**Oral and Maxillofacial surgery/Fifth year**

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**Orofacial pain**

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ain is a complex human psychophysiologic experience associated with actual or potential tissue damage. It is a multifaceted experience influenced by multiple factors such as past pain experiences, physical, cultural, cognitive, emotional and medical aspects.

The physiologic aspects of pain experience involve several processes: transduction, transmission, and modulation. The sum of these processes, when integrated with higher centers, yields the human experience of pain.

**Classification of orofacial pain**

Multiple classification systems for orofacial pain have been proposed.

At the most basic level, it is appropriate to classify orofacial pains as: somatic, neuropathic, and psychological.

**Somatic pain** arises from musculoskeletal or visceral structures interpreted through an intact pain transmission and modulation systems.

**Neuropathic pain** is defined as pain caused by a lesion or disease of the somatosensory nervous system. The etiology includes trauma, ischemia, infection, or metabolic disturbances. It arises from damage or alteration to the pain pathways; it can be classified into paroxysmal (episodic) and continuous neuropathic pain.

**Diagnostic evaluation**

The formal diagnostic evaluation contains the following components: chief complaint, history of present complaint, medical history, physical examination, diagnostic imaging, and psychosocial evaluation.

**Chief complaint**

It is the patient’s description of the pain. It may provide valuable information to reach diagnosis; pulpal pains are usually provoked by thermal stimulation, neuralgias are frequently described as sharp and lancinating, vascular headaches are throbbing, and muscle pain is described as a deep, dull ache. Many of these descriptions may overlap.

**History of present complaint**

It should include the following points:

* The intensity of the pain needs to be measured against the patient’s own experience of pain, need for medication, and effect on lifestyle.
* The patient should be asked to indicate the site of the pain or the site of maximum pain intensity and its anatomic distribution should be traced accurately in terms of local anatomy.
* The patient should be encouraged to remember the events surrounding the onset of the pain.
* The time relations of the pain should be clarified in terms of duration and frequency.
* Aggravating and relieving factors should be determined.
* The presence or absence of associated factors (redness or swelling of the face, flushing, tearing, nasal congestion, eyelid ptosis, facial numbness, or facial weakness) needs to be ascertained.

**Medical history**

A careful medical history should be taken. A thorough review of organ system disease should be performed, including surgical history, hospitalizations with special emphasis to history of trauma to the face, mouth or head, allergies, current medical treatments, and current medications, habit history, and psychosocial history.

**Physical examination**

Patients with orofacial pain should undergo a complete oral cavity, face, head, and neck examination which should include inspection, palpation, percussion, and auscultation. Muscles of mastication and muscles of neck and shoulder should be assessed and palpated for tender and trigger points, a more thorough evaluation of the masticatory muscles and TMJ includes evaluating mandibular function and measuring the maximum opening and lateral and protrusive excursions in addition to palpation of the lateral pole of the condyle for pain, swelling, clicking or crepitation. Intraoral examination includes dental occlusion and dentition for caries, gingival and periodontal health in addition to inspection and palpation for any swellings, masses, lesions, or areas of discoloration.

An important part of the examination is the neurological examination of all the cranial nerves especially trigeminal and facial nerves in addition to the upper cervical nerve roots (C2–C5), since afferents from the upper cervical spinal segments are relayed through the trigeminal brainstem complex forming trigeminocervical network which includes the 3 branches of the trigeminal nerve (the ophthalmic branch [V1], the maxillary branch [V2], and the mandibular branch [V3]) as well as the sensory nerves for the posterior head and neck (C2, C3, C4, C5). Activation of this network may result in referred pain perceived on one or both sides of the head, the eyes or sinuses, and the posterior head and neck.

Neurological examination may include; sensory testing with directional sense, sharp (pain) touch, light touch, hot and cold, pressure.

**Imaging**

Plain radiographs like panoramic (OPG) and periapical radiographs provide detailed evaluation for the jaws and teeth. More detailed imaging for the maxillofacial skeleton can be provided by computed tomography scan (CT scans), Magnetic resonance imaging (MRI) is best for soft tissue evaluation.

Ultrasonography can be used to evaluate the major salivary glands, carotid arteries, and masses in the neck.

Scintigraphy or bone scan with technetium-99m will highlight areas of increased metabolic activity within the bone and can help to identify infection, tumor, or degenerative changes in the TMJ.

**Other investigations**

These may include blood investigations, microbiological studies, diagnostic injections like local anesthetic, and biopsy of any suspicious mass or lesion.

**Odontogenic pain**

Dental pain is usually well localized, and the quality of the pain can range from a dull ache to severe, depending on the specific cause and extent of disease. NSAIDs and non-opiate analgesics can be used to alleviate most odontogenic pain but definitive treatment is dental.

**Pulpal**

Pain may be sharp, throbbing, or dull, it can be spontaneous or provoked or exacerbated by percussion, thermal or electrical stimuli. It is associated with compromised dental pulp due to deep caries, crown fracture or recent dental work. Treatment is by removal of carious lesion, tooth restoration, endodontic treatment, or tooth extraction.

**Periodontal**

Pain is localized, deep continuous associated with compromised periodontium (gingiva or periodontal ligament) exacerbated by biting, chewing or percussion. Usually there are signs of periodontal inflammation or abscess with or without tooth mobility, periapical radiographs may aid in diagnosis. Treatment options include: drainage and débridement of periodontal pocket, scaling and root planing, periodontal surgery, endodontic treatment, or tooth extraction.

**Cracked tooth**

It is associated with fractured tooth with history of trauma or restorative dental work, pain is usually sharp, spontaneous or brief provoked by biting, chewing or percussion. Fractured tooth can usually be detected by clinical examination or periapical radiograph. Treatment depends on the level of the tooth fracture; restoration or extraction of the tooth.

**Dentinal pain**

Brief, sharp pain provoked by different kinds of stimuli to the dentin (e.g., hot or cold drinks), it is caused by stimulation exposed dentin or cementum that may result from recession of periodontium or possible erosion of dentinal structure. Treatment options may include; mouthwash (fluoride), desensitizing toothpaste, tooth restoration, endodontic treatment. Patient should be educated about diet, tooth-brushing force and frequency, and proper toothpaste.

**Oral mucous membrane disorders**

Diseases of the oral mucosa are numerous and have a variety of local and systemic causes. Pain may be a symptom of the disease process, secondary to an associated process (infection), or related to damaged oral mucosa (chewing food, thermal, chemical stimuli). Typically, pain is associated with oral mucosal lesions including; ulcers, vesicles, bullae, erosions, erythema, or red and white patches. Treatment depends on the proper diagnosis; options may include topical or systemic analgesics and corticosteroids.

**Temporomandibular disorders (TMD)**

TMD encompasses a number of clinical problems that involve the masticatory musculature, the TMJ, and associated structures. They are considered to be a subclassification of musculoskel­etal disorders. TMD symptoms are more commonly seen in women than in men, and many symptoms seem to arise in adolescence or the early twenties and may continue intermittently, well into middle age.

**TMJ disorders**

Disorders of the TMJ include mainly;

* The internal derangements, such as disc displacements with and without reduction, these disorders are the result of disc–condyle inco­ordination that influences the TMJ biomechanics.
* TMJ subluxation and dislocation.
* Inflammatory disorders (e.g., capsulitis or synovitis).
* Osteoarthritis.
* Rheumatoid arthritis.

In TMJ disorders pain is localized to the preauricular area during jaw function with presence of painful click or crepitus during mouth opening, in addition to limited mouth opening (<35 mm) and deviated or painful jaw movements. There is pain on palpation of the TMJ with possible swelling in the acute phase. CT and MRI may be needed to reach the diagnosis.

Treatment is by:

* Patient education and self-care instructions.
* Medication: NSAIDs, non-opiate analgesics.
* Physical therapy through exercise program.
* Occlusal splints.
* Surgery; is only indicated when non-surgical therapy has been ineffective, and it is not indicated in patients who are asymptomatic or mildly symptomatic or as a preventive measure. Surgical interventions include; arthrocentesis, arthroscopic surgery, and open surgery.

**Arthrocentesis** is a conservative surgical intervention that involves an intra-articular lavage with or without deposition of hyaluronic acid or corticoster­oids; it is mainly indicated in cases of disc displacement without reduction.

**Arthroscopy** is a closed surgical procedure that allows direct observation joint tissue. It is performed mainly in the upper joint space and is utilized primarily for lysis and lavage but also for ablation of adhe­sions.

**Open surgery** also called arthotomy is a procedure that modifies joint anatomy, such as total or partial joint reconstruction or replacement, which is required for the patient who has advanced TMD that meets the surgical criteria and has been refractory to other modalities. It is used in cases of neopla­sia, bony or fibrous ankylosis, severe chronic arthritis, and severe chronic dislocations.

**Muscle disorders**

They can present as dull, aching pain exacerbated by jaw function or palpation. There is tenderness during palpation of masticatory muscles and tendons. Possible limited range of jaw movement and can be associated with a parafunctional habit (bruxism). Myofascial pain disorder tends to be seen in muscle pain conditions of a more chronic nature, and there are trigger or tender points in one or more groups of muscles. Pain can radiate to distant areas with stimulation. Treatment options include;

* Patient education and self-care.
* Medication: topical and systemic NSAIDs, non-opiate analgesics, muscle relaxants, antidepressants, (usually tricyclic antidepressants), anxiolytics, anticonvulsants, botulinum toxin, trigger point injections, vapocoolant spray.
* Physical therapy: transcutaneous electric nerve stimulation (TENS), massage, exercise program.
* Occlusal splints.
* Cognitive-behavioral: biofeedback, relaxation, coping skills.

**Neuropathic pain**

It has been esti­mated that the incidence of orofacial neuropathic pain is 5-10 per 100,000 people. It is divided into epi­sodic (paroxysmal) pain disorders, including trigeminal neuralgia and glossopharyngeal neuralgia, and continuous pain disorders that frequently result from deafferentation after injury in the peripheral and central nervous system, which is the case in neuromas and idiopathic trigeminal neuropathic pains such as atypical odontalgia.

**Trigeminal neuralgia**

It is a chronic paroxysmal neuropathic pain condition that is described as a severe, lancinating, and electric-like unilateral pain. It is localized most often to the second and third distri­butions of the trigeminal nerve (V2 and V3) intraorally and extraorally and can present in both distributions at the same time. The ophthalmic division is affected alone in only 4% of cases. There is usually a trigger zone in the trigeminal distribu­tion which, when stimulated, can result in an excruciatingly painful attack. Pain will occur following mild stimulation of this trigger area (e.g., washing the face, shaving, eating, brushing one’s teeth, or being exposed to a breeze). The pain attacks last seconds to minutes (about 2 minutes) followed by a refractory period in which stimulation of the trigger zone will not elicit another attack. Numerous pain episodes can be present daily. This pattern of pain must be met in order for the diagnosis of trigeminal neuralgia to be made otherwise alternative terms such as atypical trigeminal neuralgia can be applied. It may go through periods of remission where the pain can remit for months or even longer. This disorder is characterized by a protracted clinical course with increasing frequency and severity of pain. It has one of the highest suicide rates of any disease and is regarded as one of the most painful conditions known.

It typically affects individuals older than 50 years of age, although it can develop at any age, including young children. Women are affected more often than men by a ratio of 1.5: 1. The majority of cases occur sporadically; however, several reports of familial trigeminal neuralgia have been described.

**Etiology**

**Primary or idiopathic (classical) trigeminal neuralgia** has no definite cause but localized demyelination may be implicated, the superior cerebellar artery compression on the trigeminal root has been shown to be responsible for attacks of trigeminal neuralgia pain.

**Secondary or symptomatic trigeminal neuralgia** results from nonvascular compression by a cerebel­lopontine angle neoplasm, such as acoustic neuromas, meningiomas, cholesteatomas, and neurofibromas, has also been shown to result in trigeminal neuralgia. Therefore, MRI and CT scan of the brain should be requested in order to rule out any intracranial pathology.

Myelin loss due to multiple sclerosis has been shown to be a causative disorder related to the trigeminal neuralgia.

**Treatment**

Treatment of trigeminal neuralgia consists of medical and surgical therapies.

**Medical treatment**

* Anticonvulsants (e.g., carbamazepine which is the drug of first choice, gabapentin).
* Antidepressants (e.g., amitriptyline, nortriptyline).
* Non-opiate analgesics.
* Botulinum toxin injection.
* Combination of baclofen (muscle relaxant) and anticonvulsants when anticonvulsants alone are not effective, or if the therapeutic range cannot be achieved due to side effects.

**Surgical treatment**

If medical therapy is unsuccessful or not tolerated, surgical treatment should be considered which consists of numerous peripheral and intracranial procedures.

Peripheral procedures all have the goal of inducing nerve damage:

* Trigeminal nerve block which provide only temporary relief, high concentration lidocaine (10%) have been used.
* Alcohol injection may be effective for about 1 year but are painful, and fibrosis makes repeat injections technically difficult. The use of alcohol is associated with many complications include tissue toxicity, inflammation, and fibrosis.
* Peripheral neurectomy involves the avulsion or severing of the terminal branches of the trigeminal nerve with or without obturation of the foramen. Pain may recur after 2 years
* Cryotherapy of peripheral branches may provide pain relief for 6 months and may be repeated with good results.

Central procedures:

* Percutaneous trigeminal rhizotomy. These procedures are directed at the trigeminal ganglion aims to use controlled injury to interfere with the nerve's ability to transmit signals. They include radiofrequency thermal rhizotomy, glycerol injection, or balloon compression. The three modalities provide approximately equal initial pain relief (around 90%) but are each associated with different rates of recurrence and complications. Overall, radiofrequency rhizolysis consistently provides the highest rates of sustained pain relief but is associated with high frequencies of facial and corneal numbness.
* Posterior fossa exploration and microvascular decompression of the trigeminal root. It is based on the premise that trigeminal neuralgia caused by vascular compression of the nerve root, and surgically separating them may offer a permanent cure. Initial success rates for microvascular decompression are very high (approximately 90%), but long-term follow-up shows that after 10 years 30% to 40% of patients will experience a relapse
* Gamma Knife stereotactic radiosurgery; it is a minimally invasive technique that precisely delivers radiosurgical doses of 70 to 90 Gy to the trigeminal nerve root at the point of vascular compression as mapped using MRI, it provides good to excellent (60%–90%) initial pain relief.

**Glossopharyngeal neuralgia**

Glossopharyngeal neuralgia is a rare condition (0.2-1.3% of facial pain syndromes) associated with pain in the area supplied by the glossopharyngeal nerve (9th cranial nerve). Painful sites may include the nasopharynx, pos­terior part of the tongue, throat, tonsil, larynx, and ear. This disorder presents shooting paroxysms of pain that can occur multiple times a day with stimulation of the oropharyngeal region. Common triggers may include mechanical stimulation of the trigger zone as well as activi­ties including chewing, swallowing, coughing, talking, and head movement. It occurs in middle-aged or older individuals with no sex predilection.

Due to the proximity of the vagal sensory nerves, glossopharyn­geal neuralgia may coincide with a cardiac dysrhythmia such as bradycardia, asystole, and syncope in about 10% of the cases (**vagoglossopharyngeal neuralgia**). Like trigeminal neuralgia, it can be classified as classical or secondary but it is uncommon for glossopharyngeal neuralgia to be associated with multiple sclerosis.

**Treatment**

The first line of treatment is pharmacological; anticonvulsants medications (e.g., carbamazepine, oxcarbazepine, baclofen, phenytoin) may relieve the neuralgic pain for a long period, atropine can be used to prevent the related cardiac manifestations in vagoglossopharyngeal neuralgia.

When the medical treatment fails then surgical options include:

* Microvascular decompression.
* Surgical sectioning of the glossopharyngeal nerve and the upper two rootlets of the vagus nerve.
* Radiofrequency nerve ablation.
* Balloon compression.
* Stereotactic radiosurgery (Gamma Knife ablation).

If the pain is secondary to another condition then management of the underlying lesion must be addressed.

**Atypical odontalgia**

Pain that occurs when damage to the afferent pain transmission system has occurred (deafferentation). Usually, this condition is caused by trauma or surgery, including extraction and endodontic treatment. The patient usually complains of a spontaneous or provoked pain described as burning, nagging or boring pain. Pain that result from peripheral neural damage can be relieved by local anesthetic blocks but in some cases the peripheral neural damage leads to central changes in the trigeminal nucleus causing ongoing pain transmission to higher cortical centers despite minimal or even no peripheral input. Local anesthetic block does not arrest pain in this case.

Treatment is by anticonvulsants, antidepressants and non-opiate analgesics. Rhyzotomy and Gamma knife may be indicated in nonresponsive cases.

**postherpetic neuralgia**

It is a potential sequela of shingles, also known as herpes zoster which is the clinical manifestation of the reactivation of a lifelong latent infection with varicella zoster virus, usually contracted after an episode of chicken pox in early life where the virus lay dormant in the ganglia of peripheral nerves but in 10-15% of the cases the trigeminal nerve is involved in which the dermatome of the ophthalmic branch (V1) is affected in about 80% of the cases. Postherpetic neuralgia occurs immediately after the skin rash or after about 1, 3 or 6 months. Pain is spontaneous burning and tingling, but may present as dull and aching and occasional lancinating evoked pain. Clinically there may be small cutaneous vesicles or scarring, usually affecting the forehead, loss of normal skin color, corneal ulceration can occur. Sensory changes in affected area (e.g., hyperesthesia, dysesthesia).

Treatment is by acyclovir in the acute phase, anticonvulsants, antidepressants, non-opiate analgesics. Rhyzotomy and Gamma knife may be indicated in nonresponsive cases.

**Vascular pain**

**Giant cell arteritis (Temporal arteritis)**

It is an immune-mediated vasculitis that affects medium-sized and larger arteries, leading to vascular occlusion and ischemia, although it is considered as a systemic condition and can affect any vessel but the superficial temporal artery is the most commonly affected site. Patients are usually above 50 years of age with female predilection, presenting with symptoms of severe headache and scalp tenderness. A highly characteristic feature is jaw claudication, which is described as cramping pain of the masseter and temporalis muscles that increases with usage (chewing or talking) but is relieved by rest. The superficial temporal artery is sensitive to palpation and eventually appears erythematous, swollen, tortuous, or sometimes ulcerated. Rare examples of unilateral or bilateral tongue necrosis secondary to lingual artery involvement also have been described. The most significant complication in the head and neck region is vision loss, which usually is due to vasculitis of the posterior ciliary artery and ischemic optic neuropathy. If pain and stiffness affecting the shoulders, upper arms and pelvis are present in addition to the characteristic unilateral headache, a diagnosis of

**polymyalgia rheumatica** should be considered.

Investigations include elevated ESR, biopsy of the temporal artery is required which shows skip lesions of inflammatory tunica intima and media with giant cells, and usually there is narrowing of the lumen

**Treatment**

Treatment is by high-dose systemic corticosteroid therapy, the dose can be reduced as the ESR starts to fall. Methotrexate or azathioprine sometimes will be added for their steroid-sparing effects.

**Migraine**

It is vascular or neurovascular in origin. It is described as recurrent headache attacks lasting 4-72 hours, the pain is unilateral, of pulsating quality, moderate or severe intensity, aggravated by routine physical activity, certain foodstuffs (e.g. chocolate, bananas), alcohol, stress, hormonal changes during the menstrual cycle, the contraceptive pill or noise. It may be preceded by an aura which may include nausea, vomiting, visual disturbances (photophobia, flashing lights) and other disturbances of sensory and/or motor function. It occurs in females more than males with wide age range from childhood onwards.

**Treatment**

Medical treatment include; NSAIDs, Ergotamine derivatives, such as dihydroergotamine, Serotonin 5-HT1B/1D receptor agonists (triptans), such as sumatriptan. Other medications that have proven beneficial are beta adrenergic blockers such as propranolol and atenolol, calcium channel blockers such as verapamil and flunarizine, tricyclic antidepressants such as amitriptyline, serotonin antagonists such as methysergide, and antiepileptics such as topiramate and valproate.