Botulinum Toxin(BOTOX®)
in
Oral Medicine

1. **Temporomandibular disorders** are the most prevalent orofacial pain conditions for which patients seek treatment.

- Masticatory musculature /Temporomandibular joint or both.

2. Trigeminal neuropathic pain conditions

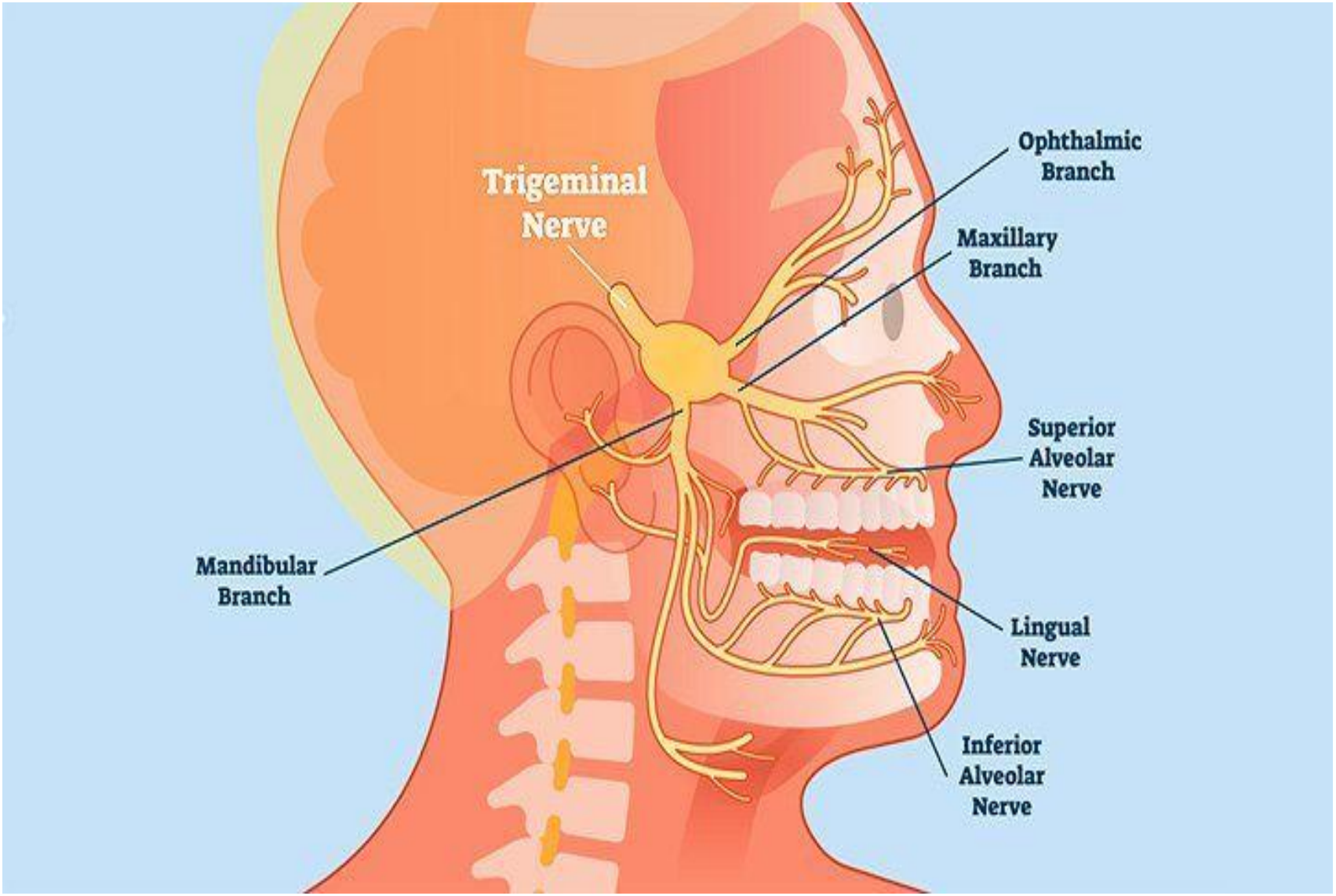
- Injury secondary to dental procedures
- Infection
- Neoplasias
- Disease / dysfunction of the peripheral and/or central nervous system

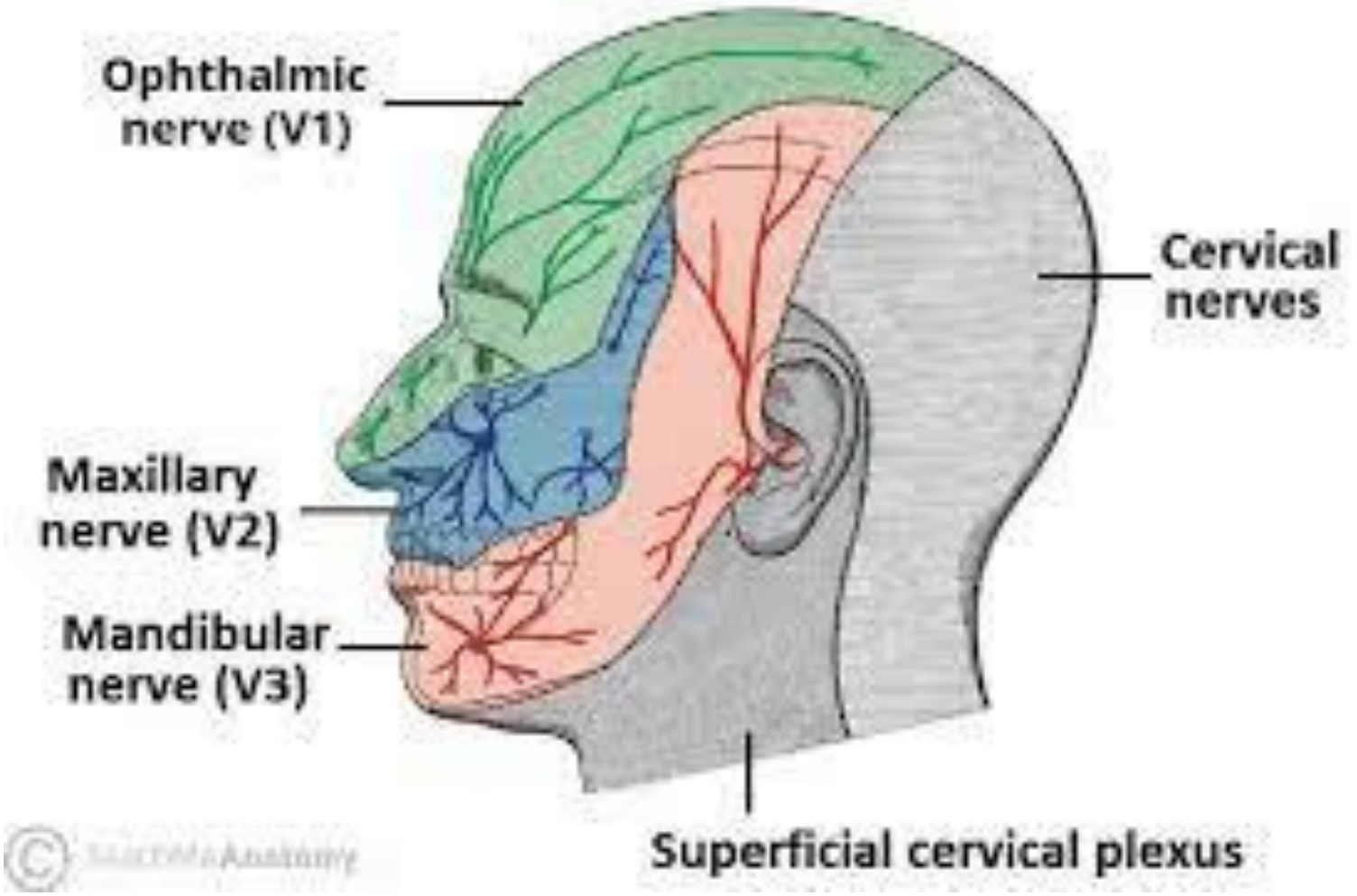
3. Neurovascular disorders

- Primary headaches, can present as chronic orofacial pain, such as in the case of facial migraine, where the pain is localized in the 2nd and 3rd division of the trigeminal nerve.

- **Together, these disorders of the trigeminal system impact the quality of life of the sufferer dramatically**

- **A multidisciplinary pain management** approach should be considered for the optimal treatment of orofacial pain disorders including both Non-pharmacological and Pharmacological modalities.


Trigeminal Nerve
Ophthalmic Branch
Maxillary Branch
Superior Alveolar Nerve
Mandibular Branch
Lingual Nerve
Inferior Alveolar Nerve


Ophthalmic nerve (V1)
Cervical nerves
Maxillary nerve (V2)
Mandibular nerve (V3)
Superficial cervical plexus

TMJ injections

Intracapsular injection of corticosteroids; triamcinolone or dexamethasone,
2% lidocaine without epinephrine,
Sodium hyaluronate

Oral lesions

Intralesional injection for oral lesions such as inflammatory, immunologic, and vascular

- Corticosteroids

1–5 injections of 12 mg per injection (range 2–36 mg).

- Platelet-rich plasma (PRP)

Most patients require up to three PRP injections, about 4 to 6 weeks apart, in order to achieve optimal results

An amount of 0.5 ml of each treatment was injected per 1 cm2 of ulcerated mucosa using a 25-gauge needle.

Botulinum Toxin(BOTOX®) in Oral Medicine

- Botulinum toxin (BT) was the **first** toxin to be used in the history of human medicine.
- It is a neurotoxin secreted by the anaerobic bacterium (Clostridium botulinum).
- Among the **Eight** known serotypes of this toxin, those currently used in medicine are types **A and B**
- I t is mainly used in the treatment of **TMJ disorders**
- Hypertrophy and hyperactivity of the masticatory muscles
- Therapeutic option to **relieve pain** in functional recovery from dental and oral and maxillofacial surgery.
- Treatment of facial pain and paralysis
- Treatment of salivary gland secretory disorders such as **sialorrhea and Frey syndrome** **which are common in patients with Parkinson's disease.**
- However, it is currently used **broadly for cosmetic purposes** such as reducing **facial wrinkles** and **asymmetry**.

Although, the therapeutic effect of BTA is temporary and relatively safe, it is essential to have knowledge about related anatomy, as well as the systemic and local adverse effects of medications that are applied to the face.

1. TMJ dislocation
2. Patients with TMD are usually administered BTA into the adjacent masticatory muscles such as the masseter and temporalis muscles. This strategy has successfully improved parafunction such as clenching as well as bruxism and TMD symptoms.

3. **Can relax** the adjacent masticatory muscles and, thereby, improve the muscle inflammation, leading to improved mouth opening.
4. Inducing **ptosis** by temporarily paralyzing the muscle by injecting BTA in the levator palpebrae superoris can prevent drying of the cornea when the eyes cannot be closed normally because of facial nerve palsy.

(Drooping of the eyelid is called ptosis. Ptosis may result from damage to the nerve that controls the muscles of the eyelid, problems with the muscle strength, such as in myasthenia gravis; or from swelling of the lid.

5. Inappropriate movements of the muscles adjacent to the **surgical site** immediately after surgery can **inhibit healing**.
Pre- or post-operative BTA injections can facilitate healing by **weakening** these muscles.

6. It has been reported that BTA injection into the masticatory muscles of patients with **jaw fractures**, which is the most common treatment in oral and maxillofacial surgery, **prevents bone displacement**.

7. In addition, a successful result was obtained by injecting BTA into the masseter and temporalis muscles, which weakened the masticatory strength when immediate or delayed loading was performed after dental implantation.

Mechanism of action

- BT is known to cause muscular paralysis by its action on the neuromuscular junction, where it blocks acetylcholine release in cholinergic neuron synapses

- Following the injection of BT, **its heavy chain** attaches to presynaptic cholinergic motor nerve terminals in the neuromuscular junction, and subsequently enters the neuron by endocytosis.

- **The light chain**, which is released from the endosome into the cell cytoplasm, cleaves synaptosome-associated protein of 25 k Da involved in the exocytosis of acetylcholine.

- Consequently, BT causes muscular relaxation by blocking acetylcholine release from presynaptic nerve fibers in the neuromuscular junction and inhibiting the depolarization of postsynaptic nerve terminals.

- BT does not inhibit the production of acetylcholine and, therefore, motor function is recovered by subsequent motor axon outgrowth step, which involves the sprouting of axons in the motor end plate after a certain time has

elapsed after toxin injection

The complications of BTA injection can be classified into three:

1**. Systemic**

overdose of BTA is injected and include nausea, fatigue, malaise, flu-like symptoms such as fever and chills, increased blood pressure, diarrhea, abdominal pain, and anaphylaxis due to allergic reactions

2.local,

which can vary based on the injection site, include headache, pain at the injection site, edema, ecchymosis, ptosis, dry eye syndrome, lagophthalmos, orofacial edema, dysphonia, and sensory abnormality **.**

Bruises or ecchymosis can develop in any area, and to prevent this, it is important to avoid superficial vessels by using the thinnest needle possible and bright light for adequate illumination during the procedure. Ptosis usually develops as the neurotoxin diffuses to adjacent areas when it is injected into the corrugator supercilii, and can be prevented by inhibiting toxin diffusion by pressing on the orbital rim with a finger

BTA is prohibited in patients with neuromuscular disorders such as peripheral motor neuropathies, Eaton- Lambert syndrome, multiple sclerosis, and myasthenia gravis

3. Reduced therapeutic effects due to **antibody formation**. Therefore, the dose used should be as low as possible in patients, and prolonging the period between injections can prevent antibody production.
When the result of treatment is not effective, antibody production should be suspected, and **when** there is no reaction even after the dose is increased twice, and antibody test should be considered.
If treatment fails because of antibody production, the product should be changed to another BT formulation.