

Orofacial Pain—Part I

Assessment and Management of Musculoskeletal and Neuropathic Causes

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■ Orofacial pain is a common complaint, affecting the lives of millions of people around the world. Chronic orofacial pain often constitutes a challenging diagnostic problem that can be complicated by psychosocial factors and typically requires multidisciplinary treatment approaches. The fundamental prerequisite for successful management of orofacial pain is an accurate diagnosis. Generating a differential diagnosis, which will ultimately lead to a definite diagnosis, requires thorough knowledge of the diagnostic range of orofacial pain. There is a vast array of orofacial pain categories including: (1) musculoskeletal, (2) neuropathic, (3) vascular, (4) neurovascular, (5) idiopathic, (6) pain caused by local, distant, or systemic pathology, and (7) psychogenic. This

article presents the salient clinical features and the therapeutic approaches for the various subtypes of musculoskeletal and neuropathic pain. Musculoskeletal pain is the most prevalent orofacial pain, with temporomandibular disorders and tension-type headache being the main examples. Neuropathic pain develops secondary to neural injury and/or irritation and can be distinguished into episodic, including trigeminal neuralgia and glossopharyngeal neuralgia, as well as continuous, such as herpetic and postherpetic neuralgia, traumatic neuralgia, and Eagle's syndrome. (KEYWORDS: Eagle's syndrome, myofascial pain, postherpetic neuralgia, temporomandibular disorders, trigeminal neuralgia)

Pain is one of the main symptoms for which patients seek medical attention. Pain in the orofacial region, in particular, can be very distressing for the patient, since it usually affects important daily living functions, such as chewing, swallowing, talking, and laughing. Temporomandibular disorders constitute the second most common cause of orofacial pain, following dental pain.¹ There is a wide variety of additional conditions that should

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be included in the differential diagnosis of the patient who presents with pain in the orofacial region (Box 1). Notably, referred pain is common in the orofacial structures, representing a challenging diagnostic problem.

A thorough history and examination is of paramount importance in arriving at an accurate diagnosis. The evaluation of the pa-

tient begins with a medical history, including history of trauma to the head and neck, significant illnesses, current medications, and complete review of systems, with special attention to systemic disorders that can cause facial pain.² A detailed description of the pain complaint in terms of pain duration, location, intensity, quality, frequency, and progression of pain since onset is an absolute prerequisite for an accurate diagnosis.³ Associated symptoms that may accompany the facial pain, as well as aggravating and alleviating factors, can provide valuable diagnostic information. A brief psychological screening should be part of the history for all chronic pain patients, since depression and anxiety are very common among this patient population.^{4,5} Psychological distress may be a contributing factor to the onset or maintenance of pain, a consequence of pain, or a coexistent problem with independent etiology.

The clinical examination of the orofacial pain patient includes assessment of cranial nerve function, cervical spine evaluation (posture and range of motion), palpation of masticatory and neck muscles, temporomandibular joint examination (tenderness to palpation, range of motion, joint sounds), and complete intra-oral and dental evaluation. Depending upon the findings of the history and clinical evaluation, appropriate laboratory tests and diagnostic imaging procedures may be required.

Orofacial pain can be classified into: (1) musculoskeletal, (2) neuropathic, (3) vascular, (4) neurovascular, (5) idiopathic, (6) pain caused by local, distant, or systemic pathology, and (7) psychogenic (Table 1). This article elaborates on the differential diagnosis and management of musculoskeletal and neuropathic orofacial pain. Temporomandibular disorders and tension-type headache constitute the main subcategories of musculoskeletal facial pain. Neuropathic orofacial pain is subdivided into episodic, including trigeminal and glossopharyngeal neuralgia, as well as continuous, such as herpetic and postherpetic neuralgia, traumatic neuralgia, and Eagle's syndrome. A comprehensive reference section has been included for those who wish to gain further information on a particular entity; many additional

BOX 1 ■ Classification of Orofacial Pain

Musculoskeletal

1. Temporomandibular disorders
 - a. Masticatory muscle disorders
 - Myofascial pain
 - Myositis
 - Myospasm
 - Local myalgia
 - b. Articular disc derangements
 - Disc displacement with reduction
 - Disc displacement without reduction
 - c. Temporomandibular joint disorders
 - Synovitis/capsulitis
 - Osteoarthritis
2. Tension-type headache

Neuropathic

1. Episodic
 - a. Trigeminal neuralgia
 - b. Glossopharyngeal neuralgia
2. Continuous
 - a. Herpetic neuralgia
 - b. Postherpetic neuralgia
 - c. Traumatic neuralgia
 - d. Eagle's syndrome

Vascular

1. Giant cell arteritis
2. Carotid artery dissection

Neurovascular

1. Migraine
2. Cluster headache
3. Chronic paroxysmal hemicrania

Idiopathic

1. Atypical facial pain
2. Atypical odontalgia
3. Burning mouth syndrome

Other diseases that can cause facial pain

1. Local pathology
2. Distant pathology (referred pain)
3. Systemic diseases

Psychogenic

1. Somatoform disorders
2. Factitious disorders
3. Malingering

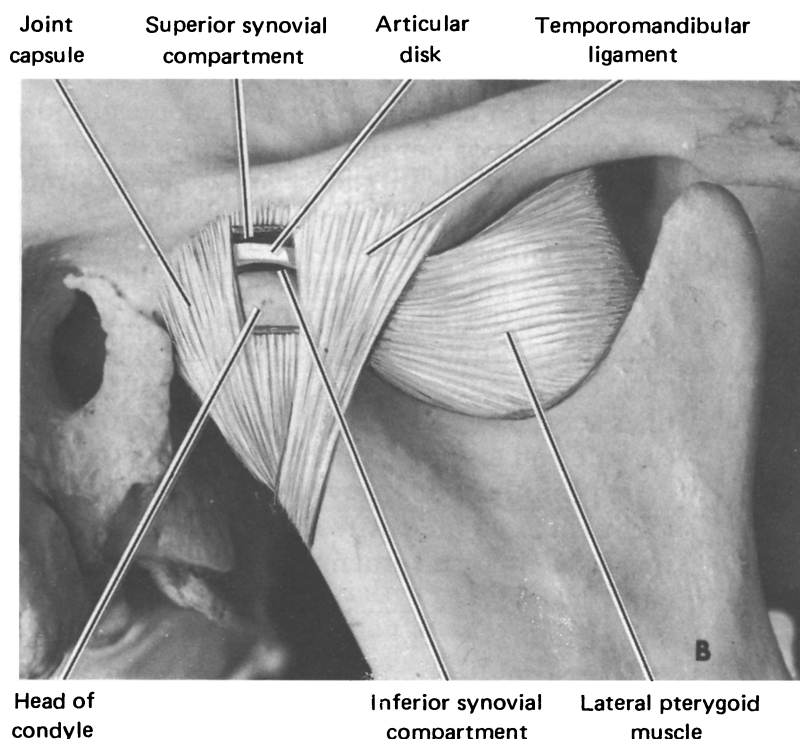


Figure 1. Temporomandibular joint anatomy. The capsule has been cut away to reveal the disc and its relationship to the articulating surfaces of the joint. Reprinted with permission from Hiatt JL, Gartner LP. *Textbook of Head and Neck Anatomy*, Ed. 1. Baltimore, Md: Williams & Wilkins; 1987.

figures and diagrams can be found in the various textbooks that are cited.

□ Musculoskeletal Pain

Temporomandibular Disorders

Temporomandibular disorders (TMD) is a collective term referring to a variety of pathologic conditions that affect the masticatory musculature, the temporomandibular joints (TMJ) or both (see Figure 1). The clinical presentation is variable and can include facial pain that is aggravated by jaw function, tenderness upon joint and muscle palpation, limited mandibular range of motion, deviation or deflection of the mandible on mouth opening, and TMJ sounds.^{6,7} Occasionally, patients may also complain of tinnitus, earaches, headaches, and dizziness. Signs and symptoms of TMD are very prevalent among the general population. It is estimated that 40% to 75% of nonpatient adults have at least

one sign, and 33% at least one symptom of TMD.⁶ Temporomandibular joint sounds are very common, presenting in approximately 30% of the general population, but they are not considered a specific indicator of pain-related pathology, and they do not warrant treatment when no other symptoms or signs are present.⁸

Temporomandibular disorders constitute the most common cause of chronic pain in the orofacial region. Approximately 12% of the general population is affected by TMD, and 5% of the population has symptoms severe enough to warrant treatment.^{1,7} The incidence of new onset TMD pain ranges between 1.6% to 3.9% per year.⁹ Temporomandibular disorders are more prevalent among women, with a female-to-male gender ratio of 2:1.⁹ Women-to-men ratio is estimated to be 8:1 among individuals seeking treatment for TMD.¹⁰ Greater willingness of women to report pain may play a role in the highly disproportionate representation of

the two sexes among clinical TMD cases.¹¹ In addition, pain severity and persistence are major predictors of use of healthcare, suggesting that women experience pain of greater average severity than men.¹² Gender is also a significant predictor of chronicity among patients who present with acute TMD.⁸ Garofalo et al¹³ reported that females presenting with acute TMD were significantly more likely to report pain at 6-month follow up compared to male patients.

Putative etiological and/or contributory factors include trauma involving local tissues, repetitive chronic microtrauma (eg, teeth grinding or clenching), unaccustomed jaw use (eg, opening the mouth wide for a long dental appointment), genetic vulnerability, increased level of emotional stress, poor sleep, and occlusal disharmony.^{7,14–18} Temporomandibular dysfunction has also been reported secondary to orotracheal intubation or endoscopy.^{19,20} Moreover, hyperexcitability of the central nervous system nociceptive pathways as well as dysfunction of the endogenous pain inhibitory systems have also been implicated in the development and/or maintenance of TMD-related chronic pain.^{21–23} Overall, the current knowledge suggests that TMD have a multifactorial origin, which might benefit from a multidisciplinary management.

Temporomandibular disorders are classified into 3 main categories: (1) masticatory muscle disorders, (2) articular disc derangements, and (3) temporomandibular joint disorders. The most common types of these TMD categories are described in the following text.

MASTICATORY MUSCLE DISORDERS: According to the American Academy of Orofacial Pain,⁶ masticatory muscle disorders can be divided into 4 subtypes: (1) myofascial pain, (2) myositis, (3) myospasm, and (4) local myalgia.

Myofascial Pain: Myofascial pain is characterized by the presence of focal, exquisitely tender muscle areas, called trigger points. Trigger points are typically found in taut muscle bands and produce a characteristic pain referral pattern on palpation.^{24,25} The patient complains of constant, dull muscle pain that is typically exacerbated by muscle use.⁶ Fre-

quently, the referred pain is the only symptom and the patient is unaware of the trigger points.²⁶ Trigger points in the masseter and temporalis muscles can refer pain to posterior teeth, while trigger points in the sternocleidomastoid and trapezius muscles often refer pain to the jaw or temple (Figure 2).^{3,25} Accordingly, the condition may present as

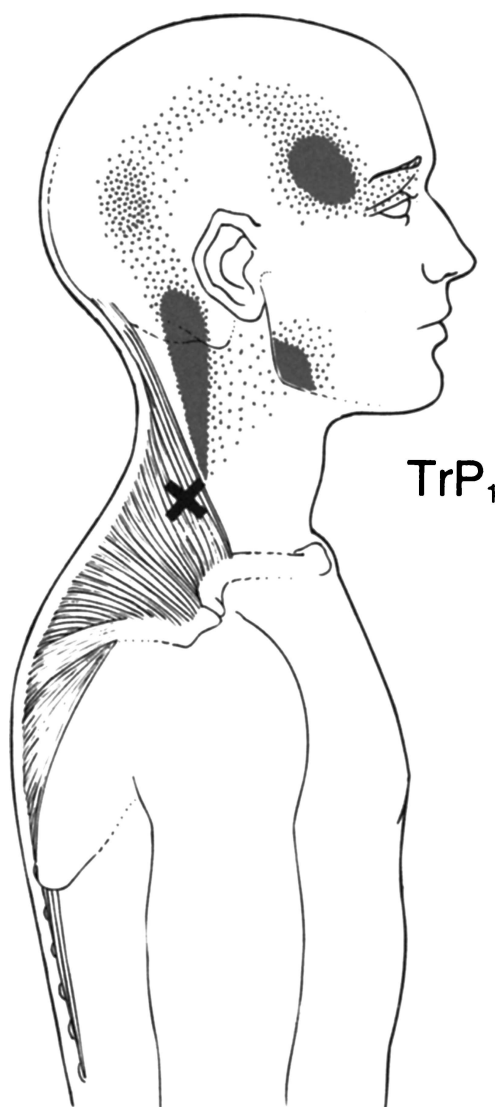


Figure 2. Pattern of pain referral and location (X) of a trigger point (TrP₁) in the upper trapezius muscle. Reprinted with permission from Simons DG, Travell JG, Simons LS. Travell and Simons' Myofascial Pain and Dysfunction: The Trigger Point Manual, Vol. 1—Upper Half of Body, Ed. 2. Baltimore, Md: Lippincott Williams & Wilkins; 1999.

toothache or headache and careful palpation of the head and neck muscles is required to reveal the trigger points. Palpation of the trigger points will elicit severe local pain, as well as aggravation of the referred pain. Replication of the patient's symptoms upon trigger point palpation will confirm the diagnosis.

Teeth clenching, trauma, somatization, and psychological distress have been identified as contributing factors for the development of chronic masticatory myofascial pain.^{14,16} In addition, Velly et al¹⁶ reported that the risk of myofascial pain was 3 times higher in women compared to men. Sleep disturbances, central nervous system dysfunction, nutritional deficiencies, or fatigue may also play a role in the etiopathogenesis of this condition.^{6,24} Treatment is aimed at elimination of precipitating factors and inactivation of trigger points by vapocoolant spray or injection of a local anesthetic, followed by stretching.²⁷ Relaxation therapy, daily stretching of the affected muscles, physical therapy modalities, and medications, such as analgesics, muscle relaxants, and tricyclic antidepressants in low doses can also be helpful.²⁶

Myositis: Myositis refers to true muscle inflammation and is an uncommon cause of orofacial pain that may result from a spreading infection or acute trauma to the muscle tissue.⁶ This condition is easy to diagnose, since it presents with the cardinal signs of inflammation; there is constant pain as well as swelling, erythema, and elevated temperature over the affected muscle.⁶ Pain upon palpation as well as limitation of mandibular range of motion secondary to pain are also apparent. Treatment of the underlying infection with antibiotics is required. In the case of muscle injury, rest should be advised in the early posttraumatic phase to limit hematoma formation.²⁸ Supporting therapy may consist of ice and nonsteroidal anti-inflammatory drugs (NSAIDs).

Myospasm: True myospasm is uncommon among TMD patients. Myospasm is an acute condition, characterized by severe muscle pain, marked limitation of mouth opening and, often, acute malocclusion.⁶ There is a significant increase in electromyographic activity, secondary to sustained, involuntary muscle contraction.⁶ Treatment is aimed at

stretching the muscle to full length; this can be achieved only after reducing the pain by ice, vapocoolant spray, or injection of local anesthetic.⁷ Injections of botulinum toxin are recommended for severe and recurrent myospasms.²⁹ In such cases, predisposing factors, including psychological distress and parafunctional activities, also should be addressed.

Local Myalgia: The term local myalgia was adopted by the American Academy of Orofacial Pain to denote muscle pain conditions that cannot be attributed to a specific cause, such as trigger points, inflammation, or muscle spasm.⁶ Local myalgia is a common temporomandibular disorder that is characterized by spontaneous muscle pain, muscle tenderness upon palpation, as well as limited mouth opening due to the pain.⁶ Typically, the symptoms are aggravated by activities requiring jaw movement, such as chewing, talking, laughing, or yawning. Treatment approaches include patient education on painless use of the mandible, moist heat applications, jaw exercises, and short-term use of NSAIDs or muscle relaxants.^{30,31} An intraoral stabilization appliance may be of benefit if jaw parafunctional activities are suspected, while relaxation therapy can be recommended for patients that report high stress levels.³² Training in diaphragmatic breathing, postural relaxation, improvement of sleep patterns, and physical activity have also been shown to be effective in reducing the symptoms.³³

ARTICULAR DISC DERANGEMENTS: Articular disc derangements are usually characterized by displacement of the articular disc anteriorly and medially. Alterations in the disc-condyle structural relation may result from elongation of the discal ligaments, secondary to trauma.⁷ High levels of anxiety and clenching and grinding of the teeth have also been implicated as risk factors.³⁴

Disc Displacement With Reduction: Disc displacement with reduction is characterized by improvement of the position of the displaced disc during opening. An opening joint clicking occurs as the condyle positions itself under the posterior band of the disc upon mouth opening, and a closing joint clicking can be heard as the condyle slips off the

disc, just before the teeth come together.⁷ The patient may complain of episodic and momentary catching of the jaw movement during mouth opening. Pain may or may not be present.⁶ Asymptomatic clicking is a very common sign among the general population and does not require treatment; however, patients should be counseled as to the nature of the clicking.³⁵

Disc Displacement Without Reduction: Disc displacement without reduction refers to an altered disc-condyle structural relation that is not improved during mouth opening. Frequently, there is a history of clicking and sudden onset of hypomobility. The displaced disc blocks the condylar movement, resulting in limited (25 to 30 mm) mouth opening and restricted lateral excursion to the contralateral side.⁷ The mandible deflects to the affected side on opening and clicking noises are absent.^{6,7} Pain is typically present in the acute condition, while chronic disc dislocation may be nonpainful. With the progression of the condition, there is a gradual increase in the mandibular range of motion.^{6,7} Milano et al³⁶ reported an association between nonreducing disc displacement and structural bone lesions, suggesting that disc displacement without reduction can predispose to the onset of osteoarthritis. When a patient with limited mouth opening requires tracheal intubation, fiberoptic intubation is indicated, since optimal visualization of the larynx cannot be achieved by direct laryngoscopy.³⁷

Magnetic resonance imaging can be used to substantiate the clinical diagnosis and rule out other pathological conditions of the TMJ that would result in blockage of condylar movement.^{38–40} In acute disc dislocation, there should be an effort to reduce the disc dislocation by manual manipulation, followed by insertion of an anterior repositioning appliance.⁷ Management of chronic disc dislocation may include a stabilization appliance, physical therapy, and NSAIDs if pain is present.⁷ Surgical arthrocentesis or arthroscopy may be beneficial for patients who are refractory to conservative treatment.^{41,42}

TEMPOROMANDIBULAR JOINT DISORDERS

Synovitis and Capsulitis: Synovitis and capsulitis are characterized by inflammation of the syn-

ovial lining of the TMJ and the capsular ligament respectively. They are grouped together since they cannot be distinguished on the basis of historical or clinical findings. Synovitis and capsulitis are characterized by constant deep pain in the TMJ, tenderness to TMJ palpation, and restricted mouth opening secondary to pain; acute malocclusion of the posterior teeth on the affected side may also be present.⁶ Nishimura et al⁴³ found a positive correlation between the concentrations of bradykinin in synovial fluid and the degree of arthroscopically-assessed synovitis.

Synovitis and capsulitis can be induced by trauma to the jaw or repetitive chronic microtrauma. In the case of acute trauma, ice should be applied to the affected joint 4 to 6 times daily for the first 24 to 36 hours, followed by moist heat applications for 10 to 15 minutes, 3 to 4 times per day. Restriction of jaw movement to a pain free range of motion and administration of NSAIDs on a regular basis for 10 to 14 days are recommended.⁷ A stabilization appliance can be of benefit, especially if parafunctional habits are present.

Osteoarthritis: Osteoarthritis is a non-inflammatory arthritic condition characterized by deterioration of the articular surfaces. It presents with pain that is exacerbated by mandibular movement, tenderness upon palpation of the joint, crepitus, and limited range of mandibular motion.^{6,7} The clinical diagnosis is substantiated by radiographic and magnetic resonance imaging evidence of structural bony changes, such as surface irregularities, flattening or erosion of the condyle, and osteophytes.^{40,44} Conservative approaches including NSAIDs, moist heat applications, painless use of mandible, jaw exercises and stabilization appliance constitute the mainstay of treatment.^{7,45} For refractory cases, one or two single injections of corticosteroids in the joint, arthrocentesis, or arthroscopic surgery may be recommended.^{46,47}

Tension-type Headache

Episodic tension-type headache (ETTH) is a common primary headache affecting the occipital, parietal, temporal, or frontal areas. According to the second edition of The International Classification of Headache Disorders,⁴⁸ infrequent ETTH refers to <12 headache days

per year, while frequent ETTH to ≥ 1 but < 15 headache days per month. The pain is typically bilateral, has a tightening or pressing quality, mild to moderate intensity, and may last from a few hours to 7 days.^{48,49} In contrast to migraine, there is no nausea, vomiting, or aggravation of the pain by routine physical activity. However, anorexia may accompany the pain, and occurrence of either photophobia (sensitivity to light) or phonophobia (sensitivity to sound) does not exclude the diagnosis. The pain may be precipitated by stress and is usually associated with fatigue and poor sleep. Treatment consists of stress management, relaxation training, and pharmacotherapy.⁴⁹ Simple analgesics and NSAIDs are typically effective in aborting the pain.⁵⁰

Chronic tension-type headache (CTTH) has characteristics similar to ETTH, but is characterized by presence of headache on ≥ 15 days per month for at least 3 months. Chronic tension-type headache usually evolves from ETTH and may be associated with no more than one of the following: photophobia, phonophobia, or mild nausea. Even though CTTH is often refractory to treatment, stress management, physical therapy, and prophylactic pharmacotherapy with tricyclic antidepressants may be beneficial.⁵⁰⁻⁵²

The pathophysiology of tension-type headache is elusive. The present review, for classification purposes, categorizes tension-type headache in the group of musculoskeletal disorders; however, it has been hypothesized that there is a continuum between tension-type headache and migraine.⁵³ Sensitization of the nociceptive central nervous system pathways secondary to nitric oxide production, has also been implicated; inhibition of nitric oxide was found effective in CTTH, providing hope for more specific and successful treatment in the future.^{50,52,54}

Chronic tension-type headache needs to be differentiated from medication overuse headache, which is the result of frequent use of anti-headache drugs (analgesics, ergot alkaloids, serotonin agonists, combined preparations with caffeine or codeine).⁵⁵ Withdrawal therapy, often under in-patient conditions, is essential for the effective management of medication overuse headache.⁵⁵

□ Neuropathic Pain

Episodic Neuropathic Pain

TRIGEMINAL NEURALGIA: Trigeminal neuralgia (TN) is an uncommon facial pain condition, characterized by episodic, lancinating pain in the distribution of one or more divisions of the trigeminal nerve.⁵⁶ The pain may be accompanied by a contraction of the facial musculature (hence the term *tic douloureux*). The disorder is more prevalent between the fifth and seventh decade; it has a female preponderance (female to male gender ratio of 1.74:1) and an annual occurrence rate of 4–5 per 100,000 population. Trigeminal neuralgia is typically unilateral (96% of cases) and affects most commonly the maxillary or mandibular division.

The pain is described as severe, electric-shock like, and lasts only for seconds; the patient is typically asymptomatic between the pain attacks. The pain paroxysms are often evoked by nonnoxious stimulation, such as light touch or vibration, to trigger areas extraorally and/or intraorally.⁵⁶ The trigger zones are localized on the affected side, but they do not always coincide with the area of pain. Common extraoral trigger zones occur above the supraorbital foramen, the inner canthus of the eye, lateral to the ala nasi, and over the mental foramen. Typically, immediately following a jab of pain, there is a refractory period during which further pain attacks cannot be evoked. Trigeminal neuralgia is characterized by spontaneous remissions that may last months or even years. Nevertheless, with time, pain attacks become more frequent, while remissions occur less often and last for a shorter time period.⁵⁷ In prolonged cases, patients may develop atypical features, such as persistent pain between episodes.⁵⁷

The intermittent temporal pattern and the severe intensity of TN resemble those of cluster headache or chronic paroxysmal hemicrania. However, the pain attacks of cluster headache and chronic paroxysmal hemicrania cannot be triggered by light touch, have a longer duration, and awaken the patient from sleep. Moreover, these headache disorders occur typically between the third and fourth decade of life and are accompanied by ipsilateral autonomic phenomena. Occasionally, the paroxysmal pain of TN affects the dentition and can be confused with dental

pain.⁵⁸ In these cases, the patient often undergoes multiple invasive dental procedures before the correct diagnosis is reached. Failure of dental treatment to provide long-term pain relief should raise the suspicion of TN. An important feature that distinguishes TN from dental pain is that TN typically does not interrupt the patient's sleep. Moreover, pain originating from dental pathology is usually progressive and its character changes with time.⁵⁹ Tooth vitality tests and radiographic examination will also serve to exclude dental pathology.

Trigeminal neuralgia may develop secondary to posterior fossa compressive lesions; intracranial tumors are detected in 2% of patients who present with typical TN.⁶⁰ Trigeminal neuralgia may also be induced by demyelinating plaques of multiple sclerosis involving the trigeminal nociceptive pathway.^{61,62} Thorough medical and neurological examinations as well as imaging are required to rule out such underlying conditions. Occasionally, neurological signs indicative of secondary causes of TN become apparent only later in the course of the disease, emphasizing the importance of repeating neurological examinations in TN patients.^{62,63}

Idiopathic TN is thought to be induced by vascular compression of the trigeminal root-entry zone, which results in demyelination of trigeminal sensory fibers.^{64,65} Histopathological examination of trigeminal nerve roots from patients with compression of the nerve root by a blood vessel reveals focal loss of myelin, close apposition of the demyelinated axons, and lack of intervening astrocytic processes.⁶⁴ Demyelination may result in ectopic generation of action potentials, presenting clinically as spontaneous pain, while ephaptic neural transmission may underlie the generation of pain by innocuous stimulation.⁶⁶ Over the course of time, excessive afferent input of nerve impulses may produce central sensitization, resulting in atypical TN features, such as constant pain.^{57,67}

Pharmacological therapy is the first line of treatment for TN; the goal is the reduction of neuronal hyperexcitability in the peripheral and/or the central nervous system.⁶⁸ Carbamazepine is the mainstay of pharmacotherapy of trigeminal neuralgia.⁶⁹

Additional potentially effective medications include oxcarbazepine, lamotrigine, phenytoin, gabapentin, and tizanidine.^{69–71} Often drug combinations are used to maximize effectiveness and minimize adverse effects.⁷² Surgical intervention is recommended if pharmacological treatment becomes ineffective or adverse effects become intolerable. Microvascular decompression of vessels compressing the nerve root is very effective and has low incidence of recurrence.^{73,74} However, it entails posterior fossa craniotomy and involves serious risks, such as hearing impairment, ataxia, brain stem infarction, cerebellar injury, and death.⁷⁴ Percutaneous ablative techniques involve lesioning at the level of the gasserian ganglion by percutaneous radio-frequency thermocoagulation, injection of glycerol, or balloon compression.^{75–77} These procedures have good initial results and carry less risk than microvascular decompression; however, they are associated with a higher incidence of pain recurrence. Potential complications include loss of touch sensation, dysesthesias, and anesthesia dolorosa. Gamma knife surgery involves lesioning of the trigeminal nerve at the root entry zone using stereotactic techniques and radiation between 70 and 90 Gy.^{78,79} This procedure is less invasive and thus suitable for medically compromised patients. Outcomes are similar to other ablative procedures.^{80–82} Facial sensory loss, paresthesias, and dysesthesias are the most common complications.^{77,78}

GLOSSOPHARYNGEAL NEURALGIA: Glossopharyngeal neuralgia is a rare pain condition that is similar to trigeminal neuralgia but it involves the distribution of the glossopharyngeal nerve. It has a prevalence of approximately 0.2% to 1.3% of that of TN and is more common in the sixth through eighth decades.⁸³ Glossopharyngeal neuralgia is characterized by severe, sudden, unilateral, stabbing pain in the ear, base of the tongue, tonsillar fossa, or beneath the angle of the mandible.⁸⁴ Pain typically lasts a few seconds to 2 minutes and can be triggered by swallowing, chewing, talking, coughing, or yawning.⁸³ Frequently, patients experience remission of pain lasting months to years. Pharmacological treatment is the same as for TN. In patients with medically intractable glossopharyngeal neuralgia,

surgical treatment may alleviate the symptoms. This involves either intracranial section of the 9th nerve and the upper two rootlets of the 10th nerve, or microvascular decompression. Microvascular decompression of the glossopharyngeal nerve has been shown to be a long-term effective procedure with minimal complications, mainly hoarseness and dysphagia.^{84,85}

Continuous Neuropathic Pain

HERPETIC AND POSTHERPETIC NEURALGIA: Following a chickenpox infection, the varicella-zoster virus becomes latent in the cranial nerve and dorsal root ganglia. Reactivation of the virus later in life can result in herpes zoster, which is characterized by vesicular eruption and associated severe pain in the distribution of the affected branch. Often, prodromal symptoms, such as pain, itching, and malaise precede the rash.⁸⁶ Herpes zoster disproportionately affects the immune-compromised as well as the elderly, possibly due to an age-related decline in varicella-zoster virus-specific, T-cell mediated immunity.^{86,87} The trigeminal ganglion is involved in approximately 10% of cases, with the ophthalmic division being most commonly affected. Antiviral drugs and systemic corticosteroids are the mainstays of treatment; opioids may also be required to control the severe pain that accompanies the herpetic rash.⁸⁶

Pain that persists longer than 3 to 4 months following the outbreak of herpes zoster eruption is referred to as postherpetic neuralgia. Postherpetic neuralgia affects 5% to 20% of herpes zoster patients, mainly elderly individuals.^{88,89} Additional risk factors for the development of persistent pain following a herpes zoster infection include female gender, prodromal pain, adverse psychosocial factors, as well as greater rash severity and higher acute pain intensity.^{86,88,90} Postherpetic neuralgia presents as continuous severe, burning pain with sharp exacerbations. Tactile allodynia (pain in response to an innocuous stimulus) and hyperalgesia (exaggerated pain in response to a noxious stimulus) are often present, compromising significantly the patient's quality of life. Since no apparent peripheral pathology is evident upon clinical examination, careful

questioning is needed to rule out a history of vesicular/ulcerative lesions, which would lead to a definite diagnosis.

Medications that have been proven effective in controlled clinical trials include the lidocaine patch 5%, gabapentin, tricyclic antidepressants, and opioids.^{91,92} Topical capsaicin may also be helpful, but it is poorly tolerated.⁸⁶ Frequently, a combination of various medications is necessary in order to obtain adequate pain control.⁹¹ Bowsher et al⁹³ reported that tricyclic antidepressants treatment is more efficacious when initiated early in the course of the disease. Postherpetic neuralgia is often refractory to treatment. Thus, emphasis is placed on its prevention. Administration of antiviral agents during the herpetic rash not only attenuates the severity of the acute infection, but also significantly reduces the risk of chronic pain.^{89,94} In addition, recent animal studies suggest that administration of gabapentin can alleviate the herpes zoster pain, as well as reduce the incidence of postherpetic neuralgia.⁹⁵ These findings, however, remain to be tested in human subjects.

TRAUMATIC NEURALGIA: Traumatic neuralgia occurs following direct neural injury and deaf-ferentation. Occasionally, there is a delay in the onset of pain after the initial injury.⁹⁶ The pain is typically described as constant and burning, while superimposed lancinating exacerbations may also occur.⁹⁶ Abnormal sensations, such as allodynia and hyperalgesia, and neural sensory or motor deficits often accompany the pain.³ Pharmacological management consists of tricyclic antidepressants; administration of anticonvulsant medications, such as carbamazepine or gabapentin, is also advocated if there are sharp, shooting qualities to the pain.⁹⁷ Topical applications of capsaicin may desensitize the affected area and alleviate the symptoms, while sparing the patient of the adverse effects of systemic drugs.^{3,98}

EAGLE'S SYNDROME: Eagle's syndrome is an uncommon condition resulting from compression of the glossopharyngeal nerve by an elongated styloid process or an ossified stylohyoid ligament. The condition is more prevalent among females.^{99,100} The chief signs and symptoms include dull and persistent neck

and throat pain, dysphagia, otalgia, and a foreign body sensation.^{99–101} Radiation of the pain to the TMJ or the upper limb has also been reported.¹⁰⁰ The pain may have a neuralgic component, mimicking glossopharyngeal neuralgia, and it is usually exacerbated by rotation of the head to the contralateral side, swallowing, extending the tongue, and yawning.¹⁰¹ It is suggested that abnormal bone formation, leading to an elongated styloid process or an ossified stylohyoid, may be secondary to soft tissue injury, such as tonsillectomy.¹⁰² However, most patients report no previous history of trauma or tonsillectomy.¹⁰⁰

Plain radiographs and computed tomography examination will reveal elongation of the styloid process or ossification of the stylohyoid ligament.^{101,103} Elongation of the styloid process occurs in approximately 4% to 28% of the general population; however, in most cases it is asymptomatic.^{102,103} In addition, patients with bilateral elongation frequently complain of unilateral neck pain. Therefore, establishment of the diagnosis is greatly facilitated by replication of the patient's symptoms on palpation of the tonsillar fossa, as well as alleviation of the pain by injection of local anesthetic.^{100,103} Surgical resection of the styloid process or the calcified stylohyoid ligament, through an intraoral or extraoral approach, results in resolution of the symptoms in most patients.^{99,100,103,104} Concurrent treatment with NSAIDs is advocated, in order to prevent re-ossification following the surgical procedure.¹⁰²

□ Summary

Musculoskeletal pain, including temporomandibular disorders and tension-type headache, is very common among orofacial pain patients. Temporomandibular disorders can be subdivided into various muscle, articular disc, and joint pathoses, which present with different clinical characteristics and require diverse treatment. Conservative, reversible approaches are recommended as the first line of treatment. Arthrocentesis and arthroscopy may be indicated for disc and joint conditions that are refractory to conservative therapy, but are not beneficial for muscle disorders.

Neuropathic pain occurs following neural injury or irritation and can be distinguished as episodic or continuous. Trigeminal and glossopharyngeal neuralgia constitute the main examples of episodic neuropathic pain. The unique clinical characteristics of these disorders, including their temporal pattern, distinct distribution, and nonnoxious triggering of the pain, greatly facilitate the diagnosis. Continuous neuropathic pain, such as postherpetic and traumatic neuralgia, is often described as burning, may be accompanied by allodynia and hyperalgesia, and can be resistant to various treatment approaches.

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