

BDS Year 4 Regular batch Academic Year 2023-2024 Subject: Oral Medicine Topic: ORO-FACIAL PAIN PART-I

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Definition

(International Association for the Study of Pain:IASP)

An unpleasant sensory and emotional experience associated with actual or potential tissue damage or described in terms of such damage



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CLASSIFICATION

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AXIS I (PHYSICAL)

1.SOMATIC PAIN

- ✤ SUPERFICIAL
- DEEP
- **2. NEUROPATHIC PAIN**
- * EPISODIC
- CONTINUOUS

AXIS II(PSYCHOLOGICAL)

1. MOOD DISORDER

2. ANXIETY

3. SOMATOFORM DISORDERS

4. OTHER CONDITIONS SUCH AS PSYCHOLOGICAL FACTORS AFFECTING A MEDICAL CONDITION



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AXIS I- PHYSICAL- Somatic pain

- 1. Superficial somatic pain
 - a) Cutaneous pain
 - b) Mucogingival pain
- 2. Deep somatic pain
 - a) Musculoskeletal pain Muscle pain Protective co-contraction Delay onset muscle soreness Myofascial pain Myospasm Myositis

Myositis TMJ pain

Ligamentous pain Retrodiscal pain Capsular pain Arthritic pain Osseous and periosteal pain Soft connective tissue pain Periodontal dental pain



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AXIS I- PHYSICAL- Somatic pain

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b) Visceral pain Pulpal dental pain Vascular pain **Arteritis** Carotidynia Neurovascular pain Migraine with aural **Migraine without aura Cluster headache** Paroxysmal hemicrania Neurovascular variants Visceral mucosal pain Glandular ocular, auricular pain





AXISI- PHYSICAL - Neuropathic pain

- 1. Episodic neuropathic pain
 - a) Paroxysmal neuralgia
 - a) Trigeminal neuralgia
 - **b)** Glossopharyngeal neuralgia
 - c) Geniculate neuralgia
 - d) Superior laryngeal neuralgia
 - e) Nervous intermedius
 - b) Neurovascular pain
- **2.** Continuous neuropathic pains
 - a) Neuritis
 - a) **Peripheral neuritis**
 - **b)** Herpes zoster
 - c) Postheraptic neuralgia
 - b) Deafferentation pain
 - a) Neuroma
 - **b)** Atypical odontalgia
 - c) Sympathetically maintained pain





AXIS II- PSYCHOLOGICAL

1. Mood disorders

- a) **Depressive disorders**
- **b) Bipolar disorders**
- c) Mood disorders
- 2. Anxiety disorders
 - a) Generalized anxiety disorders
 - b) **Posttraumatic stress disorders**
 - c) Anxiety due to a medical condition



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PHYSICAL>>SOMATIC>>>SUPERFICIAL

1) CUTANEOUS PAIN

2) MUCOGINGIVAL PAIN

QUALITY:--

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- 1. Bright, stimulating character
- 2. Subjective excellent localization—anatomically
- 3. Site and location corelates
- 4. Provocation==intensity,incidence and location
- 5. Topical anaesthtic—reduces pain





PHYSICAL>>SOMATIC>>>DEEP

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QUALITY:--

- 1. Dull, depressing quality
- 2. Variable and diffuse localization
- 3. Site may not match source
- 4. Provocation—true in intensity and incidence but not localization
- 5. Presentsecondary central excitory effect is frequently associated





PHYSICAL>>NEUROPATHIC>>>EPISODIC

QUALITY:-

- 1. Periods of intense pain with remission LATER—SECONDS TO HOURS
- 2. Site location is correct but source is not located.
- 3. Paroxysmal or neurovascular
- 4. Paroxysmal:--intense, few seconds,, nerve related, between episodes patient is normal
- 5. Continuous---pulsatile, debilitating, intense to mild pain for 4-6 hours each episode,---similar to visceral





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PHYSICAL>>NEUROPATHIC>>>CONTINOUS

- Interference with transmission in afferent neurons
- Persistent, unremitting, burning sensation
- The area of pain sensation varies in size
- Nerve trauma is a common cause
- Burning mouth syndromes and atypical odontalgia
- Long standing effects can also result in redness, swelling





PSYCHOLOGICAL

- Site of pain are not related
- Multiple locations and may change site could be bilateral
- Therapy response are strange or surprising



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TYPES OF PAIN: Fast pain/Slow pain

- Immediately after an injury (i.e., stimulus for pain) a sharp, localised pain is felt and is carried by A-delta fibres at higher speed.—myelinated fibres
- After the fast pain, a diffuse, dull, intense and unpleasant pain sensation occurs and is carried by C fibres at slower speed. –non-myelinated



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TYPES OF PAIN: Epicritic pain/Protopathic pain

- Epicritic pain shows low threshold but accurate localisation.
- On the other hand, protopathic pain shows high threshold but poor localisation.



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TYPES OF PAIN: Deep pain/Superficial pain

- Pain arising from deeper structures like periosteum, muscle, tendon, etc. and is poorly localised.
- The superficial pain originates from superficial structures like skin and can be localised.





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TYPES OF PAIN:

Somatic pain/Visceral pain

- Somatic pain arises from somatic structures and may be superficial or deep.----Envelop
- Visceral pain originates from viscera due to inflammation, spasm, stretching. It is poorly localised, i.e., diffuse in character.
- Visceral pain may radiate or referred.





RATIONALE FOR PAIN MEASUREMENT

- ✤ A scientific way to characterize and quantify pain
- Needed for evaluation of pain treatments
 - Reduction in intensity
 - Effect on quality/characteristics
 - Eg burning, stabbing etc



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Visual Analogue Scales VAS

- *intensity* only
- simple, sensitive, reproducible
- also used for other variables eg nausea, satisfaction features
 - ✤ 100mm, horizontal line,
 - ✤ no markings,
 - * anchors at each end eg ' no pain', 'worst pain ever'
 - use same wording from staff at each measurement. No other cues to use.
 - More valuable when measured at rest and with movement



Visual Analogue Scales VAS

Limitations:

- Limited use in some patients
 - eg cognitively impaired, ICU, elderly, children
- Modified VAT (thermometer) were slider is moved for patient until level is reached
- Assumes pain is one-dimensional
- If used to assess pain relief then may be influenced by bias (past memory, expectation of effect)





VERBAL AND NUMERICAL SYSTEMS

- Numerical rating score
 - ✤ rate pain intensity from 0 to 10
 - easy to use but affected by assigning specific cues
 - Does not need any equipment
- Verbal rating scale
 - using words
 - * 'severe', 'moderate', 'mild', 'none'
 - quick & easy to apply
 - limited precision by restriction in number of points on scale





Magill Pain Questionnaire

- Previous systems only rated *intensity*
- Need assessment of other qualities of pain
- Magill Pain Questionnaire
 - ✤ Assesses 20 features of pain using 105 descriptors
 - sensory, affective, evaluative components
 - provides data that on qualitative differences in pain and pain relief methods



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Paroxysmal Neuralgia

Trigeminal Neuralgia

- TIC DOULOUREX or Trifacial Neuralgia or Fothergill's disease
- Etiology can be idiopathic (Ischemia of nerve, hypersensitivity reactions), due to neoplasms, multiple sclerosis etc

Accepted theory : Atherosclerotic blood vessel (usually the superior cerebellar artery) pressing on and grooving the root of the trigeminal nerve : results in focal demyelinization and hyper-excitability of nerve fibers



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Ignilias hypothen. 1. Compression of the nerve from different intracranial structures/abnormalities causing focal demyelination (Fig. 19.1).

Compression of the nerve due to an artery aneurysm or tumour

Might cause coupling of nerves fibres among each other (ephiphatic coupling)

The excitation of one nerve fibre will lead to hyperexcitation of all nerve fibres, which are coupled

So that individual area will be called as trigger zone

Such a sudden synchronous discharge could underlie the sudden jont of pain characteristic of TN pain attack (Ignition hypothesis)

> Secondarily transmission of impulse to other nerve fibre



Primary nerve impulse

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| Fig. 19.1 Mechanism of hyperexcitation after nerve compression I





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* CLINICAL FEATURES

- The pain characteristically has an electric shock-like quality and is unilateral except in a small Percentage of cases
- Right side commonly involved.
- Commonly seen in middle and old aged females
- Maxillary and mandibular branches commonly involved
- Sudden stabbing or lancinating pain, which persists for few seconds or minutes followed by pain free intervals



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TRIGGER ZONES : Hypersensitive areas, which can be stimulated by touch, mastication, movement or wind leading to painful episodes

- Ala of nose, vermillion border of lip, cheeks and around the eyes
- There is no sensory loss along the distribution of trigeminal nerve
- TIC DOULOUREX : If only spasmodic contractions present without painful episodes



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Diagnosis

- Based on the history
- Cranial nerve examination
- Local anesthetic blocks, which temporarily eliminate the trigger zone, may also be helpful in diagnosis.
- MRI of the brain : tumors, multiple sclerosis, and vascular malformations.





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Management

* Anticonvulsant drugs

- Carbamazepine drug -between 200-1200 mg
 - /day.

SIDE EFFECTS:

- Sodiu drowsiness, fatigue, dizziness, or nausea
- 100 m decreased bone marrow function (bone marrow depression)
 - blood disorder (acute intermittent porphyria).
 - Maxir Blood, Renal and hepatic monitoring
- Baclo





Evidence

Authors	Chole R ¹ , Patil R, Degwekar SS, Bhowate RR.
Title	Drug treatment of trigeminal neuralgia: a systematic review of the literature. J Oral Maxillofac Surg. 2007 Jan;65(1):40-5 Level 1a
Aim	review of the literature on drug treatment of trigeminal neuralgia. articles published between January 1960 to February 2005. Studies with high level of evidence were included. The levels of evidence of the articles were classified after the guidelines of the Oxford Centre for Evidence-Based Medicine.
Results	Of 770 publications, only 21 publications showed a high level of evidence (6 randomized controlled trials and 15 clinical trials/controlled clinical trials), with a total of 348 patients. A total of 749 publications were not included in the review as they showed a low level of evidence.
Interpretation	Anticonvulsants are effective in treating trigeminal neuralgia; however, few studies with high levels of evidence were found. It is quite difficult to compare or even combine their outcomes in a scientifically meaningful manner. Due to insufficient research data, there is a need for high-quality randomized controlled trials in this area of medicine.



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Oxcarbazepine (Trileptal)

- Sodium-channel modulator, calcium-channel blocker
- 300 mg bid. Max.total daily dose of 2400 mg when given as monotherapy
- Fewer side effects or drug interactions: Dizziness, diplopia, ataxia, nausea, somnolence, headache, hyponatremia (more often than with carbamazepine);
 Rash in 25% of patients who develop a rash from carbamazepine
- Monitor serum sodium, especially in patients receiving other drugs, such as diuretics, that can also cause hyponatremia





Gabapentin (Neurontin)

- Antiepileptic drug
- 300 mg tid. Maximum daily dose of 3600 mg
- Somnolence, dizziness, ataxia, fatigue,
 - nystagmus, tremor
- Good for adjunctive treatment





Phenytoin (Dilantin)

- Antiepileptic drug, sodium-channel modulator
- 100 mg po tid, or 300 mg once per day
- Comes in an intravenous form
- Many drug interactions ; Ataxia, slurred speech, rash
- Monitor blood levels





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- ✤ Baclofen (Lioresal)
 - Antispasmodic ; Structurally similar to gabapentin
- 5-10 mg tid. Increase by 10 mg every other day until 60-80 mg daily
- Few drug interactions
- Ataxia, lethargy
- Possibly synergistic with carbamazepine

- Lamotrigine (Lamictal)
- Antiepileptic drug, sodium-channel modulator
- A dose of 25 mg/d, increasing by 25 mg every third day, to a maximum of 400 mg/d, has been used for patients with trigeminal neuralgia
- Rash; may be severe; Many drug interactions; Dizziness, incoordination, vomiting



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Drug name	MAO (Mechanism of action)	Daily Doses	Mail senance dose/ day	Side effect
Carbamazepine	By blocking sodium channels, sodium will not be available for depolarization, thus nerve excitation will be diminished	100mg tid for first 3 days then 200 mg tid for next 3 days then 200/300 mg tid for 1 month based on response	600- 300 mg	 Aplastic anaemia SJS (Stevens Johnson Syndrome Lupus erythematosus Visual blurring Skin rashes Precaution Before treatment and during treatment hematological studies should be carried out once in 2 weeks
Oxcarbazepine	Same as carbamaz epine	300 mg bid	1200-1800 mg	Less than carbamazepine
Gabapentin	Enhances GABA release, GABA is inhibitory neuro-	300 mg tid 2/day	180u mg	Mild sedation, tiredness, dizziness and unsteadiness
Baclofen Junofen	Baclofen, a central spasmolytic, is a GABA-B agonist	5 mg tid	60- 10 mg	Decrease in muscle tone Transient sedation are expected
Lemotrigine	It acts at voltage sensitive sodium	25 mg bid 1-2/dawy	200-400 mg	Ataxia Constipation Vomiting and rash Steven Johnson syndrome

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Pharmacological Treatment



Treatment

- Surgical treatment: Patients with TN who are refractory to medical therapy are candidates for surgery.
 - procedures performed on the peripheral portion of the nerve, where it exits the jaw;
 - At the gasserian ganglion; and on the brainstem, at the posterior cranial fossa.





Microvascular decompression for TN caused by compression of the trigeminal nerve root

- Ablative procedures, including:
- Rhizotomy with radiofrequency thermocoagulation, mechanical balloon compression, or chemical (glycerol) injection
- Radiosurgery







- Peripheral neurectomy and nerve block:
- Peripheral neurectomy can be performed on the branches of the trigeminal nerve, which are the supraorbital, infraorbital, alveolar, and lingual nerves.
- Neurectomy is accomplished by incision, alcohol injection, radiofrequency lesioning, or cryotherapy.
- Cryotherapy involves freezing of the nerve using special probes, in theory to selectively destroy the pain fibers.





Differential diagnosis

/ Odontogenic pain

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 Inflamed pericoron: Tap along with partially erupted tooth for pericoronitis. Check for any swell, 3 within jaw for cyst.
 Pain of AFP is continuous, persist even on night time Usually AFP will not follow anatomic pathways Trigger point are absent in AFP
 Pain will be limited to the branch of glossopharyngeal nerve
 Pain persisting more than 3 months after the onset of the rash with history of herpes zoster.
 MPDS IS characteri d by presence of click, tender masticatory muscle, inability to open mouth.
 These are recurrent ttacks of unilateral pain, usually involve the orbital or periorbital region, accompany with lacrimation, conjunctival injection, nasal congestion or rhinorrhoea, ptosis or miosis and periorbital oedema.
 Migraine is throbburg moderate to severe, unilateral or bilateral pain which last for longer time. Does not have trigger zones
 It is a tumour of neopharynx. Causes pain in lower jaw, tongue, side of head with middle ear deafness. Exhibit asymmetry and defective mobility of soft palate and affected side.

Differentiating features

percussion.

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S. Lecturer Dr. Akansha Budakoti

Check for the featur for pulpitis like decayed tooth with or without tender on



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GLOSSOPHARYNGEAL NEURALGIA

- Paroxysmal pain that is similar to, though less intense than, the pain of TN.
- * The location of the trigger zone and pain sensation follows the distribution of the glossopharyngeal nerve, namely, the pharynx, posterior tongue, ear, and infraauricular retromandibular area.
- * Pain is triggered by stimulating the pharyngeal mucosa during chewing, talking, and swallowing.



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the two nerves.





- **Clinical Diagnosis:** The application of a topical anesthetic to the pharyngeal mucosa eliminates glossopharyngeal nerve pain and can aid in distinguishing it from the pain of other neuralgias.
- Etiology : intracranial or extracranial tumors and vascular abnormalities that compress CN IX.
- **Treatment:** is similar to that for TN, with a good response to carbamazepine and baclofen.
- Refractory cases are treated surgically by intracranial or extracranial section of CN IX,
- Microvascular decompression in the posterior cranial fossa, or by percutaneous radiofrequency thermocoagulation of the nerve at the jugular foramen



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NERVOUS INTERMEDIUS (GENICULATE) NEURALGIA (VII)

- Paroxysmal neuralgia characterized by pain in ear & (less frequently) the anterior tongue or soft palate.
- Not as sharp or intense as in TN & some degree of facial paralysis.
- Trigger zones: External auditory canal & a small area on the soft palate & post.auricular region.
- Geniculate neuralgia commonly results from herpes zoster of the geniculate ganglion and nervus intermedius of CN VII, a condition referred to as Ramsay Hunt syndrome.
- Viral vesicle on ear canal or on the tympanic membrane.





Management

- 2 to 3 weeks of high-dose steroid therapy
- Acyclovir
- Carbamazepine and antidepressants.
- sectioning of nervus intermedius.

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OCCIPITAL NEURALGIA

- Distribution of the sensory branches of the cervical plexus
- Unilateral in the neck and occipital region.
- Etiology Trauma, neoplasms, infections, and aneurysms involving the affected nerve(s).
- Trigger Factors-
- Pressure over the occipital nerves may amplify the pain, but no clear trigger.
- Trigger zones-Neck, temple & frontal regions.
- neurolysis
- ✓ Avulsion
- blocking the nerve with local anesthetic.





POSTHERPETIC NEURALGIA

- **Etiology and Pathogenesis**
- Herpes zoster caused by the reactivation of latent varicellazoster virus infection that results in both pain and vesicular lesions along the course of the affected nerve.
- The pain resolves **within a month** after the lesions heal.
- Pain that **persists longer than a month** is classified as
 - postherpetic neuralgia (PHN).



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PHN may occur at any age, but the major risk factor is increasing age

The pain and numbness of PHN results from a combination of both central and peripheral mechanisms. The varicella-zoster virus injures the peripheral nerve by demyelination, wallerian degeneration, and sclerosis, but changes in the CNS, including atrophy of dorsal horn cells in the spinal cord, have also been associated with PHN.





•This combination of peripheral and central injury results in the spontaneous discharge of neurons and an exaggerated painful response to no painful stimuli. **CLINICAL MANISFESATTION:** Patients with PHN experience persistent pain, paresthesia, hyperesthesia, and allodynia months to years after the zoster lesions have healed.

- •The pain is often accompanied by a sensory deficit, and
- there is a correlation between the degree of sensory



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deficit and the severity of pain.



Management

- **Topical therapy** –
- Lidocaine used either topically or injected or Analgesics capsaicin- C fiber neutrotoxin – depletes substance P gives short-term relief from severe pain.
- EMLA Cream (AstraZeneca) have also been helpful.
- Tricyclic antidepressants such as amitriptyline,nortriptyline, doxepin, and desiprimine
- Gabapentin
- > Carbamazepine or phenytoin.
- Nerve blocks or surgery at the level of the peripheral nerve or dorsal root have been effective
- Antiviral drug -famciclovir, along with a short course of systemic corticosteroids during the acute phase of the disease may decrease the incidence and Severity of PHN.



Evidence

Authors	Volmink J, Lancaster T et al
Title	Treatments for postherpetic neuralgia— a systematic review of randomized controlled trials RCT, 1996 Level 1a
Aim	To determine the efficacy of available therapies for relieving the pain of established postherpetic neuralgia.
Results	Based on evidence from randomized trials, tricyclic anti-depressants appear to be the only agents of proven benefit for established postherpetic neuralgia.
Interpertatio n	Along with steroids and capsaicin, even antidepressants can be effective in patients suffering from post herpetic neuralgia.



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1. In the trigeminal neuralgia :

- a) Trigger zones are present
- b) Unilateral Pain
- c) Right side is commonly involved
- d) All
- 2. Neuralgia which can be associated with Herpes zoster
- a) Post herpetic neuralgia
- b) Geniculate neuralgia
- c) Trigeminal neuralgia
- d) All of the above





3. Geniculate neuralgia is caused in the nervea) VII

- b) IX
- c) II
- d) X

4. Ramsay Hunt syndrome is

- a) Geniculate neuralgia
- b) Trigeminal Neuralgia
- c) Glossopharyngial Neuralgia
- d) None

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5. Initial Dose of Carbamazepine is

- a) 100 mg
- b) 200 mg
- c) 300 mg
- d) 400 mg



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THANK YOU

