

Differential Diagnosis of Temporomandibular Disorders and Other Orofacial Pain Disorders

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- Temporomandibular disorders
- Temporomandibular joint disorders • Neuropathic pain
- Migraine • Tension type headache

The focus of this article is on the differential diagnosis of pain disorders that typically fall outside the realm of dental diseases. There are a great variety of such conditions—too many to review here. Therefore, this article discusses the differential diagnosis of a few of the more common orofacial pain conditions the dentist may face in a practice. These conditions are divided into two sections: temporomandibular disorders (TMD) and other common orofacial pain disorders. The first section reviews common TMD that are the responsibility of the dentist to identify and manage. The second section reviews some common orofacial pain disorders that the dentist needs to recognize but for which the dentist may not be the primary care provider.

TMD

TMD is a collective term that includes a number of clinical complaints involving the muscles of mastication, the temporomandibular joint (TMJ), or associated orofacial structures.¹ TMD are a major cause of nondental pain in the orofacial region and are considered a subclassification of musculoskeletal disorders. In many TMD patients the most common complaint originates from the muscles of mastication rather than from the TMJ. Therefore, the terms TMJ dysfunction or TMJ disorder are inappropriate for many complaints arising from the masticatory structures. It is

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for this reason that the American Dental Association adopted the term “temporomandibular disorder.”²

Signs and symptoms associated with TMD are a common source of chronic pain complaints in the head and orofacial structures. These complaints can be associated with some generalized musculoskeletal problems and even somatization, anxiety, and depression. The primary signs and symptoms associated with TMD originate from the masticatory structures and, therefore, are associated with jaw function. Patients often report pain in the preauricular areas, face, or temples. Reports of pain during mouth opening or chewing are common. Some individuals may even report difficulty speaking or singing. TMJ sounds are also frequent complaints and maybe described as clicking, popping, grating, or crepitus. In many instances, the joint sounds are not accompanied by pain or dysfunction, and are merely a nuisance to the patient. However, on occasion, joint sounds may be associated with locking of the jaw during opening or closing, or with pain. Patients may even report a sudden change in their bite coincident with the onset of the painful condition.

It is important to appreciate that pain associated with most TMD is increased with jaw function. Because this is a condition of the musculoskeletal structures, function of these structures generally increases the pain. When a patient’s pain complaint is not influenced by jaw function, other sources of (orofacial) pain should be suspected.

TMD can be subdivided into two broad categories related to their primary source of pain and dysfunction: masticatory muscle disorders and intracapsular (TMJ) disorders. A description of the most common disorders in each category is reviewed below. A more complete review of TMD can be found elsewhere.³

Masticatory Muscle Disorders

Definition

Functional disorders of masticatory muscles are probably the most common TMD complaint of patients seeking treatment in the dental office. With regard to pain, they are second only to odontalgia (ie, tooth or periodontal pain) in terms of frequency. They are generally grouped in a large category known as masticatory muscle disorders.

Clinical features

The two major symptoms of functional TMD problems are pain and dysfunction. Certainly the most common complaint of patients with masticatory muscle disorders is muscle pain, which may range from slight tenderness to extreme discomfort. Pain felt in muscle tissue is called myalgia. The pain is commonly described in terms such as dull, achy, or tender. The symptoms are often associated with a feeling of muscle fatigue and tightness. Patients will usually describe the location of the pain as broad or diffuse, and the pain is often bilateral. This complaint is quite different than the specific location reported in intracapsular disorders.

The severity of muscle pain is generally directly related to the amount of functional activity. Therefore, patients often report that the pain affects their ability to open their mouth, chew, and speak. If the patient does not report an increase in pain associated with jaw function, the disorder is not likely related to a masticatory muscle problem and other diagnoses should be considered.

The clinician should appreciate that not all muscle pain is the same.³ Some patients suffer with relatively simple overuse muscle pain called “local muscle soreness.” Clinically, the local muscle soreness manifests as tenderness or pain on palpation. Other patients may experience a more regional muscle condition such as myofascial pain. Clinically, myofascial pain is characterized by the presence of localized, firm, hyper-sensitive bands of muscle tissue called trigger points.⁴ These areas create a source

of deep pain input that can lead to central excitatory effects resulting in pain referral. This condition manifests as pain on palpation with referral of pain in the surrounding or remote tissues. Sometimes these remote tissues can be teeth, and the primary complaint that the patient may have is tooth pain. It is important that the dentist realizes that the site of pain is not always the same as the source of the pain. In this hypothetical case of myofascial pain, there is nothing wrong with the teeth (the site of the pain); however, it would be easy for the dentist to miss the correct diagnosis if the muscles of mastication (the source of the pain) were not included in the diagnostic work-up. It is also important to realize that myofascial pain originating from cervical muscles may refer pain into the orofacial region. Treating the site of pain in these instances would not result in improvement of the symptoms.

Dysfunction is a common clinical symptom associated with masticatory muscle disorders. Clinically, this may be seen as a decrease in the range of mandibular movement. When muscle tissues have been compromised by overuse, any contraction or stretching increases the pain.⁵ Therefore, to maintain comfort, the patient restricts movement within a range that does not increase pain. The restriction may be at any degree of opening depending on where discomfort is felt. The restriction may also be partly due to contraction of the antagonistic muscles, a phenomenon that is called protective cocontraction. In many myalgic disorders the patient is able to slowly open wider but this increases the pain.

Acute malocclusion is another condition that is often associated with masticatory muscle pain. Acute malocclusion refers to any sudden change in the occlusal position that has been created by a disorder. An acute malocclusion may result from a sudden change in the resting length of a muscle that controls jaw position. When this occurs the patient describes a change in the occlusal contacts of the teeth. The mandibular position and resultant alteration in occlusal relationships depend on the muscles involved. For example, the malocclusion associated with slight functional shortening of the inferior lateral pterygoid is clinically evident as disclusion of the posterior teeth on the ipsilateral side and premature contact of the anterior teeth (especially the canines) on the contralateral side. With functional shortening of the elevator muscles (clinically a less detectable acute malocclusion), the patient will generally complain of a sensation of heavier tooth contact on the ipsilateral side. It is important to realize that an acute malocclusion is the result of the muscle disorder and not the cause. Therefore, treatment should never be directed toward correcting the malocclusion by means of occlusal adjustments. Rather, it should be aimed at eliminating the muscle disorder. When the muscle disorder is resolved, the occlusal condition will return to normal.

Etiologic considerations

Myalgia can arise from a number of different causes. The most common cause is an increased level of muscle use. Although the exact origin of this type of muscle pain is debated, some investigators suggest it is related to vasoconstriction of the relevant nutrient arteries and the accumulation of metabolic waste products in the muscle tissues.⁵ Within the ischemic area of the muscle certain algogenic substances (eg, bradykinins, prostaglandins) are released, causing muscle pain.

Increased muscle use maybe the result of activities that are outside the normal functional activities of chewing, swallowing, and speaking. Such activities are considered parafunctional and these activities are more likely to compromise the muscle tissues and create pain. Activities such as daytime clenching of the teeth or sleep-related bruxing are common parafunctions that may lead to muscle pain.⁶ In addition to clenching and bruxing, habits such as chewing gum and biting lips, cheeks, and finger

nails are also considered to be parafunctional activities. It should be appreciated that these activities are very common and do not lead to pain in most individuals.

A misunderstood but very important concept for the clinician to appreciate is the fact that masticatory muscle pain is not strongly correlated with increased muscle activity such as in spasm.⁷⁻⁹ In fact, studies reveal that the resting activity of the masticatory muscles as measured by electromyography of patients with chronic muscle pain is no different than that of asymptomatic controls. Therefore the majority of masticatory muscle pain patients are not experiencing spasms.

It is now appreciated that muscle pain can be influenced and actually initiated by the central nervous system through antidromic effects leading to neurogenic inflammation in the peripheral structures. When these peripheral structures are muscles, this is clinically felt as muscle pain. This type of muscle pain is referred to as "centrally mediated myalgia" and can be a difficult problem to manage for the dentist since the peripheral structures, such as teeth, jaw, and muscles are not the significant cause of the pain. In these instances, the central mechanisms need to be addressed, which cannot be accomplished by classic dental therapies. Owing to the involvement of central mechanisms, increased levels of emotional stress and other sources of deep pain may likely influence masticatory muscle pain disorders.¹⁰

Management considerations

A detailed description of the management of each of the conditions is not the goal of this article and, therefore, other sources should be consulted. However, because some simple behavior modifications may reduce parafunctional use of the muscles, a few first-tier management options are described in this section. Making the patient aware that his or her maxillary and mandibular teeth should not be touching each other except to speak, chew, or swallow is an important first step. In addition, for most masticatory muscle conditions, soft diet, rest, moist heat, and (possibly) nonsteroidal anti-inflammatory drugs (NSAIDs) are usually helpful. Teaching the patient to keep the teeth apart and the mouth relaxed when the jaw is not used is very beneficial if not crucial in reducing pain.¹¹ If sleep-related bruxism is suspected, a stabilization appliance may be helpful.

TMJ

Definition

The signs associated with functional disorders of the TMJ are probably the most common findings when examining a patient for masticatory dysfunction. Many of these signs do not produce painful symptoms and, therefore, the patient may not seek treatment. When present, however, they generally fall into three broad categories: derangements of the condyle-disc complex, structural incompatibility of the articular surfaces, and inflammatory joint disorders. The first two categories have been collectively referred to as disc-interference disorders. The term disc-interference disorder was first introduced by Welden Bell¹² to describe a category of functional disorders that arises from problems with the condyle-disc complex. Some of these problems are due to a derangement or alteration of the attachment of the disc to the condyle; others to an incompatibility between the articular surfaces of the condyle, disc, and fossa; still others to the fact that relatively normal structures have been extended beyond their normal range of movement. With time, inflammatory disorders can arise from a localized response of the TMJ tissues to loading or trauma. These disorders are often the result of chronic or progressive disc derangement disorders.

Clinical features

The two major symptoms of functional TMJ problems are pain and dysfunction. Joint pain can arise from healthy joint structures that are mechanically abused during

function, from impingement of tissues, or from structures that have become inflamed. Pain originating from healthy structures or impingements is felt as sharp, sudden, and (sometimes) intense pain that is closely associated with joint movement. When the joint is rested, the pain resolves instantly. The patient often reports the pain as being localized to the preauricular area. If the joint structures have become inflamed, the pain is reported as constantly dull or throbbing, even at rest, yet accentuated by joint movement.

Dysfunction is common with functional disorders of the TMJ. Usually it presents as a disruption of the normal condyle-disc movement often with the production of joint sounds. The joint sounds may be a single event of short duration known as a click. If this is loud, it may be referred to as a pop. Crepitation is a multiple, rough, gravel-like sound described as grating or grinding. Dysfunction of the TMJ may also present as catching sensations when the mouth is opened. Sometimes the jaw can actually lock. Dysfunction of the TMJ is always directly related to jaw movement.

A single click during opening of the mouth is often associated with an anterior displaced disc that is returned to a more normal position during the opening movement. This condition is referred to as a “disc displacement with reduction.”³ Often when the patient closes the mouth a second click is felt which represents the re-displacement of the disc to the anterior displaced position (**Fig. 1**). This single opening click associated with disc displacement with reduction should be fairly repeatable. When the patient reports a single, loud, popping or cracking sound that cannot be easily repeated, the clinician should think about the possibility of an adherence.¹³ An adherence can occur as a result of prolonged static loading of the joint. In such a case, the lubrication is squeezed out of the contacting joint surfaces and this causes the surfaces stick together. On opening, this union can be disrupted and normal mouth opening resumes.

For some patients the disc displacement progresses and the disc may not be able to return to its normal relationship with the condyle during opening. This condition is referred to as a “disc displacement without reduction.”³ When this occurs, the patient often cannot open fully because the disc is blocking the translation of the condyle. For this reason the condition is often referred to as a “closed lock” (**Fig. 2**). Additional clinical characteristics include a deflection to the ipsilateral side on opening and protrusion, and restriction of movement to the contralateral side due to the limited ability of the condyle to translate.

Crepitation is usually related to roughness of the articular surfaces because of remodeling or osteoarthritis. Typically this is found in patients who have experienced a disc displacement without reduction or in whom radiographically notable bony changes are present. It can also be a sign of perforation of the disc or the retrodiscal tissues. If crepitation is the only symptom or sign a patient presents with, treatment is not usually indicated. Likewise, if the patient presents with a painless clicking TMJ that does not affect the quality of life, treatment is not indicated. However, the patient should be reassured and educated with regard to the origin of the sounds and, if indicated, instructed to avoid parafunctional activities.

Etiologic considerations

The cause of intracapsular disorders of the TMJ is most commonly related to trauma.^{14–16} This trauma may manifest itself as either macrotrauma or microtrauma. In cases of macrotrauma a single blow to the mandible can lead to a disruption of the normal biomechanical functions of TMJ. The traumatic event typically injures joint structures—elongating ligaments or damaging articular surfaces. Once ligaments have been elongated their biomechanical function is changed—often creating instability of the joint. This could eventually lead to disc displacement. Instability and

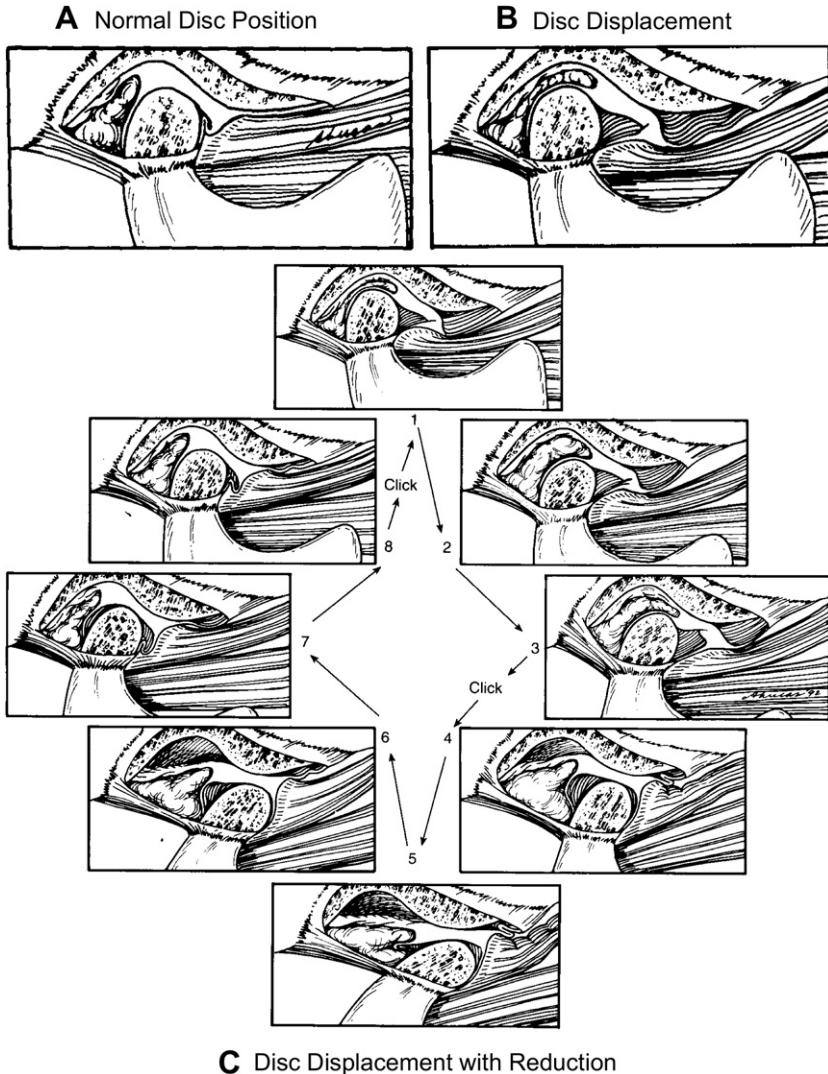
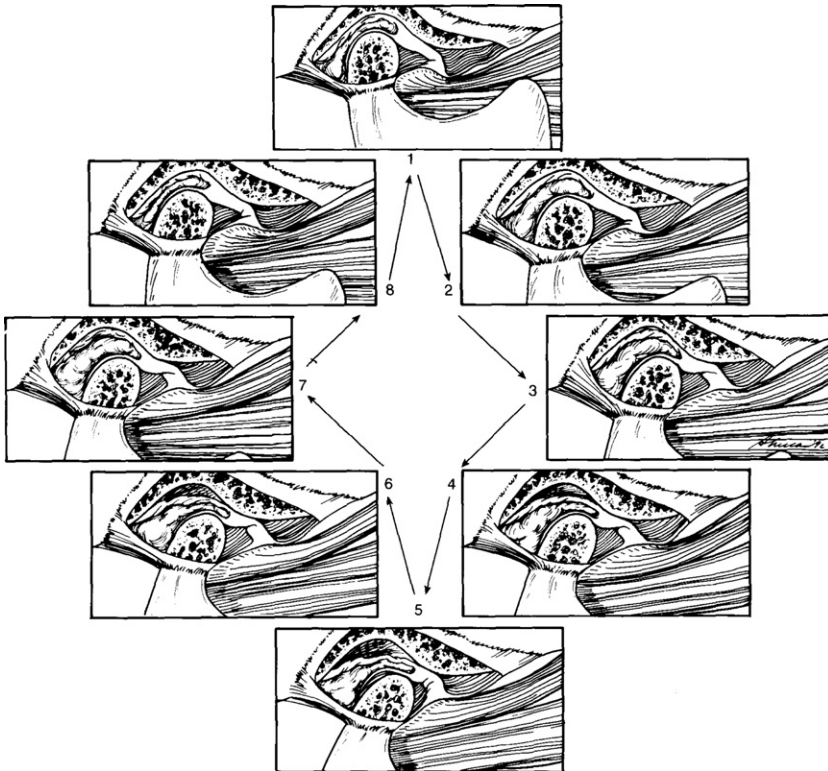


Fig. 1. (A) Normal condyle-disc relationship. (B) Disc displacement. (C) The movement of the condyle with a disc displacement. Note the clicking between 3 and 4, and again between 8 and 1. (Adapted from Okeson JP. Management of temporomandibular disorder and occlusion. 6th edition. St Louis (MO): CV Mosby; 2008. p. 181; with permission.)

disc displacement may both cause abnormal or unfavorable loading in the TMJ, and this may lead to osteoarthritic changes.¹⁷

Microtrauma, a small amount of loading force repeated over a long period of time, may lead to changes in joint structures. When the teeth are brought into heavy contact and the joint structures are loaded, there is a momentary reduction of blood flow in the small capillaries that supply the joint structures, resulting in hypoxia (a reduced supply of oxygen). Under circumstances of hypoxia, the metabolism of the local cell populations may alter. The byproducts of the altered metabolism may form free radicals when oxygen becomes available again, once the load on the tissues is reduced and the

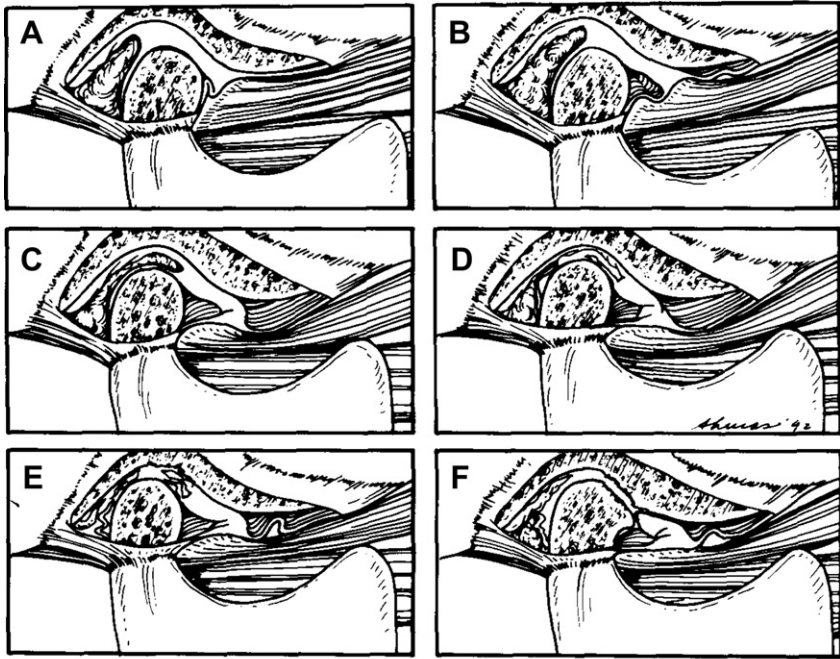


Disc Displacement without Reduction

Fig. 2. The movement of the condyle with a disc displacement without reduction. Note the disc is constantly maintained in the dislocated position (a closed lock). (*Adapted from Oke-son JP. Management of temporomandibular disorder and occlusion. 6th edition. St Louis (MO): CV Mosby; 2008. p. 185; with permission.*)

capillaries are reperfused. Free radicals may also be generated by direct mechanical trauma and tissue damage. Free radicals are very unstable molecules with a strong affinity for electrons. If these electrons are taken from adjacent healthy tissues, the integrity of these tissues can be compromised. This process is known as a “hypoxia-reperfusion injury.”^{13,18} The subtle changes that may occur could consist of a decrease in the lubrication quality of the synovial fluid creating more friction during joint movement. It may also affect the articular surfaces of the joint creating a softening of this tissue called “chondromalacia.” The compromised lubrication and softening of articular surfaces can cause the disc to displace from its normal position between the condyle and fossa.

Once the disc is displaced, joint loading can occur on nonarticular surfaces such as the retrodiscal tissue behind the disc. Because these tissues are highly vascularized and well innervated, compression often leads to pain. With further loading these tissues can breakdown allowing the condyle to directly load the articular fossa. Continued loading of these structures can result in loss of the articular surface of the condyle and fossa. The end result of this breakdown is osteoarthritis or degenerative joint disease (**Fig. 3**).



Possible Progressive Stages of Intracapsular Disorders

Fig. 3. The various states of internal derangement of the TMJ. (A) Normal joint. (B) Slight disc displacement. (C) Disc displacement. (D) Impingement of the retrodiscal tissues. (E) Retrodiscitis. (F) Osteoarthritis. (Adapted from Okeson JP. Management of temporomandibular disorder and occlusion. 6th edition. St Louis (MO): CV Mosby; 2008. p. 197; with permission.)

Management considerations

As previously mentioned, a detailed description of the management of each of the conditions is not the goal of this article and, therefore, other sources should be consulted.³ However, because some simple behavior modifications may reduce the loading of the joints, a few first-tier management options are described in this section. Because comparative studies have shown that conservative therapies provide similar results to the more aggressive ones, the general rule should always be to provide the most conservative therapy first.

The principle concept for managing most intracapsular disorders is to reduce loading of the joint structures so that remodeling and adaptation of the involved structures can take place.¹⁷ It is important to note that for adaptation to take place and for normal function to return, it is not necessary to restore the disc position. The patient needs to know that, if loading can be controlled, intracapsular disorders are often self limiting. Teaching the patient to reduce loading by simple approaches such as a softer diet with slower chewing can be very helpful. The patient should be instructed to avoid nonfunctional tooth contacts, such as clenching the teeth, chewing gum, and other oral parafunctional activity such as biting the fingernails or a pencil. If nighttime bruxism is suspected, a stabilization appliance may be considered for sleep-related bruxism. In cases of a painful disc displacement with reduction, an anterior positioning appliance may be useful, but care should be taken to use this appliance only during sleep, as longer use such as during the day and night may result in malocclusion. Clock-regulated NSAIDs may

also be helpful. Only after these types of therapies fail to control the patient's pain should more aggressive therapies be considered (eg, arthrocentesis or arthroscopy).

OTHER COMMON OROFACIAL PAIN DISORDERS

There are many conditions that manifest painful symptoms in the orofacial structures. In fact, there are many textbooks that have been devoted solely to these conditions.^{19,20} However, it is not the goal of this article to elaborate on all orofacial pain conditions. The reader merely needs to appreciate that the scope of pain in the orofacial structures is multifaceted and complex. This article highlights a few of the more common types of orofacial pain disorders that should be recognized by the clinician so that patients may receive the most appropriate care. Many of these conditions fall outside the normal realm of the dental practice and, therefore, should be referred to other more appropriate health care providers. This article discusses four common orofacial pain conditions. The first two conditions are neuropathic pain conditions. Neuropathic pain is a painful condition that has its origin within the neural tissue itself, either peripherally or centrally. With neuropathic pain there is nothing wrong with the somatic tissues; instead, the problem lies in how the nervous system is transmitting information to the sensory cortex. Neuropathic pain is divided into episodic neuropathic pain and continuous neuropathic pain disorders. The next two are migraine and tension-type headache. These conditions are considered primary headaches by the International Headache Society.²¹

Episodic Neuropathic Pains

Definition

As described earlier, pains that arise from abnormalities in the neural structures are called "neuropathic pains." Episodic neuropathic pains are characterized by a quick off-and-on pattern of pain.

Clinical features

Episodic neuropathic pains are characterized by periods of very brief, but intense, electrical shock-like pain followed by total remission. Usually, the individual is able to localize the site of pain quite well. The site, however, does not identify the correct source, because many are projected heterotopic pains. The term paroxysmal neuralgia has been used to describe this electrical shock-like pain. The most common is trigeminal neuralgia. Trigeminal neuralgia is characterized by a bright, stimulating, electric shock-like-quality pain that radiates into one of the three branches of the trigeminal nerve. The maxillary branch is affected most often, followed by the mandibular branch. The ophthalmic branch is affected the least often. The pain is extremely intense, usually lasting only a few seconds. On occasion it may last minutes, but this is rare. The pain is typically brought on by innocuous stimuli such as touching the face, shaving, or brushing the teeth. Between episodes the individual is usually pain free; however, if the episodes are frequent, there may be a lingering, dull aching pain.²² Other neuralgias are glossopharyngeal neuralgia, geniculate neuralgia, superior laryngeal neuralgia, and nervus intermedius neuralgia.²³

Etiologic considerations

The most common cause of trigeminal neuralgia is thought to be related to a demyelination of the nerve root as it exits the pons before reaching the foramen.²⁴ This demyelination may be secondary to pressure applied to the nerve by either a vessel in the brain²⁵ or an intracranial tumor.²⁶ Systemic demyelination disorders such as multiple sclerosis may also lead to symptoms indicative of trigeminal neuralgia.²⁷

However, in the majority of cases, none such factors can be identified, which renders the cause unknown.

Management considerations

Management of trigeminal neuralgia begins initially with medications that attempt to stabilize the nerve membranes. The most effective medications are typically anticonvulsive medications such as carbamazepine and oxcarbazepine, even though these medications may have serious side effects.²⁸⁻³⁰ Medications with less evidence of effectiveness but with a better side-effect profile include gabapentin³¹ or pregabalin.³² If medications do not adequately resolve the pain, there are several surgical options varying from peripheral procedures such as rhizotomy to central procedures, including microvascular decompression surgeries and gamma knife procedures that may be considered.¹

Continuous Neuropathic Pains

Definition

Some neuropathic pains may have fluctuating intensities but never go away. These disorders are called continuous neuropathic pains and are currently some of the most difficult orofacial pain conditions to successfully manage.

Clinical features

Continuous neuropathic pains are characterized by a dull, yet burning, pain. The pain is ongoing and unremitting, yet the intensity can show patterns of fluctuation. The pain is often accompanied by other neurologic signs (ie, anesthesia, paresthesia, hypoaesthesia, hyperaesthesia). Although the pain is present in a particular location, there is no evidence of any tissue changes or disease. When this pain is felt in the region of the teeth it can be a difficult challenge for the dentist. The pain may have the same clinical characteristics of a true toothache, which makes the correct diagnosis challenging for the clinician. However, helpful hints to consider are the duration of the pain and the fact that stimulation of the site of neuropathic pain (ie, with hot or cold) does not typically influence the intensity of the pain.³³

Etiologic considerations

The cause of continuous neuropathic pain is not well understood. Certainly, trauma to a peripheral nerve can result in deafferentation (ie, an ongoing pain condition).³⁴ However, some continuous neuropathic pains seem to spontaneously appear without any obvious cause. It is believed that the central nervous system can change (neuroplasticity) resulting in the processing of information that is not appropriate for the peripheral stimulus (central sensitization).

Management considerations

Management of continuous neuropathic pains is very difficult. Research is only beginning to help us understand the central mechanisms that seem to contribute to this painful condition. Medical management is the first line of treatment; however, the medications that are presently available do not consistently help all patients. At best, we can reduce pain and improve quality of life, but total elimination of this type of pain is very difficult. Sometimes the tricyclic antidepressants³⁵ such as amitriptyline or desipramine can be helpful. The anticonvulsive medications such as gabapentin or pregabalin may also be helpful.^{31,32,36} Often a trial of some of these medications and others may be needed to determine the most effective treatment. It is important to remember that neuropathic pains typically do not respond to NSAIDs or opioids. A complete list of management options for continuous neuropathic pain can be found elsewhere.

Migraine

Definition

Migraine is a common, intense and debilitating headache. It is what the general public refers to whenever someone has a “really bad headache.” It is a primary headache with central etiologies. The International Headache Society has two major designations for migraine: migraine with aura and migraine without aura.³⁷

Clinical features

Migraine is characterized by throbbing, moderate-to-severe, often debilitating pain. Sixty percent of the time the headache is unilateral and often reported in the temple or behind the eye. Migraine can be felt in the maxillary arch, thus referred to as “mid-face migraine.” This can be a diagnostic problem for the dental clinician because the pain can be felt in or around the teeth. The patient will often report nausea, photophobia, phonophobia, and osmophobia, and will seek a dark, quiet room. The pain is aggravated by routine physical activity and sometimes even simple head movements. The pain episodes may occur at any time of the day or night but most frequently occur on arising in the morning. The pain episode commonly lasts 4 to 72 hours in adults and 2 to 4 hours in children.³⁸ Scalp tenderness occurs in two-thirds of the patients during or after the headache.

Some migraine patients report a complex of focal neurologic symptoms that immediately precedes the headache.³⁹ This is called the “aura” and usually develops in 5 to 20 minutes and lasts less than 1 hour. When present, the aura is commonly characterized by visual, sensory, or motor phenomena, and may even include language and brainstem disturbances. The visual symptoms are the most common phenomena associated with aura. Visual symptoms can be characterized by sensations of flashes of light before the eyes (photopsia), the partial loss of sight (scotoma), or a zigzag, flashing colored phenomenon that migrates across the visual field (teichopsia). Sensory symptoms such as paresthesia⁴⁰ can occur. Motor effects may present as focal fatigue or difficulty with speech.

Etiologic considerations

Migraine affects approximately 12% of the population, with about 18% females and about 6% males being affected.⁴¹ Migraine most often appears in the first 3 decades of life.

Studies suggest that migraine patients have a genetic susceptibility to this pain condition, with 50% to 60% of migraine patients having parents that also experience migraines.⁴² Migraine is considered a neurovascular phenomenon because both neuralgic and vascular structures are involved in the pathophysiology. This system of neural innervation of the intracranial vessels is called the trigeminovascular system. Present evidence suggests that there is a neurologic trigger in the brainstem that initiates a cascade of events that result in neurogenic inflammation of the cranial vessels producing the headache.⁴³

Management considerations

The management of migraine with or without aura involves patient education and pharmacologic approaches. Patients who experience migraine headaches need to understand basic information about their pain condition. They need to know that even though the pain is very severe, it is still benign. An important aspect of education is having the patient identify any triggering factors that initiate the migraine attack. Triggers may be initiated by exposures to certain foods, alcohol, odors, stress, or even changes in eating or sleeping patterns. The patient should be asked to maintain a pain diary which helps identify factors that are associated with the initiation of the

headache. Once these factors are identified, efforts are made to avoid them so as to reduce the number of migraine attacks.

Pharmacologic management of migraine can be divided into two types: medications that are used to abort a migraine at its start and medications that are used to prevent migraine attacks. The choice of which management strategy to use is determined by the frequency of the migraine attacks. As a general rule, migraine attacks that are infrequent are managed with abortive medications so that treatment is immediately initiated during the onset of the attack. When migraine attacks occur so often that they significantly interfere with the patient's daily activities, preventive medications should be considered.⁴⁴ If abortive medications are used 2 days per week or more, the patient may develop rebound headache or medication-overuse headache.

A class of medications that has been proven helpful in aborting a migraine is the triptans, of which there are many.⁴⁴ These drugs seem to stop the neurogenic inflammation in the meningeal (dural) vasculature⁴⁵ and they may also act within the brain. Frequent migraines are best managed by prescribing daily medication so as to prevent them from occurring. Beta-adrenergic agents (beta blockers) such as propranolol or metoprolol⁴⁶ or calcium channel blockers such as nifedipine or verapamil⁴⁷ have proven effective. In addition, the tricyclic antidepressants have shown to be useful—especially amitriptyline.^{44,48} Finally, anticonvulsants such as topiramate and divalproex sodium also have proven to be efficacious in the prevention of migraines.⁴⁹

Dentists do not normally prescribe such medications unless they have advanced training in orofacial pain, oral medicine, or oral surgery because many of the drugs have significant side effects, especially on the cardiovascular system (eg, propranolol, sumatriptan). Although most are quite safe in a healthy patient, the medically compromised patient or chronic pain patient who uses other medications may experience significant problems that will need proper attention by appropriate health care professionals.

Tension-type Headache

Definition

Tension-type headache is a primary headache felt as a bilateral dull, aching pain usually felt in the shape of a tight band around the head. It is estimated that as many as 74% of the general population experience this type of headache at least once a year.⁵⁰

Clinical features

Tension-type headache is the most common headache reported in the general population. The headache is described as a dull, nonpulsatile tightness, or pressure felt in the occipital, parietal, temporal, and frontal regions. In 90% of the cases, the pain is felt bilaterally.⁵⁰ Some will describe the feeling of a tight “headband” compressing their head as if they were wearing a tight cap. Most tension-type headaches are of mild or moderate intensity, rarely becoming debilitating as with migraine. Most tension-type headaches are episodic, lasting an average of 12 hours, although the duration can vary greatly (30 minutes to 72 hours).⁵¹ Accompanying symptoms may consist of either photophobia or phonophobia but not both. Nausea is not associated with tension-type headache.⁵²

Etiologic considerations

Although tension-type headache is the most common headache experienced by humans, its pathophysiology remains unclear. Part of the problem may be that tension-type headache likely has a central etiologic mechanism, especially involving

the limbic structures. Emotional stress, anxiety, and depression seem to present causal relationships with tension-type headaches.^{53,54} However, there are many other disorders that result in headache that present with the same clinical characteristics of tension-type headache. For example, trigger points associated with myofascial pain (discussed previously) result in a headache at the referred site that is often clinically described by the patient as a tension-type headache. This type of headache is secondary to the myofascial condition and, therefore, should not be classified as a tension-type headache. Similarly, patients with sleep bruxism may awake with headache as a secondary symptom. Also morning headache in the temporal area is frequent associated with sleep respiratory disorders related to snoring or sleep apnea.^{55,56} The headache should always be classified to the primary disorder, which will assist in selecting the proper treatment.

Management considerations

Like many pain disorders, management of tension-type headache begins with patient education. The sufferer needs to identify those factors that aggravate the condition as well as those that help relieve it. It is often helpful to have the patient maintain a headache diary so that factors that are not commonly considered be recognized. The patient should be encouraged to decrease intake of caffeine (coffee, tea, soft drinks) and alcohol, as well as any medications that have been chronically used for the headache (rebound headache). The patient should be informed that eliminating these substances may at first increase the frequency and intensity of the headaches. After 1 to 2 weeks, the withdrawal effects should subside.

Since emotional stress often plays an important role in tension-type headache, the patient should be assessed for any significant stressors and, if identified, corrective behaviors or avoidance should be encouraged. Stress management skills can be important therapies with tension-type headache. Relaxation training and biofeedback techniques^{53,54} can also be very helpful. All these are frequently performed by a psychologist trained in cognitive-behavioral therapy. If a major depression disorder or anxiety disorder is present, these conditions need to be managed by the proper health care provider.

As with migraines, depending on the frequency of the headache, tension-type headaches are treated either with abortive or preventive medications. However, there are to date no evidence-based guidelines indicating which medications are most effective. To abort infrequent tension-type headaches, judicious use of mild analgesics (eg, aspirin, ibuprofen) may be needed, but the patient should be aware of the potential complications. NSAIDs are often helpful, especially if the patient has not been using them previously. If one NSAID is not effective, another should be tried. To prevent frequent tension-type headaches low dosages of a tricyclic antidepressant such as nortriptyline and amitriptyline can be helpful. They are best taken before bed time because of their sedative effects.

When the tension-type headache symptoms are secondary to another disorder, therapy needs to be extended to that disorder. For example, when the headache is associated with a masticatory muscle disorder, the muscle disorder needs to be managed.³ Headache upon awaking may be related to nocturnal bruxism or sleep breathing disorders (apnea-hypopnea syndrome) and several approaches can be used to address this.

SUMMARY

There are many types of pain conditions that produce orofacial pain. The most common are dental and periodontal pains, which are highlighted elsewhere in this

issue. Some of the other common pain disorders are musculoskeletal, which in the orofacial structures are called TMD. These disorders need to be identified by the dentist. In most cases they can be managed by relatively simple strategies. There are still many other pain disorders of the head and neck that are unrelated to the dental structures. The dentist should be able to differentiate these and refer the patient to the appropriate health care provider for appropriate care.

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