**. Treatment lasts **3–4 weeks**.

A course of **nicotinic acid therapy** is also recommended. It is administered intravenously as a **1% solution**, starting from **1 ml (diluted in 10 ml of 40% glucose solution)**. The dose is increased by 1 ml daily, reaching **10 ml on the 10th day**, then gradually decreased. Nicotinic acid must be administered slowly, after meals, with the patient in a supine position, as it can lower blood pressure.

Conservative treatment also includes B-group vitamins, antihistamines, biogenic stimulants (FIBS, Aloe, Biosed, Peloidin, etc.), hypotensive and antispasmodic drugs.

Physiotherapy (electrophoresis or phonophoresis with analgesics or anesthetics, diadynamic currents, UV irradiation, UHF therapy, etc.) may be prescribed as indicated.

Surgical treatment of trigeminal neuralgia of central origin by maxillofacial surgeons generally does not produce positive results.

Differential Diagnosis of Trigeminal Neuralgia

Several syndromes and ganglionitis conditions can mimic trigeminal neuralgia.

Below are the key distinguishing features:

Pterygopalatine Ganglionitis (Sluder's Syndrome)

Characterized by sharp pain in the **periorbital region, eyeball, nasal root, upper and lower jaws**, and sometimes in the **teeth**.

Pain radiates to the **tongue, palate, temple, occiput, ear, neck, and shoulder**.

Autonomic symptoms include **facial hyperemia, edema, lacrimation, and rhinorrhea**.

Pain ceases after **topical anesthesia of the posterior nasal cavity**, which serves as a diagnostic criterion.

Semilunar (Gasserian) Ganglionitis

This is an inflammation of the **sensory ganglion** of the trigeminal nerve, located in Meckel's cave on the anterior surface of the temporal bone pyramid.

It is triggered by **infections, vascular disorders, or intoxications**.

Characterized by **multi-focal pain** involving all three branches of the trigeminal nerve, often radiating to one side of the head, with **sensory disturbances** and **herpetic eruptions** in the affected areas.



Ciliary (Oppenheim's) Ganglionitis

Manifests with **acute orbital pain**, usually at night, accompanied by **lacrimation, photophobia, rhinorrhea, and conjunctival hyperemia**.

Pain may last from 30 minutes to several hours.

Herpetic eruptions may appear on the **forehead and nose**, sometimes with **keratitis or conjunctivitis**.

Nasociliary Neuralgia (Charlin's Syndrome)

Characterized by **severe orbital and nasal pain**, often nocturnal, with **autonomic symptoms** such as lacrimation and rhinorrhea.

Palpation of the **inner eye corner or nasal side** induces pain.

Herpetic lesions and **keratoconjunctivitis** are common.

Pain relief after **anesthesia of the anterior nasal cavity** confirms the diagnosis.

Otic Ganglionitis

Presents with **burning pain** in front of the **external auditory canal** and in the **temporal region**, radiating to the **jaw and neck**.

Pain attacks last from minutes to an hour and may be accompanied by **hypersalivation**.

Pain disappears after **intra-dermal anesthesia near the tragus**, serving as a diagnostic test.

Auriculotemporal Neuralgia (Frey's Syndrome)

Occurs after **parotid gland surgery, facial trauma, or mandibular fractures**.

Characterized by **burning pain, redness, sweating, and warmth** in the **parotid-masseteric area**, often triggered by eating.

Novocaine blockade of the auriculotemporal nerve relieves symptoms; **paraffin therapy and electrophoresis** are also effective.

Glossopharyngeal Neuralgia

Paroxysmal pain starts at the **tongue root or tonsil**, radiating to the **palate, pharynx, ear, eye, or neck**.

Attacks last **1–3 minutes** and may be triggered by **swallowing or talking**.

Pain is relieved by **local anesthetic application** (e.g., dicaine or pyromecaine) to the affected area.

Eagle's Syndrome

Caused by **elongation of the styloid process** of the temporal bone (>25 mm).

Symptoms include **pain when swallowing or moving the tongue**, radiating to the ear, dizziness, and nausea due to pressure on the **carotid artery**.

Surgical shortening of the styloid process provides **sustained relief**.

Tympanic Nerve Neuralgia (Reichert's Syndrome)

Characterized by **cutting pain** in the **external auditory canal**, radiating to the **face and mastoid area**.

Pain is provoked by **touching the ear canal**.

Superior Laryngeal Neuralgia

Pain originates in the **larynx**, triggered by **swallowing or eating**, and radiates to the **ear**.

A painful point can often be found **above the thyroid cartilage**.

Lingual Nerve Neuralgia

Sharp, shooting pain in the **anterior two-thirds of the tongue**, sometimes with **hyperesthesia or loss of taste and pain sensitivity**.

Submandibular Ganglionitis



Episodes of **acute pain in the submandibular area**, lasting from **minutes to an hour**, triggered by **spicy or heavy food**.

Pain radiates to the **jaw, lip, occiput, and neck**.

Salivation increases during attacks.

Conclusion

Trigeminal neuralgia, whether of central or peripheral origin, remains one of the most challenging neuropathic pain syndromes in clinical neurology and dentistry. The presented analysis demonstrates that despite the similarity of clinical manifestations — characterized by recurrent paroxysmal facial pain — the underlying pathophysiological mechanisms differ significantly between central and peripheral forms.

In central trigeminal neuralgia, vascular, infectious, and neurodegenerative factors dominate, while in peripheral forms, odontogenic and inflammatory conditions of the oral cavity, paranasal sinuses, and surrounding soft tissues play a crucial etiological role. The persistence of chronic infection foci within the maxillofacial region contributes to the chronic course and recurrence of neuralgic episodes.

A comprehensive diagnostic approach — including detailed anamnesis, neurological examination, and the use of diagnostic local nerve blocks — is essential for accurate differentiation between the two forms. Proper identification of the lesion site allows for targeted treatment, ensuring both pain relief and prevention of relapse.

Therapeutic management should include a combination of anticonvulsant and anti-inflammatory medications, vitamin B group supplementation, biogenic stimulants, and physiotherapeutic modalities. Local anesthetic blockade remains an effective diagnostic and therapeutic method, particularly for peripheral neuralgia. In contrast, surgical intervention for central trigeminal neuralgia demonstrates limited efficacy and should be reserved for strictly selected cases.

In conclusion, successful management of trigeminal neuralgia requires a **multidisciplinary approach**, integrating neurologic, dental, and physiotherapeutic care. Early diagnosis, pathogenetic treatment, and elimination of etiological factors significantly improve patient outcomes and quality of life. Continued clinical and neurophysiological research is essential for the development of more effective and safer therapeutic strategies for this complex pain disorder.

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