Temporomandibular Disorder Pain and Dental Treatment of Obstructive Sleep Apnea

Robert L. Merrill, DDS, MS

KEYWORDS

• Temporomandibular disorder • Dental treatment
• Obstructive sleep apnea

TEMPOROMANDIBULAR DISORDER AND OROFACIAL PAIN

Occasionally, treatment of sleep apnea with mandibular advancement devices (MADs) may be associated with the development of symptoms of temporomandibular disorder (TMD). The clinician needs to determine whether the problem was caused by the MAD or if the problem occurred coincidentally with use of the device. The use of the MAD may cause transient TMD symptoms when the device is first worn, but usually these symptoms resolve within a few days. For those problems that become persistent, treatment of the symptoms should be focused. This article discusses the different types of TMD/orofacial pain (OFP) problems that may occur during treatment of obstructive sleep apnea (OSA) with a MAD. It is critical that the general dentist who is providing dental devices for OSA perform a thorough physical and neurologic assessment of the temporomandibular joint (TMJ) and associated structures before providing such a device so that preexisting problems are identified and discussed with the patient.

ADVERSE EFFECTS OF MADs

Muscle and Joint Tenderness

Use of MADs may be associated with problems such as muscle pain or joint tenderness.[1–4] Jaw tenderness is one of the most common complaints after patients start using the device. It is important to document the presence of muscle or joint tenderness before the delivery of the device. Pain problems including a headache history should be explored in the face-to-face history. The physical examination should

UCLA School of Dentistry, A3-026 CHS, 10833 Le Conte Avenue, Room 10-157, Los Angeles, CA 90095, USA
E-mail address: rmerrill@ucla.edu

Dent Clin N Am 56 (2012) 415–431
doi:10.1016/j.cden.2012.01.004 **dental.theclinics.com**
0011-8532/12/$ – see front matter

include a neurologic examination, evaluation of jaw function, and a palpation examination of the TMJs and cervical and masticatory muscles. A common complaint of patients with OSA is morning headache. However, muscle pain and most particularly myofascial pain (MFP) are frequently associated with or cause headache. A careful palpation examination, performed as part of the initial examination, helps to document preexisting muscle pain and associated headache.

TMJ tenderness can occur with use of MADs. When an appliance holds the jaw in a protrusive position during the night, the joint may become inflamed and tender to palpation. The general term for this condition is capsulitis. Preexisting capsulitis should have been identified before delivery of the appliance, and a definitive diagnosis made at that time. Joint tenderness can be caused by macrotrauma (a sudden injury from a major force) or microtrauma (small repetitive injury) to the joint. Most frequently microtrauma is caused by excessive parafunction both during the day and while sleeping.[5]

Joint Sounds

The presence of joint noises such as clicking or crepitus should also have been determined, diagnosed, and noted in the chart before MAD therapy. Clicking sounds may indicate anterior disk displacement with reduction, whereas crepitus indicates degenerative changes of the condyles. Anterior repositioning is often used to reduce or eliminate clicking. Using a MAD for sleep apnea or snoring may eliminate the TMJ clicking during the night, but the appliance cannot be used in the daytime and the clicking usually returns. Using repositioning appliances 24 hours per day to control or eliminate clicking causes permanent occlusal changes and should not be practiced. Longitudinal studies following clicking over extended periods of time have all shown similar outcomes (ie, the clicks do not get worse and become less of a problem or resolve completely with time).[6–9] Nonpainful clicking does not need to be treated.[10,11]

Joint crepitus (the rubbing sound heard during jaw opening and closing) is often an indication of articular surface remodeling. If the joint is tender to palpation, joint imaging, preferably cone-beam computed tomography (CBCT), should be obtained to determine if degenerative changes have occurred. The combination of crepitus, joint tenderness, and degenerative changes seen on the images is diagnostic of osteoarthritis. If the TMJs are painful at the time of the intake examination, the joint condition should be treated before placement of a MAD because the sleep appliance can aggravate the condition. The clinician should have the patient sign an informed consent form that discusses the current status of the joints and changes that can occur with the use of a MAD. A MAD should be delivered only if the condyles are stable as determined on examination and by palpation and radiographs. Areas of active condylar resorption with pain (eg, osteoarthritis) countermand use of a MAD until the joint inflammation is resolved.[12,13]

Bite Changes

Bite changes have been reported in patients using MADs.[14–16] Commonly, temporary occlusal changes are observed in the morning when the device is removed, requiring the patient to perform some exercises to bring the posterior teeth back together. However, evidence is mounting that long-term use of MADs causes permanent changes in the occlusal relationship. Although patients are given instructions regarding the necessity of performing exercises to bring the posterior teeth back into contact, patients may not perform the exercises as directed and the bite changes can become permanent.[17,18] Changes in the relationship of the maxilla to the mandible have also been documented.[19] These changes represent a gross shift in the jaw

relationship in patients with initial class II malocclusions involving a maxillary overjet, or in class III malocclusions in which the anterior incisors are in an edge-to-edge relationship with no maxillary teeth interference with protrusion beyond the edge-to-edge relationship. Although dentofacial and occlusal changes can be attributed to use of an MAD, a recent report shows that long-term use of the CPAP (continuous positive airway pressure) mask without a MAD can also cause dentofacial changes.[20]

Epidemiology of TMD Symptoms and Bite Changes Associated with MAD Use

Increased occurrences of TMD symptoms are not generally associated with use of MADs in the treatment of OSA.[1,21] However, Clark and colleagues[22,23] reported a prevalence estimate of between 10% and 13% of patients using a MAD who developed TMD symptoms that prevented use of the appliance. In addition, it has been reported that the changes became irreversible in 10% of patients using a MAD.[13,24]

Classification of TMD/OFP

Dentists treating snoring and OSA should be familiar with the classification of TMDs and know how to diagnose and treat these problems when they occur. TMDs are broken down into 3 general categories: masticatory muscle disorders, TMJ articular disorders, and inflammatory disorders. The status of the TMJs and musculature must be determined before treatment of OSA with a MAD. The following sections describe the evaluation, diagnosis, and treatment of these disorders.

Masticatory Muscle Disorders

Myalgia

Myalgia is described as a dull, aching, and continuous pain associated with muscle function. The subjective description of the disorder is then confirmed by palpation of the muscles and looking for replication of the pain complaint. If the palpation-induced pain spreads to sites remote from the normal neurosensory distribution of the muscle area, this indicates MFP and not simply myalgia.

MFP

Orofacial MFP has been described in the literature.[8,25] MFP is defined as muscle pain associated with active or latent trigger points that radiate pain to remote sites such as adjacent muscle groups or nonmuscle structures such as the TMJs, sinuses, or teeth. In performing a differential diagnosis for OFP of unclear origin, the clinician should palpate all of the muscle groups that can potentially refer into the area of the pain complaint to see if the source of the pain is coming from the active or latent trigger point. MFP is the great imitator of other painful conditions. The MFP trigger points may also be associated with autonomic features that could confuse and mislead the unwary clinician into thinking the pain was caused by another problem such as neuropathy, dental pain, or a neurovascular disorder when the source of the pain was muscle.[26] **Fig. 1** shows known trigger points in the orofacial and cervical regions that refer pain into the teeth and head.

A clinical examination is accomplished by a thorough muscle palpation of the masticatory and cervical muscle to evaluate for muscle pain. If the patient has muscle pain, finger pressure on the individual muscles causes pain (myalgia) and may generate a referral of pain, as shown in **Fig. 1**. MFP is typically described as continuous, aching, and variable in intensity. MFP can be confirmed by injecting 0.5% procaine or lidocaine without epinephrine into the trigger point or by using ethyl chloride spray while stretching the involved muscle. The pain should decrease by at least 50% to confirm the diagnosis of MFP.[27,28] Myalgia does not refer remotely.

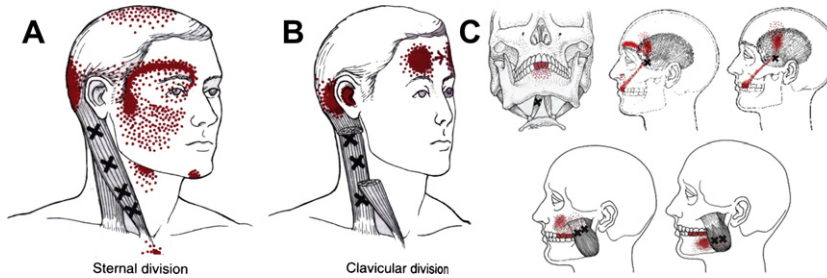

Fig. 1. Myofascial trigger point referral patterns, showing referrals in the orofacial region that are associated with pain and headache. The trigger points are shown as black x's and the referral patterns as the red stippled areas. (*A*) Referrals from the sternal fibers of the sternocleidomastoid muscle. (*B*) Referrals from the clavicular fibers of the sternocleidomastoid muscle. (*C*) Referrals from other masticatory muscles into the teeth. (*From* Simons DG, Travell JG, Simons LS. Travell & Simons' myofascial pain and dysfunction: the trigger point manual. Upper half of body, vol. 1. Baltimore (MD): Lippincott Williams & Wilkins; 1999; with permission.)

Muscle trismus

Muscle trismus or splinting is a protective mechanism that occurs when the muscle fibers shorten and become painful as a protective mechanism limiting movement, or because of trauma. The pain and shortening of the muscle generally avoid repeated trauma. Masticatory muscle splinting is associated with limitation of range of motion and rigidity of the jaw when manipulated. Trismus may be induced as a hysterical reaction caused by psychological distress associated with the pain. Protective splinting is not associated with muscle contraction and increased electromyographic activity when the affected muscle is at rest; consequently, the muscle becomes painful only with function, and splinting increases with stretching of the muscle.[29]

Myositis

Myositis is an inflammatory disorder of muscle caused by infection or trauma within the muscle tissue or by a noninfectious process induced by systemic disease such as polymyositis.[30] Affected pain fibers in the muscles release inflammatory mediators (eg, substance P and calcitonin gene-related peptide [CGRP]), causing the classic signs of inflammation (ie, rubor, dolor, calor, tumor). Characteristically, the myositic muscle is tender to light touch (allodynia), palpation, and functional movement, and, in addition, signs of classic inflammation such as redness and swelling are evident. By comparison, myalgia and MFP are not usually associated with swelling and redness, although pain is induced by palpation and jaw function. Furthermore, in myositis, the inflammation is generalized and the entire muscle is usually affected. In myositis, an increased sedimentation rate is expected, but not in myalgia.

TMJ Articular Disorders

Disk derangements are common in the general population, with prevalence estimates ranging from 40% to 75% of the population.[31] Major trauma also may damage the disk or ligaments (eg, fight, fall, sports injury, oral surgery, or motor vehicle accident), causing the disk to become displaced. Excessive parafunctional activity, such as gum chewing, bruxism, bracing, or clenching, can cause condylar remodeling due to the microtrauma from these parafunctional activities. These behaviors are believed to produce repetitive strain of the joint tissues. In addition, generalized disk ligament

laxity may allow the disk to slip forward, leading to disk clicking. Furthermore, disk noises may be an early manifestation of the changes seen in a developing systemic arthritic disease process, altering the condylar form and allowing disk slippage.[32]

The TMD mechanical problems are subcategorized as follows:

1. Disk displacement with reduction: the joint clicks
2. Disk displacement without reduction (close lock): the joint used to click but is now silent
3. Open dislocation
4. Open lock
5. Posterior disk displacement
6. Ankylosis.

Clicking joints

Historically, TMJ clicking was treated with full-time use of an anterior repositioning appliance in an attempt to reduce the anteriorly displaced disk.[33] Although this treatment is still advocated, more recent longitudinal studies suggest that the clicking eventually returns. Further, studies have thrown into doubt the theory that the disk was recaptured.[34–38] However, a subsequent magnetic resonance imaging (MRI) study showed that the displaced disk was not reduced, although the clicking was eliminated.[38] Another study comparing repositioning appliances with flat-plane stabilization appliances for treatment of disk displacements[39] concluded that the repositioning appliance was no better than the stabilization appliances as a treatment option for disk displacement. Treatment of clicking joints is not advocated unless severe pain and dysfunction are associated with the dislocation.[40] In painful clicking and joint dysfunction, the clinician may need to consider fabricating an anterior advancement splint for nighttime use until joint inflammation subsides and the joint adapts to the mechanical dysfunction. Use of a MAD sleep appliance can provide this kind of stabilization. Most anteriorly displaced disks do not cause pain and do not need to be treated.[6,40–42] We do not fully understand why disk slippage occurs but condylar remodeling or stress-induced alterations of the fibrocartilage lining of the joint may predispose the disk to slip forward and occasionally cause pain. In addition, clicks in the joint may not indicate displacement but can be caused by tears or injury to the disk. Joint noises may also be caused by a stick-slip phenomenon, in which the articular surface of the joint is inadequately lubricated, causing the disk to briefly stick to the anterior surface of the eminence. Evidence has accumulated showing that most clicks do not progress to locking, so MRI is not necessary since it would not substantially alter the treatment approach used. In the presence of pain and increased sticking of the disk, CT imaging of the joints should be obtained as part of the diagnostic workup before initiating any joint procedures such as steroid injections or mandibular repositioning.

Locking joints

If the joint is locking and painful, the patient should be referred to a specialist for treatment. The locked joint may need to be manipulated under local anesthetic or an arthrocentesis or lysis and lavage performed to relieve the condition. Stabilization or repositioning appliances may be used after the joint has been unlocked, but these devices cannot be expected to change the status of the lock without some type of joint procedure to remobilize the joint. After a joint procedure, a repositioning appliance may need to be used full time for 1 to 2 weeks to stabilize the joint, allow the inflammation to subside, and discourage the disk from slipping and relocking. This is

a short-term expedient and long-term 24-hour-per-day use of this type of splint should be limited because it may cause permanent bite changes.

Inflammatory Joint Disorders

Although several monoarthritic and polyarthritic conditions can affect the TMJs, this article focuses only on the most likely conditions to be confronted by a general dentist who is treating OSA (ie, capsulitis, osteoarthritis, and rheumatoid arthritis). TMJ arthritis is not a unified disorder. Several inflammatory conditions can affect the TMJ but many are obscure and not often seen; however, most show overlapping clinical symptoms. The diagnosis of many inflammatory joint disorders is made based on the clinical signs and radiological and laboratory findings. However, most TMJ arthritis seen in a clinical setting is degenerative or osteoarthritic in nature and is not reflective of systemic disease. The diagnosis of osteoarthritis is made from the history, clinical signs, and symptoms rather than from laboratory findings. The TMJ apparatus, including disk function, is susceptible to arthritic changes, and pain may precede the degenerative changes.

Arthralgia/capsulitis

Inflammatory conditions of the TMJs are categorized as localized arthralgia (capsulitis), localized arthritis, and polyarthritis involving the TMJs. Localized and specific joint pain or tenderness is called arthralgia or capsulitis. These terms are used after tenderness has been confirmed with palpation. Arthralgia is used to describe palpable joint pain with no evidence of crepitus or osseous changes on the radiographs. The terms retrodiscitis or synovitis are occasionally used when the dorsal aspect of the TMJ capsule is tender to palpation, but these terms imply a diagnosis that can be made only through biopsy evaluation of the tissue.

The most important cause of arthralgia is trauma, either from external injury or traumatic parafunction. Trauma induces a local inflammation in the joint that is associated with inflammatory mediators released into the joint space. The mediators cause sensitization of the joint nociceptors, joint swelling, warmth, and pain. The inflammatory response causes alterations of the soft and hard tissues in the joint, leading to bony remodeling. Other causes of joint pain may be infection or a localized manifestation of a polyarthritic disorder.

Trauma may induce arthritis when the intra-articular soft tissues are compressed from trauma. The mandible may deviate to the side of the injury in the intercuspal position as a result of damage to the joint structure, muscle guarding, or swelling of the involved joint. The opening is restricted by inflammation in the joint and by guarding and trismus of the masticatory muscles. This situation often causes a shift in muscle-guided closing and opening positions of the jaw. If the joint is edematous, there may be no tooth-to-tooth contacts on the ipsilateral side.

The pain is described as aching in the preauricular area and often in the ear itself. It is aggravated by jaw function such as chewing, talking, opening wide, and lateral movements of the jaw. Palpation of the lateral and dorsal capsules of the joints replicates the pain. In addition, pain is caused by manually loading the joint, pushing the condyle up and back in the fossa, or by having the patient bite on a tongue blade placed between the posterior teeth on the opposite side. Wearing a MAD may cause capsulitis in some patients, particularly when the appliance is removed in the morning and the patient clenches the jaw to bring the posterior teeth into contact. Because capsulitis represents a localized inflammation of the capsule and retrodiscal tissues, the condition needs to be treated to lessen the likelihood that the continuation of joint inflammation leads to degenerative joint changes. Capsulitis is not seen with

radiographic examination unless it is severe and associated with inflammatory mediated joint effusion. Excessive loading of the joints is associated with free radical production and release of inflammatory neuropeptides such as substance P and CGRP, which cause neurogenic inflammation and swelling in the joints.[34,43,44] This process leads to breakdown of the TMJ and osteoarthritis.

Osteoarthritis

As mentioned earlier, TMJ osteoarthritis is one of the most common joint problems seen in the population that is being treated for OSA with a dental device. The prevalence of osteoarthritis increases with age, and this is the same age group in which the prevalence of OSA is more likely to occur. Osteoarthritis of the TMJ may be limited to 1 joint (monoarticular) or both (diarticular). The monoarticular form is more likely a result of trauma to the joint, whereas bilateral changes are more often caused by systemic factors or traumatic parafunctional activity. In addition, a previous nonreducing disk displacement may lead to degenerative changes in the affected joint.[45]

The criteria for making a diagnosis of osteoarthritis are (1) palpable joint pain, (2) crepitus, and (3) radiological evidence of joint degeneration. Most arthritic TMJs have crepitant noises with joint movement. The presence of osteoarthritis needs to be ascertained in the initial examination. In addition, degenerative changes and crepitus may be present without pain. The degenerative changes could make the TMJ more susceptible to abnormal stresses with the use of a MAD.

If no pain is found on palpation of the joints but crepitus is present, or if the joint is palpably tender and crepitant, further evaluation with joint radiographs is warranted. Usually, TMJ osteoarthritis does not manifest swelling or redness because the magnitude of any inflammation is small when compared with the systemic autoimmune inflammatory disorders. Nevertheless, an acute inflammation of an arthritic joint may be associated with swelling and subsequent loss of posterior occlusal contacts on the side of the swelling. Once the joint has been treated appropriately, the occlusal contacts normalize.

There are 2 major types of traumatic injuries that induce local osteoarthritis: macrotrauma, which is caused by an external sharp force against the jaw and microtrauma as a result of repeated parafunctional activity. Another less recognized form of trauma leading to TMJ osteoarthritis under conditions of normal function occurs when a patient has had a previous disk derangement (displacement without reduction), causing joint degeneration probably as a result of loss of the disk, the shock absorber of the joint.[46] As discussed earlier for capsulitis, the neurogenic inflammatory process related to pain and swelling in the TMJ also leads to both soft tissue and bony degeneration of the TMJs.

There are no biologic markers for osteoarthritis that can be determined in laboratory tests, so, as noted earlier, the diagnosis relies on palpation to confirm the pain is of joint origin, auscultation of joint noises, and radiographs (CBCT) (**Fig. 2**) of the TMJ and other joints to confirm if the arthritic remodeling process is present.

Rheumatoid arthritis

Rheumatoid arthritis is an autoimmune disorder that causes the destruction of the patient's own tissues. It starts as an inflammatory soft tissue disease, which may not show radiographic evidence of bony degeneration until the later stages of the disease. This form of arthritis is not as common as osteoarthritis but is seen frequently in older patients, who also may have OSA. The antigenic trigger of rheumatoid arthritis is the synovial tissues and not the articular surface of joint, but as the destruction of the synovia progresses, the entire joint is affected. Typically, rheumatoid arthritis in the

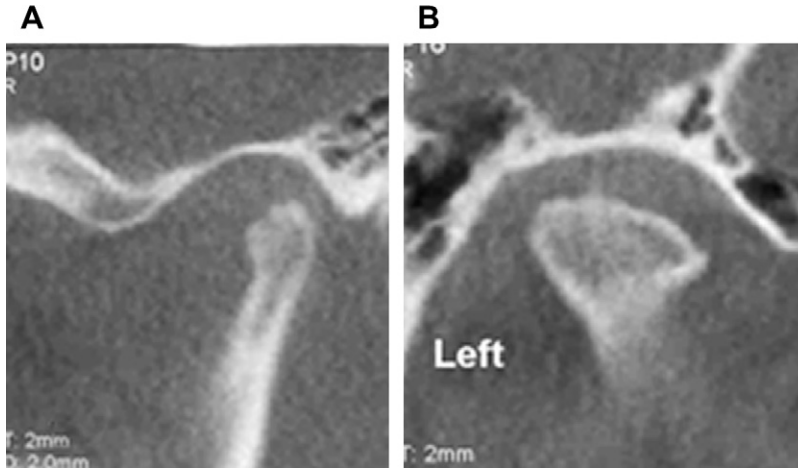

Fig. 2. CBCT of left TMJ with osteoarthritic changes. (*A*) Sagittal view of the condyle, with obvious flattening and roughness of the articular surface. (*B*) Coronal view of the condyle, with flattening particularly obvious on the lateral pole of the condyle.

TMJ is also accompanied by pain in other joints, and the older patient may present with joint disfigurement. Rheumatoid patients present with restricted opening and a progressive anterior open bite. The patient should be under the care of a rheumatologist and a referral should be made if the disease is suspected. Use of a MAD for sleep apnea is problematic because the forces of the appliance could adversely affect the joint. Laboratory tests are performed to look for positive rheumatoid factor (RF) or anti-CCP (anticyclic citrullinated peptide antibody). These tests are positive in 70% of rheumatoid patients but if the RF factor is positive, the anti-CCP test does not give any additional useful information.

THE TMD EXAMINATION AND DIAGNOSIS

There are several parts of the physical examination for the evaluation of a patient with sleep apnea. This article focuses on the OFP examination. The data gleaned from this examination should identify potential problems that may develop or be aggravated when a patient is using a MAD. The overall OFP examination is broken down into 5 parts: (1) the chief complaint and history of present illness, (2) the cranial nerve examination, (3) the stomatognathic examination, (4) the palpation examination, and (5) other examinations and tests.

Chief Complaint and History of Present Illness

The chief complaint and history of present illness as well as a review of current pain symptoms include onset, quality, location, temporal pattern, course of pain over a 24-hour period, modifying factors, and impact of the pain. Because headache is often a symptom of sleep apnea, the pain history should also include a detailed headache history. It is important in reviewing the headache history to ask about headache accompaniments such as sensitivity to light and sound, presence of nausea (symptoms of migraine), and autonomic features that occur with the headache such as nasal congestion, conjunctival injection, ptosis, and tearing (symptoms of the autonomic cephalalgias).[47] In addition, the clinician should always be aware that not all

headaches are benign or primary headache disorders. Important red flags always to consider when evaluating patient are:

1. A new headache that is different from previous headaches
2. The worst ever headache
3. A headache that gets worse when the patient lies down
4. A headache that reaches maximum intensity within seconds to a few minutes
5. A headache associated with neurologic symptoms such as numbness, loss of vision, confusion, and loss of consciousness.

Cranial Nerve Examination

The cranial nerve screening examination evaluates the gross function of the cranial nerves. This examination is performed to help rule out an intracranial or neuropathic source of the pain. Expanding intracranial lesions are space occupying and begin to put pressure on structures and nerves that cause pain or impair function (**Table 1**).

The Stomatognathic Examination

The stomatognathic examination evaluates the general function of the jaw, including the range of comfort opening, active opening, and passive stretch opening, as well as the extent of lateral jaw movements and whether the opening path deviates or

Table 1
Cranial nerves and function. The cranial nerve examination should be part of the OFP and sleep examination. A demonstration of the examination can be viewed at http://www.neuroexam.com

Cranial Nerve	Name	Function
I	Olfactory	Smells
II	Optic	Sees
III	Occulomotor	Extraocular eye movements, constricts pupils
IV	Trochlear	Extraocular eye movement (looks to nose and down)
V	Trigeminal	Sensory to face, motor to muscles of mastication
VI	Abducens	Extraocular eye movement (looks to side)
VII	Facial	Motor for facial muscles of expression, sudomotor for crying and spitting, visceral sensory for taste from two-thirds of lateral border of tongue, somatic sensory to anterior wall of external auditory meatus
VIII	Vestibulocochlear	Hearing and balance
IX	Glossopharyngeal	Elevates palate, parotid salivation, somatic sensory from middle ear, external auditory meatus, pharynx and posterior one-third of tongue. Taste from posterior one-third of tongue, chemoreceptors, and baroreceptors of carotid body
X	Vagus	Swallows, parasympathetic enervation to heart lungs, digestive tract, sensation from pharynx, meninges and external auditory meatus, tastes from epiglottis and pharynx, chemoreceptors and baroreceptors of aortic arch
XI	Accessory	Turns head (sternocleidomastoid muscle) and shrugs shoulders (trapezius muscle)
XII	Hypoglossal	Intrinsic muscles of tongue. Sticks out the tongue

deflects during opening. The examination also determines the presence of joint noises, both crepitus and clicking, and whether there are anomalies in the tooth-to-tooth contacts. These deviations from normal should be charted carefully because wearing a MAD may change the tooth-to-tooth contacts and jaw relationships. Severity of tooth wear and tongue/cheek ridging are also charted.

The Palpation Examination

The palpation examination evaluates the TMJs and muscles for pain by separately palpating each joint and muscle to determine the presence of pain and possible referral patterns from the muscles. In this part of the examination, the examiner is trying to replicate the patient's pain complaint. The dorsal and lateral poles of each condyle are palpated separately and the patient is asked if it is painful. Each of the masticatory muscles is palpated separately, and during the palpation the patient is asked how severe the pain is on a scale of 0 to 3 and if the pain is radiating away from the palpation site. This is an important distinction that helps to differentiate myalgia from MFP. As shown in **Fig. 3**, each muscle is palpated separately with 1 finger that rubs across the muscle fiber at the palpation site with a pressure of approximately 1.8 to 2.3 kg (4 to 5 pounds). This test allows a myofascial trigger point to develop the characteristic radiation pattern for that muscle (**Fig. 4**).

TREATING TMD PROBLEMS DURING MAD THERAPY FOR OSA

Treatment of TMD is based on the type of problem that has occurred. In addition to joint pain, the most common problems associated with TMDs are myalgia and MFP. As discussed earlier, it is important to distinguish between the 2 disorders in the examination by looking for referral pain and radiation of tenderness from the palpated tender areas of the muscles. In addition, changes in occlusion that may occur during the use of a MAD need to be addressed early in the MAD treatment because changes that have been ignored or unevaluated for weeks to months are most likely irreversible with simple exercises, and more involved dental restorative procedures or orthodontics may be needed to resolve the problem.[48,49]

Capsulitis (The Tender Joint)

If the patient is complaining of pain in the TMJs and the joints are tender to palpation, the patient should be instructed to go on a soft diet, avoiding chewing any hard foods,

Fig. 3. Muscle palpation examination showing the palpation of the muscles using a single digit and palpating each muscle individually. This test is performed to allow a myofascial trigger point, if present, to develop and radiate.

Fig. 4. Joint palpation is performed by pushing on the lateral and dorsal aspects of the TMJ.

cutting food (eg, apples) into small bites, and steaming vegetables. We may think of salads as soft but they usually require a lot of chewing, so the patient should be wary of eating chewy salads until the pain resolves. The patient also should be instructed to reduce jaw function by not chewing gum. In addition, the patient should be instructed to use moist heat on the jaw 2 to 3 times per day. This task can be achieved by folding a bath towel into a square, wetting the towel, wringing the excess water out, placing the towel in a plastic bag, and heating it in a microwave oven for 5 minutes. The towel is then removed from the microwave oven and plastic bag and covered with a dry towel. This hot packet is then placed over the painful joint and held for about 10 minutes. This process should be repeated 2 to 3 times per day (**Fig. 5**).

Antiinflammatory medications such as naproxen (Naprosyn 500 mg taken twice per day or 2 Aleve twice per day) should be prescribed or recommended. If ibuprofen is to be used, a prescription should be written for 600-mg to 800-mg tablets, with instructions to take 1 tablet 3 times per day. These medications should be taken with food. If the patient has a history of stomach irritation such as gastroesophageal reflux disease

Fig. 5. Wet bath towel being folded to heat in the microwave. The towel should be placed in a plastic bag and then heated in the microwave for 5 minutes. After being removed from the microwave, the towel is removed from the plastic bag and covered with a dry towel before placing on the jaw.

or reflux, they can take 20 mg of omeprazole or lansoprazole per day to reduce acidity. If the nonsteroidal antiinflammatory drug is not helpful, a prescription for a methylprednisolone (Medrol) dose pack may be helpful in decreasing the inflammation. For an acutely painful joint, if these procedures are not helping, the patient should be referred to an OFP specialist or oral surgeon, who can inject a steroid or hyaluronic acid into the joint.

In addition to decreasing joint loading by softening the diet, the patient should be instructed to use what has been termed a hinge axis exercise to rotate the condyles in the fossae without opening wide enough to cause translation of the condyles down the eminence. This exercise is performed by having the patient quickly open and close the jaw about a finger width without tapping the teeth in the close position. The procedure is to be performed 10 times consecutively and this process should be repeated several times through the waking day. The movement stimulates the production of synovial fluid, lubricating the joint and helping to decrease inflammation.

The oral appliance needs to be reevaluated to see if it is advancing the jaw too much. The clinician should achieve a balance between the amount of advancement versus the increase in vertical. Studies have shown that increasing the vertical is as important as mandibular advancement.[21] Increasing the vertical is usually less irritating to the TMJ than advancing the mandible.

Anterior Disk Displacement with Reduction (The Clicking Joint)

The presence of a click should have been determined and documented before treatment with a MAD. The differential diagnosis for clicking includes anterior disk displacement with reduction, joint remodeling as a result of mechanical stress to the joint, and osteoarthritis. Joint imaging is not needed if there is no associated pain or crepitus. Clicking as a result of anterior disk displacement does not usually occur with the use of a MAD because the mandibular advancement is expected to reduce the likelihood of the disk slipping anteriorly. However, mandibular advancement and the irritation of the appliance may exacerbate bruxism during the night, with consequent development of clicking associated with the increased working of the joints. In addition, heavy parafunction at night can cause a stick-slip phenomenon, in which the excessive loading of the joint pushes out the lubricating synovial fluid from between the eminence and the disk, causing the disk to stick to the eminence and subsequently reduce and click when the space is relubricated.

Anterior Disk Displacement Without Reduction (The Locked Joint)

Patients who have developed a nonreducing anterior displacement should be seen by an OFP specialists or an oral surgeon who has had experience in dealing with this problem. Treatment involves joint manipulation of the anesthetized joint, arthrocentesis, and pumping of the joint, or arthroscopic surgery. Open joint surgery is rarely needed, and although there are several case series in the literature describing relative success for surgical procedures to remove the disk, plicate the posterior ligament, and suture or staple the disk in place, there are no good prospective, randomized, double-blind clinical trials that show benefit from these procedures.[50]

MYOGENOUS PAIN

Masticatory and cervical muscle tenderness is the most common problem associated with TMD. Treatment requires the use of physical therapy, physical medicine techniques, and possibly medication management to optimize the treatment. The patient who develops muscle tenderness while using a MAD should be given instructions for

Fig. 6. Stretching exercises. The left image shows the tongue on the palate behind the upper front teeth and the jaw being stretched to the point at which the tongue would be pulled off the palate if the patient opened wider. The right image shows the patient stretching the posterior neck muscles that are often painful in association with the jaw muscles.

stretching the masticatory muscles during the day. This process should start after removal of the MAD and the exercises to reestablish posterior tooth-to-tooth contacts. The patient should be instructed to stretch the jaw by placing the tongue on the palate behind the upper front teeth and stretching open as far as they can stretch without pulling the tongue off of the palate (**Fig. 6**). The stretch is held for 6 seconds, repeated 6 times, and the process is repeated 6 times per day. This exercise stretches the masseter, medial pterygoid, and temporalis muscles. If the muscles are severely tender, the patient should use moist heat for 5 minutes before the stretching exercises and then ice to cool down the muscles for 5 minutes after the stretching.

The patient should also be instructed to assume a jaw rest position during the day. This practice helps the patient to avoid daytime clenching that can keep the muscles tender. The patient is instructed to place the tongue on the palate similar to the jaw position assumed for the stretch in **Fig. 6**, but the lips are brought together and the teeth kept slightly apart. The patient is also instructed to breathe through the nose in this position. This rest position helps to reduce masseter, medial pterygoid, and temporalis muscle activity.

ADJUNCTIVE MEDICATIONS

In addition to the medications discussed earlier, other classes of medications can be helpful in mediating the pain. Muscle relaxants can be a helpful addition to treating the

Table 2
Muscle relaxants

Generic	Proprietary	Dose
Cyclobenzaprine	Flexeril	10 mg at bedtime
Tizanidine	Zanaflex	2–4 mg (dosed 3 times a day up to 16 mg)
Baclofen	Lioresal	5–10 (dosed 3 times a day up to 80 mg)
Metaxalone	Skelaxin	800 mg 3 to 4 times a day
Methocarbamol	Robaxin	500–750 mg (1500 mg 4 times a day)
Soma	Carisoprodol	250–350 mg 3 times a day. Use for maximum of 2 weeks

jaw pain (**Table 2**). These medications individually are associated with side effects. The exact mechanism of action is not understood. All of the medications cause sedation, and this may be the main effect they have on muscle activity. Some medications may affect blood pressure and heart rate (tizanidine and cyclobenzaprine) or can cause weight gain (cyclobenzaprine). Carisoprodol is metabolized to meprobamate, which is a major tranquilizer and is addictive.

SUMMARY

This article discusses the evaluation and treatment of TMJ problems from the perspective of symptoms that may be associated with the use of a MAD for treating OSA. A thorough head and neck examination should be performed before initiating appliance therapy for sleep, and any indications of joint dysfunction, pain in joints or muscles, and bite discrepancies should be documented, discussed with the patient, and, if need be, treated before initiating therapy with a MAD. When pain occurs after the patient has started wearing the appliance, procedures for evaluating and treating the pain are discussed. Usually, if pain develops with initial use of the appliance, it is temporary, but the stretching exercises for muscle pain and the hinge axis movement for joint pain help to ameliorate it. If the pain becomes more persistent, the patient should be advised to discontinue use of the appliance while pain management procedures are put in place. The appliance should be reassessed to see if the horizontal advancement can be reduced and the vertical opening can be increased. Often, a change of 1 mm can make a significant change to the pain. Patients who have significant painful arthritic changes in the joints may not be able to wear an appliance until the joint inflammation is controlled by a joint procedure, such as arthrocentesis, lysis and lavage, or arthroscopy. This procedure should be performed only by an OFP specialist or an oral surgeon who has experience in managing acute joint problems.

REFERENCES

1. Hoekema A, Stegenga B, De Bont LG. Efficacy and co-morbidity of oral appliances in the treatment of obstructive sleep apnea-hypopnea: a systematic review. Crit Rev Oral Biol Med 2004;15(3):137–55.
2. Sutherland K, Cistulli P. Mandibular advancement splints for the treatment of sleep apnea syndrome. Swiss Med Wkly 2011;141:w13276.
3. Ferguson KA, Cartwright R, Rogers R, et al. Oral appliances for snoring and obstructive sleep apnea: a review. Sleep 2006;29(2):244–62.
4. Hammond RJ, Gotsopoulos H, Shen G, et al. A follow-up study of dental and skeletal changes associated with mandibular advancement splint use in obstructive sleep apnea. Am J Orthod Dentofacial Orthop 2007;132(6):806–14.
5. Clark GT, Sakai S, Merrill R, et al. Cross-correlation between stress, pain, physical activity, and temporalis muscle EMG in tension-type headache. Cephalalgia 1995;15(6):511–8 [discussion: 451].
6. Mejersjo C, Carlsson GE. Long-term results of treatment for temporomandibular joint pain-dysfunction. J Prosthet Dent 1983;49:809–15.
7. Greene CS, Laskin DM. Long-term evaluation of conservative treatment for myofascial pain-dysfunction syndrome. J Am Dent Assoc 1974;89:1365–8.
8. Greene CS, Laskin DM. Long-term evaluation of treatment for myofascial pain-dysfunction syndrome: a comparative analysis. J Am Dent Assoc 1983;107:235–8.

9. Magnusson T, Carlsson GE, Egermark I. Changes in subjective symptoms of craniomandibular disorders in children and adolescents during a 10 year period. J Orofac Pain 1993;7:76–82.
10. Barghan S, Merrill R, Tetradis S. Cone beam computed tomography imaging in the evaluation of the temporomandibular joint. J Calif Dent Assoc 2010;38(1): 33–9.
11. Clark GT, Merrill RL. The diagnosis and treatment of masticatory muscle pain and dysfunction. In: Sarnat BG, Laskin D, editors. The temporomandibular joint: a biologic basis for clinical practice. 4th edition. Philadelphia: W.B. Saunders, Publisher; 1991. Chapter 21.
12. Kobayashi T, Izumi N, Kojima T, et al. Progressive condylar resorption after mandibular advancement. Br J Oral Maxillofac Surg 2011. [Epub ahead of print].
13. Clark GT. Mandibular advancement devices and sleep disordered breathing. Sleep Med Rev 1998;2(3):163–74.
14. Rose EC, Staats R, Virchow C Jr, et al. Occlusal and skeletal effects of an oral appliance in the treatment of obstructive sleep apnea. Chest 2002;122(3):871–7.
15. Marklund M. Predictors of long-term orthodontic side effects from mandibular advancement devices in patients with snoring and obstructive sleep apnea. Am J Orthod Dentofacial Orthop 2006;129(2):214–21.
16. Otsuka R, Almeida FR, Lowe AA. The effects of oral appliance therapy on occlusal function in patients with obstructive sleep apnea: a short-term prospective study. Am J Orthod Dentofacial Orthop 2007;131(2):176–83.
17. Ueda H, Almeida FR, Lowe AA, et al. Changes in occlusal contact area during oral appliance therapy assessed on study models. Angle Orthod 2008;78(5): 866–72.
18. Ueda H, Almeida FR, Chen H, et al. Effect of 2 jaw exercises on occlusal function in patients with obstructive sleep apnea during oral appliance therapy: a randomized controlled trial. Am J Orthod Dentofacial Orthop 2009;135(4):430 e431–7 [discussion: 430–1].
19. Tsuiki S, Ryan CF, Lowe AA, et al. Functional contribution of mandibular advancement to awake upper airway patency in obstructive sleep apnea. Sleep Breath 2007;11(4):245–51.
20. Tsuda H, Almeida FR, Tsuda T, et al. Craniofacial changes after 2 years of nasal continuous positive airway pressure use in patients with obstructive sleep apnea. Chest 2010;138:870–4.
21. de Almeida FR, Bittencourt LR, de Almeida CI, et al. Effects of mandibular posture on obstructive sleep apnea severity and the temporomandibular joint in patients fitted with an oral appliance. Sleep 2002;25(5):507–13.
22. Clark GT, Arand D, Chung E, et al. Effect of anterior mandibular positioning on obstructive sleep apnea. Am Rev Respir Dis 1993;147(3):624–9.
23. Clark GT, Kobayashi H, Freymiller E. Mandibular advancement and sleep disordered breathing. CDA J 1996;24(4):49–61.
24. Petit FX, Pepin JL, Bettega G, et al. Mandibular advancement devices: rate of contraindications in 100 consecutive obstructive sleep apnea patients. Am J Respir Crit Care Med 2002;166(3):274–8.
25. Laskin DM, Greene CS, Hylander WL, editors. TMDs: an evidence-based approach to diagnosis and treatment. Chicago: Quintessence Publishing; 2006.
26. Part 2, Head and neck pain. In: Simons DG, Travell JG, Simons LS, editors. Travell & Simons' myofascial pain and dysfunction: the trigger point manual, vol. 1. Upper half of body. 2nd edition. Baltimore (MD): Lippincott Williams & Wilkins; 1999. p. 237–77. Chapter 5.

27. Graff-Radford SB. Myofascial trigger points: their importance and diagnosis in the dental office. J Dent Assoc S Afr 1984;39:249–53.
28. Graff-Radford SB. Myofascial pain: diagnosis and management. Curr Pain Headache Rep 2004;8(6):463–7.
29. Clark GT, Seligman DA, Solberg WK, et al. Guidelines for the treatment of temporomandibular disorders. J Craniomandib Disord 1990;4(2):80–8.
30. Clark GT, Seligman DA, Solberg WK, et al. Guidelines for the examination and diagnosis of temporomandibular disorders. J Craniomandib Disord 1989;3(1):7–14.
31. Dworkin SF, LeResche L. Research diagnostic criteria for temporomandibular disorders: review, criteria, examinations and specifications, critique. J Craniomandib Disord 1992;6(4):301–55.
32. Clark GT, Merrill RL. The diagnosis and treatment of internal derangements. In: Sarnat BG, Laskin D, editors. The temporomandibular joint: a biologic basis for clinical practice. 4th edition. Philadelphia: W.B. Saunders, Publisher; 1991. Chapter 23.
33. Farrar WB. Diagnosis and treatment of anterior dislocation of the articular disc. N Y J Dent 1971;41(10):348–51.
34. Clark GT. A critical evaluation of orthopedic interocclusal appliance therapy: design, theory and overall effectiveness. J Am Dent Assoc 1984;108:359–62.
35. Okeson JP. Long-term treatment of disk-interference disorders of the temporomandibular joint with anterior repositioning occlusal splints. J Prosthet Dent 1988;60(5):611–6.
36. Lundh H, Westesson PL, Kopp S, et al. Anterior repositioning splint in the treatment of temporomandibular joints with reciprocal clicking: comparison with a flat occlusal splint and an untreated control group. Oral Surg Oral Med Oral Pathol 1985;60(2):131–6.
37. Lundh H, Westesson PL. Long-term follow-up after occlusal treatment to correct abnormal temporomandibular joint disk position. Oral Surg Oral Med Oral Pathol 1989;67(1):2–10.
38. Tallents RH, Katzberg RW, Macher DJ, et al. Use of protrusive splint therapy in anterior disk displacement of the temporomandibular joint: a 1- to 3-year follow-up. J Prosthet Dent 1990;63(3):336–41.
39. Tecco S, Festa F, Salini V, et al. Treatment of joint pain and joint noises associated with a recent TMJ internal derangement: a comparison of an anterior repositioning splint, a full-arch maxillary stabilization splint, and an untreated control group. Cranio 2004;22(3):209–19.
40. De Leeuw R, editor. Orofacial pain; guidelines for assessment, diagnosis, and management. 4th edition. Hanover Park (IL): Quintessence Books; 2008.
41. Clark GT. Treatment of jaw clicking with temporomandibular repositioning: analysis of 25 cases. J Craniomandibular Pract 1984;2:263–70.
42. Seligman DA, Pullinger AG, Solberg WK. The prevalence of dental attrition and its association with factors of age, gender, occlusion, and TMJ symptomatology. J Dent Res 1988;67(10):1323–33.
43. Fukuoka Y, Hagihara M, Nagatsu T, et al. The relationship between collagen metabolism and temporomandibular joint osteoarthrosis in mice. J Oral Maxillofac Surg 1993;51(3):288–91.
44. Hutchins B, Patel H, Spears R. Attenuation of pro-inflammatory neuropeptide levels produced by a cyclooxygenase-2 inhibitor in an animal model of chronic temporomandibular joint inflammation. J Orofac Pain 2002;16(4):312–6.
45. Pullinger AG, Seligman DA. Multifactorial analysis of differences in temporomandibular joint hard tissue anatomic relationships between disk displacement with and without reduction in women. J Prosthet Dent 2001;86(4):407–19.

46. Sylvester DC, Exss E, Marholz C, et al. Association between disk position and degenerative bone changes of the temporomandibular joints: an imaging study in subjects with TMD. Cranio 2011;29(2):117–26.
47. Silberstein SD, Lipton RB, Dodick D, et al. In: Wolff's headache and other head pain. 8th edition. Oxford (UK); New York: Oxford University Press; 2008. p. 379–430. Chapter 14.
48. Almeida FR, Lowe AA, Sung JO, et al. Long-term sequellae of oral appliance therapy in obstructive sleep apnea patients: part 1. Cephalometric analysis. Am J Orthod Dentofacial Orthop 2006;129(2):195–204.
49. Almeida FR, Lowe AA, Otsuka R, et al. Long-term sequellae of oral appliance therapy in obstructive sleep apnea patients: part 2. Study-model analysis. Am J Orthod Dentofacial Orthop 2006;129(2):205–13.
50. Laskin DM. Surgical management of internal derangements. In: Laskin D, Greene CS, Hylander WL, editors. TMDs: an evidence-based approach to diagnosis and treatment. Chicago: Quintessence Publishing; 2006. p. 469–81.