#### **REVIEW ARTICLE**

#### MEDICAL PROGRESS

## Temporomandibular Disorders

Steven J. Scrivani, D.D.S., D.Med.Sc., David A. Keith, B.D.S., D.M.D., and Leonard B. Kaban, D.M.D., M.D.

THE TEMPOROMANDIBULAR JOINT (TMJ) MAY BE AFFECTED BY INFLAMMAtory, traumatic, infectious, congenital, developmental, and neoplastic diseases, as seen in other joints. However, the most common affliction of the TMJ and masticatory apparatus is a group of functional disorders with associated pain that occurs predominantly in women and was previously known as the TMJ pain dysfunction syndrome. Since 1978, there have been substantial changes in the study of etiologic factors, pathophysiology, diagnosis, and management of what are now called temporomandibular disorders.<sup>1,2</sup> The general perception that all symptoms in the head, face, and jaw region without an identifiable cause constitute a "TMJ" problem is clearly unfounded.

Temporomandibular disorders are defined as a subgroup of craniofacial pain problems that involve the TMJ, masticatory muscles, and associated head and neck musculoskeletal structures. Patients with temporomandibular disorders most frequently present with pain, limited or asymmetric mandibular motion, and TMJ sounds.<sup>3,4</sup> The pain or discomfort is often localized to the jaw, TMJ, and muscles of mastication. Common associated symptoms include ear pain and stuffiness, tinnitus, dizziness, neck pain, and headache. In some cases, the onset is acute and symptoms are mild and self-limiting. In other patients, a chronic temporomandibular disorder develops, with persistent pain and physical, behavioral, psychological, and psychosocial symptoms similar to those of patients with chronic pain syndromes in other areas of the body<sup>5-7</sup> (e.g., arthritis, low back pain, chronic headache, fibromyalgia, and chronic regional pain syndrome), all requiring a coordinated interdisciplinary diagnostic and treatment approach.

Temporomandibular disorders are classified as one subtype of secondary headache disorders by the International Headache Society in the International Classification of Headache Disorders II (2004).<sup>8</sup> The American Academy of Orofacial Pain has expanded on this classification, as shown in Table 1.<sup>9</sup>

The prevalence among adults in the United States of at least one sign of temporomandibular disorders is reported as 40 to 75% and among those with at least one symptom, 33%.<sup>7,9,10</sup> TMJ sounds and deviation on opening the jaw occur in approximately 50% of otherwise asymptomatic persons; these are considered within the range of normal and do not require treatment.<sup>11</sup> Other signs, such as decreased mouth opening and occlusal changes, occur in fewer than 5% of the general population.<sup>12</sup> Temporomandibular disorders are most commonly reported in young to middle-aged adults (20 to 50 years of age). The female-to-male ratio of patients seeking care has been reported as ranging from 3:1 to as high as 9:1.<sup>10,13</sup> Despite the high prevalence of temporomandibular disorders, signs, and symptoms, only 5 to 10% of those with symptoms require treatment, given the wide spectrum of symptoms and the fact that the natural history of this disorder suggests that in up to 40% of patients the symptoms resolve spontaneously.<sup>7,14</sup>

In this review we focus on the most common forms of temporomandibular dis-

N ENGL | MED 359;25 WWW.NEIM.ORG DECEMBER 18, 2008

From the Craniofacial Pain and Headache Center, Tufts University School of Dental Medicine, Tufts Medical Center (S.J.S.); the Department of Oral and Maxillofacial Surgery, Massachusetts General Hospital, and the Harvard School of Dental Medicine (D.A.K., L.B.K.); and Harvard Vanguard Medical Associates (D.A.K.) all in Boston. Address reprint requests to Dr. Kaban at the Department of Oral and Maxillofacial Surgery, Massachusetts General Hospital, 55 Fruit St., Warren Bldg., Suite 1201, Boston, MA 02114, or at Ikaban@partners.org.

N Engl J Med 2008;359:2693-705. Copyright © 2008 Massachusetts Medical Society.

Table 1. Temporomandibular Disorders.*	
Articular disorders	
Congenital or developmental First and second branchial arch disorders: hemifacial microsom Treacher Collins syndrome, bilateral facial microsomia Condylar hyperplasia Idiopathic condylar resorption (condylysis)	iia,
Disk-derangement disorders Displacement with reduction Displacement without reduction (closed lock) Perforation	
Degenerative joint disorders Inflammatory: capsulitis, synovitis, polyarthritides (rheumatoid psoriatic arthritis, ankylosing spondylitis, Reiter's syndrome Noninflammatory: osteoarthritis	arthritis, , gout)
Trauma Contusion Intracapsular hemorrhage Fracture	
TMJ hypermobility Joint laxity Subluxation Dislocation	
TMJ hypomobility Trismus Postradiation therapy fibrosis Ankylosis: true ankylosis (bony or fibro-osseous), pseudoankylo	sis
Infection	
Neoplasia	
Masticatory muscle disorders	
Myofascial pain disorder	
Local myalgia	
Myositis	
Myospasm	
Myofibrotic contracture	
Neoplasia	

\* Classification scheme adapted from the guidelines of the American Academy of Orofacial Pain.<sup>9</sup> TMJ denotes temporomandibular joint.

> orders seen by the primary care physician: myofascial pain disorder, intra-articular disk derangement disorders, osteoarthritis, and rheumatoid arthritis.

#### ETIOLOGY

In 1934, James Costen, an otolaryngologist, evaluated 13 patients who presented with pain in or near the ear, tinnitus, dizziness, a sensation of ear fullness, and difficulty swallowing.<sup>15</sup> He observed that these patients had many missing teeth and, as a result, their mandibles were overclosed. The symptoms seemed to diminish when their missing teeth were replaced and the proper vertical dimension of the occlusion was restored. Costen believed that the malocclusion and improper jaw position were the cause of both of the "disturbed function of the temporomandibular joint" and the associated facial pain. Thereafter, the emphasis of treatment for this condition focused on altering the affected patient's dental occlusion.

More recently, advances in the understanding of joint biomechanics, neuromuscular physiology, autoimmune and musculoskeletal disorders, and pain mechanisms have led to changes in our understanding of the cause of temporomandibular disorders. The cause is now considered multifactorial, with biologic, behavioral, environmental, social, emotional, and cognitive factors, alone or in combination, contributing to the development of signs and symptoms of temporomandibular disorders.<sup>9</sup> Some of the current views of the pathogenesis of muscle and intra-articular disorders are shown in Figure 1.

Various forms of trauma to the TMJ structures (ligaments, articular cartilage, articular disk, and bone) can lead to intra-articular biochemical alterations that have been shown to produce oxidative stress and to generate free radicals. Subsequent inflammatory changes in synovial fluid with the production of a variety of inflammatory cytokines can then lead to alteration in the functioning of normal tissues and degenerative disease in the TMJ.<sup>16-20</sup>

Genetic marker studies of genes involved with catecholamine metabolism and adrenergic receptors suggest that certain polymorphisms (e.g., in the catechol *O*-methyltransferase [*COMT*] gene) might be associated with changes in pain responsiveness and pain processing in patients with chronic temporomandibular disorders.<sup>21-23</sup> Differences in pain modulation have been reported between women and men with these disorders, with women showing decreased thresholds to noxious stimuli and more hyperalgesia. In addition, some studies suggest that the affective component of pain in women with temporomandibular disorders may be enhanced during the low-estrogen phase of the menstrual cycle.<sup>24-27</sup>

Functional brain imaging studies showing changes in cortical circuitry support the concept that temporomandibular disorders are very similar to other chronic pain disorders and may be related to abnormal pain processing in the trigeminal system.<sup>28,29</sup> In particular, muscle pain

Downloaded from www.nejm.org at KAISER PERMANENTE on May 20, 2010 . Copyright © 2008 Massachusetts Medical Society. All rights reserved.



disorders appear to have little, if any, abnormality of the muscles or peripheral tissues and may represent a pain-producing process caused by central sensitization. In addition, numerous biobehavioral studies suggest that there is a connection between chronic temporomandibular disorders and coexisting psychopathology (anxiety and depression disorders, post-traumatic stress disorder, and childhood physical, sexual, and psychological abuse).<sup>29-36</sup>

#### CLINICAL EVALUATION

Although temporomandibular disorders are a common cause of craniofacial pain, it is imperative for the health care provider to obtain a comprehensive history, perform a careful physical examination, and obtain appropriate diagnostic studies to exclude other potentially serious disorders. The differential diagnosis should include odontogenic (caries, periodontal disease) and nonodontogenic causes of facial pain, primary or metastatic jaw tumors, intracranial tumors and tumors of the base of the skull, disorders of other facial structures (including the salivary glands), primary and secondary headache syndromes, trigeminal neuropathic pain disorders, and systemic disease (cardiac, viral, and autoimmune disease, diabetes, and temporal arteritis).

The most common symptom reported by patients with temporomandibular disorders is unilateral facial pain. The pain may radiate into the ears, to the temporal and periorbital regions, to the angle of the mandible, and frequently to the posterior neck. The pain is usually reported as a dull, constant ache that is worse at certain times of the day. There can be bouts of more severe, sharp pain typically triggered by movements of the mandible. The pain may be present daily or intermittently, but many patients have pain-free intervals. Mandibular motion is usually limited, and attempts at active motion, such as chewing, talking, or yawning, increase the pain. Patients frequently describe "locking" of the jaw, either in the closed-mouth position, with inability to open (most common), or in the open-mouth position, with inability to close the jaw. These symptoms are often worse in the morning, particularly in



patients who clench or grind their teeth during sleep. Clenching, grinding of the teeth, and other nonfunctional, involuntary mandibular compensatory movements (so-called oral parafunctional habits) are common.

Along with limitation of motion there is often deviation to the affected side of the mandible on opening and a "clicking" or "popping" noise in the joint. While some of the conditions mentioned previously as important to consider in the differential diagnosis can occur with facial pain, temporomandibular disorders often have a stereotypical presentation, which helps in the diagnostic process. It is well appreciated that pain of cardiac origin (ischemia or myocardial infarction) may coincide with neck, jaw, and face pain, but cardiac pain is generally acute in nature, with very different associated signs and symptoms.<sup>37</sup>

A basic working knowledge of normal anatomy and function of the stomatognathic complex (mouth, jaws, and closely associated structures) is necessary before conducting a physical examination. Figure 2 shows the normal anatomy of the craniofacial region and Figure 3 the gross anatomy of the TMJ. Normal jaw opening begins with contraction of the suprahyoid muscles and inferior head of the lateral pterygoid muscle, causing a rotation of the head of the condyle in the glenoid fossa with the articular disk between the condyle and the articular eminence of the glenoid fossa (mouth opening of up to 20 mm). With further contraction of the inferior and superior heads of the lateral pterygoid muscles (the major jaw-opening muscles), the condyle continues rotational and translational movement (along an arc of motion) down and forward along the articular eminence of the glenoid fossa, with the articular disk moving in a posterior direction over the anteriorly moving condyle to ensure smooth movement (normal mouth opening up to 35 to 55 mm) (Fig. 4).

Physical examination should include observation and measurement of mandibular motion (maximal interincisal opening, lateral movements, and protrusion), palpation of the muscles of mastication (masseter, temporalis, medial and lateral pterygoid muscles) and the cervical musculature, palpation or auscultation of the TMJ, and examination of the oral cavity, dentition, occlusion, and salivary glands and inspection and palpation of the anterior and posterior neck. Auscultation of the carotid arteries and examination of the cranial nerves, with special attention to the trigeminal system, should also be part of the physical examination.

As already noted, noise in the TMJ on mandibular movement is frequently present in patients with temporomandibular disorders. However, noise alone is also a very common finding in people who are completely asymptomatic and may represent a range of normal rather than intra-articular dysfunction.11-14 Muscle tenderness, producing pain or discomfort, is generally found on both extraoral and intraoral palpation of the masticatory muscles. Tenderness may also be present in the anterior neck muscles (suprahyoid muscles and sternocleidomastoid muscles), posterior cervical paraspinal muscles (semispinalis capitus, splenius capitus, and suboccipital muscles), and the upper shoulder muscles (trapezius and levator scapulae).<sup>9,11</sup> There may be mandibular hypomobility and deviation on opening. Finally, the neurologic examination is typically normal, without any objective neurosensory or motor deficits of the trigeminal nerve or other focal cranial-nerve abnormalities.

Diagnostic studies are designed to rule out other disorders. They may include the use of blood and serum inflammatory markers (to rule out autoimmune disorders and vasculitides), imaging,



Figure 3. Gross Anatomy of the TMJ.

In this sagittal section of a specimen from a cadaver, the head of the mandibular condyle (MC) can be seen positioned in the glenoid (or articular) fossa (GF) of the temporal bone. The condyle articulates with the anterior aspect of the anterior articular eminence (E) of the articular fossa. The fibrocartilaginous intra-articular disk (ID) appears in its normal position between the anterior aspect of the mandibular condyle and the inferior aspect of the articular eminence of the temporal bone. The posterior aspect of the intra-articular disk, or bilaminar zone (BZ), is the vascular and ligamentous portion of the disk that attaches to the posterior aspect of the TMJ capsule. Also shown is the insertion of the lower head of the lateral pterygoid muscle (LP) into the anterior aspect of the TMJ capsule, the intra-articular disk (superior head), and the anterior aspect of the neck of the mandibular condyle (inferior head). The proximity of the external auditory canal (EAC) to the TMJ is also evident.

diagnostic nerve blocks, muscle trigger-point injections, and dental models for maxillomandibular analysis.

#### IMAGING

The most important diagnostic advances in temporomandibular disorders during the past 30 years have occurred in imaging techniques for the TMJ. The panoramic radiograph (a single-cut tomogram of the entire jaw) remains the most useful screening tool. Plain radiographs have been almost completely replaced by computed tomography (CT) for evaluation of bony morphology and pathology of the joint, mandibular ramus, and condyle. Conebeam maxillofacial CT is a newer and faster technique, with a lower radiation dose, than conventional whole-body CT.<sup>38</sup> This technique provides thin-slice images on the axial, coronal, and sagittal planes that can be modified with an interactive viewing program that allows close examina-



The major mouth-closing muscles (Panel A) are the masseter, medial pterygoid, and temporalis muscles. In the closed-mouth position, the mandibular condyle is positioned in the anterior aspect of the glenoid (articular) fossa of the temporal bone, with the intra-articular disk in between the bony components. This provides biomechanical functional support for the bony components. The major mouth-opening muscles (Panel B) are the lateral pterygoid and suprahyoid. With the initial contraction of these muscles, the mandibular condyle rotates within the glenoid fossa, primarily through the action of the superior head of the lateral pterygoid muscle. The mandibular condyle continues with rotational and then anterior and inferior translational movement down along the articular eminence through the action of the superior heads of the lateral pterygoid muscle. The intra-articular disk initially moves anteriorly, with the movement of the condyle and then, with progressive translational movement of the condyle for full mouth opening. With full mouth opening, the disk is being stabilized by the posterior bilaminar zone and TMJ capsule attachment and the superior head of the lateral pterygoid muscle and anterior bilaminar zone and TMJ capsule attachment and the superior head of the lateral pterygoid muscle and anterior bilaminar zone and TMJ capsule attachment and the superior head of the lateral pterygoid muscle and anterior bilaminar zone and TMJ capsule attachment and the superior head of the lateral pterygoid muscle and anterior bilaminar zone and TMJ capsule attachment and the superior head of the lateral pterygoid muscle and anterior bilaminar zone and TMJ capsule attachment and the superior head of the lateral pterygoid muscle and anterior joint capsule.

tion of all details of the maxillofacial skeleton. There is also a three-dimensional program that allows for analysis of the potential benefits of surgical procedures. CT images provide fine, threedimensional, anatomic detail of the TMJ and surrounding skeleton.

Magnetic resonance imaging (MRI) has replaced other imaging methods for evaluation of soft-tissue abnormalities of the joint and surrounding region. The anatomy of the joint and the position and structure of the intra-articular disk can be accurately visualized both at rest and in motion (Fig. 5). MRI allows for analysis of the blood supply and vascularity of the condyle and for detection of pathologic accumulations of fluid within and around the joint. Continuous cine MRI allows for evaluation of the joint structures during mandibular movement. In addition, investigators have shown that in intra-articular disk derangement disorders, there is poor correlation between signs and symptoms and displacement of the articular

disk when determined on the basis of imaging studies alone.<sup>39</sup>

Skeletal scintigraphy is useful for evaluating developmental or growth abnormalities of the mandible but is not particularly helpful for diagnosis of temporomandibular disorders.<sup>40-42</sup>

Arthroscopy is a minimally invasive surgical technique that allows direct visualization of the anatomy of the TMJ. Patients who have had nonsurgical therapies for 3 to 6 months and continue to have persistent pain in the joint and decreased range of mandibular movement and find that the condition still interferes with normal daily activities such as talking and chewing may benefit from diagnostic or therapeutic arthroscopy.<sup>43-47</sup> Synovitis, adhesions, cartilage degeneration, cartilage tears, loose bodies, disk degeneration, perforations, and capsular attachment tears can all be documented with arthroscopy.<sup>43,44</sup> Therapeutic surgical arthroscopy of the TMJ (like other joints) can address cartilage and bony pathology, correct

# Figure 5. $T_1$ -Weighted MRI Studies of the TMJ, Closed and Open, Sagittal View.

In the closed-mouth view of the TMJ (Panel A), the mandibular condyle can be seen in the normal position in the glenoid fossa. The cortices of the mandibular condyle and the glenoid fossa components of the joint have low signal intensity and appear black (a). The marrow of the mandibular head and neck of the condyle and the articular eminence have high signal intensity and appear white (b). The intra-articular disk is biconcave, with the posterior band lying over the condyle (c). The central zone articulates against the anterior aspect of the condyle and the articular eminence. The fibrocartilaginous disk has low signal intensity. The posterior disk attachment has relatively high signal intensity as compared with the posterior band of the disk itself because of the fatty tissue in the posterior attachment (bilaminar) zone. The upper and lower heads of the lateral pterygoid muscle (d) are seen extending anteriorly (e) from the TMJ. In the open-mouth view of the joint (Panel B), the condyle (a) is seen in its normal anterior and inferior position at the anterior aspect of the articular eminence of the glenoid fossa. The central thin zone of the normal articular disk (b) has low signal intensity and is visualized between the condyle and the anterior articular eminence. The normal posterior zone of the attachment of the disk (c) is seen as soft tissue posterior to the condyle in the fossa. Both heads of the lateral pterygoid muscle are easily visualized (d).

articular disk and ligament abnormalities, and remove true-joint pathology (synovial chondromatosis, osteoarthritis).

Despite the improvements in diagnostic techniques and new insights into the structure and function of the TMJ, the specific pathophysiology of temporomandibular disorders is not completely understood.

#### TREATMENT OPTIONS

Currently, management of temporomandibular disorders consists of a combination of home selfcare, counseling, physiotherapy, pharmacotherapy, jaw-appliance therapy, physical medicine, behavioral medicine, and surgery. Surgery is performed only to treat structural anatomic pathology that is producing pain and dysfunction. Surgical procedures include arthrocentesis, arthroscopy, open arthrotomy, and combined joint and reconstructive jaw procedures.

The vast majority of temporomandibular disorders (approximately 85 to 90%), whether articular or muscular, can be treated with noninvasive, nonsurgical, and reversible interventions.<sup>9,46,47</sup> For patients with intra-articular disorders that do not



respond to a reasonable course of nonsurgical interventions — generally 3 to 6 months in length — surgical therapy may be considered if the pain is substantial and the limitation of function severe enough to interfere with activities of daily living.

#### COMMON CLINICAL DISORDERS AND THEIR TREATMENT

#### MYOFASCIAL PAIN DISORDER

Myofascial pain disorder of the masticatory muscle system is the most common of all temporomandibular disorders (Fig. 1A). The vast majority of patients present with facial pain, limitation of jaw motion, muscle tenderness and stiffness, along with any number of associated symptoms in the head, face, and neck region. Imaging studies of the TMJ usually show no evidence of anatomic pathology. Patients with myofascial pain disorder

Downloaded from www.nejm.org at KAISER PERMANENTE on May 20, 2010 . Copyright © 2008 Massachusetts Medical Society. All rights reserved. generally respond to the simple, noninvasive treatments described below.

#### Reassurance and Counseling

It is important for the patient to receive counseling on the natural history and course of temporomandibular disorders, the role of stress and parafunctional habits such as clenching and grinding of the teeth, the frequency of the problem in the population, and the self-limited nature of the disorder. Likewise, it is important for the patient to hear from a health care professional that both the physical suffering and the emotional suffering associated with temporomandibular disorders are understood.

#### Rest

Although it is not prudent to immobilize the mandible, the patient should be instructed to avoid extremes of mechanical movements (e.g., yawning, laughing, and jaw clenching). Certain habits that Jaw Appliances may affect jaw function, such as chewing gum and biting fingernails or pencils, should be eliminated.

#### Heat

The application of heat to the sides of the face with a heating pad, hot towel, or hot-water bottle will be comforting and will help to relieve muscle pain. More vigorous treatment may be achieved with ultrasound or short-wave diathermy heat treatments, which are widely available in physical therapv offices.48,49

#### Medications

Nonsteroidal antiinflammatory agents are often of value in the acute stage.50-54 Initial treatment is usually administered for 10 to 14 days, at which time the patient should be reevaluated. Muscle relaxants are frequently used for episodes of acute pain but have not been proven efficacious in chronic conditions.50,55,56 Long-term use of opioid analgesics should be avoided, if at all possible.50,54 Antidepressants have a long history of effectiveness for the treatment of chronic pain. Their use is often justified, especially when the pain and dysfunction are part of the complex of generalized muscle pain with signs and symptoms of depression.57-63 Tricyclic antidepressants are the most widely used, and a bedtime-only schedule of 10 to 50 mg of nortriptyline, desipramine, or doxepin can be expected to alleviate symptoms in 2 to 4 weeks.57 Treatment, if successful, is maintained for 2 to 4 months and then tapered to a low maintenance dose. Selective serotonin-reuptake inhibitors have also been used as part of the treatment regimen.<sup>50,54</sup> However, some of these agents (fluoxetine and paroxetine) have been implicated in producing increased masticatory muscle activity (bruxism), especially during sleep, and are generally not recommended.64-67 The tricyclic antidepressants and some of the newer selective norepinephrine-reuptake inhibitors (e.g., duloxetine) can be recommended and show some efficacy. Anxiolytic agents, such as the benzodiazepines, are also commonly used.<sup>50,54-56,68</sup> Short-term use (a few weeks) of the long-acting benzodiazepines in low doses, typically at night, are recommended (diazepam, 2.5 to 5.0 mg; clonazepam, 0.5 mg). Because of the potential for dependency, benzodiazepine use should be limited and follow-up should be frequent.

Many types of intraoral occlusal orthotic appliances exist for the treatment of temporomandibular disorders, and their multiplicity suggests that the optimal design has yet to be discovered. These devices are worn on the teeth like a retainer or a removable denture and are usually made of processed, hard acrylic. They are designed to improve function of the TMJ by altering joint mechanics and increasing potential mobility, to improve the function of the masticatory motor system while reducing abnormal muscle function, and also to protect the teeth from jaw clenching and potential tooth fracture or attrition. It has been hypothesized that these devices may make patients more conscious of their oral parafunctional habits, altering proprioceptive input and central motor system areas that initiate and regulate masticatory function (often called the oral central pattern generator).69-71

The most common appliance is one that is custom-made of hard acrylic and fits over all the teeth in the dental arch (upper or lower). The patient's dentist should be able to construct and supervise the use of such an appliance. Because of the difficulty in controlling for a study using any type of appliance that is placed in the mouth, there have been few good randomized, controlled, and blinded studies of the long-term efficacy of these oral orthotic appliances. In a 2004 Cochrane database review, it was reported that there is insufficient evidence either for or against the use of oral-appliance therapy.<sup>72</sup> However, with appropriate adjuvant therapies, these devices may play a role in alleviating the pain and dysfunction of temporomandibular disorders in 70 to 90% of patients.<sup>73-76</sup>

There is little evidence to support the belief that malocclusion, loss of teeth, and tooth-to-tooth occlusal interferences are primary causes of TMJ and muscle symptoms.<sup>77-80</sup> However, as a general principle, maxillomandibular tooth-to-tooth interferences and discrepancies in anterior–posterior jaw position (a bad bite) should be eliminated and missing teeth replaced in an effort to achieve optimal dental occlusion and masticatory function. In addition, the long-term efficacy of repositioning nongrowing adult jaws with occlusal splints or functional appliances has not been proven by the available data.

#### **Behavioral Approaches**

Counseling, relaxation techniques, stress management, work pacing, guided imaging, biofeedback, cognitive therapy and other behavioral approaches to treatment have all been reported as helpful.<sup>80-84</sup> A 1996 National Institutes of Health Consensus Conference on Behavioral Medicine in the Management of Chronic Pain outlined techniques that are considered effective and indications for using them.<sup>85</sup> The most important factor, however, is the therapeutic interaction (context effect) of the practitioner with the patient.

#### **Physical Medicine**

Manual manipulation, massage, ultrasonography, and iontophoresis are helpful in reconditioning and retraining the masticatory and the other craniocervical muscles that are usually involved in temporomandibular disorders.<sup>49,86-89</sup> Passive motion has also been reported as effective in rehabilitating some of the biochemical and biomechanical changes that occur in injured synovial joints, muscles, and periarticular tissues.<sup>90,91</sup> Several commercial passive motion devices for the jaw (similar to those used for the knee) are currently in use for temporomandibular disorders.

#### INTRA-ARTICULAR DISK DERANGEMENT DISORDER

Disk derangement disorder is defined as a temporomandibular disorder resulting from displacement of the TMJ disk from its normal position or from deformation of the disk. This may lead to synovitis, pain, and limitation of motion (Fig. 1B).<sup>9</sup> The diagnosis is confirmed by history, clinical examination, and MRI scan in the open- and closedmouth positions. Diagnostic or therapeutic arthroscopy may also be helpful in confirming the diagnosis and providing minimally invasive surgical manipulation, if necessary.<sup>47,92</sup>

Internal derangements may include anterior displacement of the disk, with reduction or without reduction.9 Anterior displacement with reduction is defined as disk displacement in the closed-mouth position that reduces (with a click) to the normal relationship at some time during opening. Reduction implies that to some extent the disk is gliding normally, with opening and translational movement. In these circumstances, the patient reports a click with a variable amount of pain on opening. Often, patients have no pain with this condition. The mandible deviates to the affected side on opening until the click occurs and then returns to the midline. This situation may worsen, and there may be intermittent locking of the disk.

Intermittent locking may progress over time to anterior disk displacement without reduction (closed lock).9 This implies that the dislocated disk acts as a mechanical obstruction to the opening and translation of the condyle. These patients have a marked decrease in mandibular opening on the affected side and a variable amount of pain. It feels to them as if there is a mechanical obstruction to opening in the joint. Maximal opening may be limited to 20 to 25 mm (the normal range of maximal interincisal opening ranges from 35 to 55 mm, with a mean of 40 to 43 mm), with restricted movement to the contralateral side. There may also be a history of clicking with intermittent locking. MRI shows a displaced disk without reduction on opening (closed lock) and may also reveal degenerative changes in the condyle. In such cases, the signs and symptoms of degenerative joint disease may also be present.93

Initial treatment for internal derangement consists of the same noninvasive therapies used in treating myofascial pain dysfunction syndrome: behavioral medicine, occlusal appliances, heat, muscle relaxants, nonsteroidal antiinflammatory agents, physical therapy, and so forth. These strategies are often successful in patients with an anteriorly displaced disk with reduction (intermittent locking). In contrast, patients with a closed lock, especially one that is long-standing, will most often require interventions such as intra-articular injection with steroids, arthrocentesis, or arthroscopy.<sup>94,95</sup>

#### OSTEOARTHRITIS

Osteoarthritis of the TMJ may result from trauma (acute or chronic), infection, metabolic disturbances, and previous joint surgery.<sup>9</sup> The patient reports pain on moving the mandible, limited motion, and deviation of the jaw to the affected side. There may be acute tenderness to palpation of the joint. Joint sounds are described as grating, grinding, or crunching, but not as clicking or popping. Imaging studies typically reveal degenerative changes, remodeling, and a loss of joint space.<sup>96</sup>

The features of degenerative disease of the TMJ are different from those of most other joints in the body. There is a strong predilection for the disease among women in their third or fourth decade. Only a few patients have generalized osteoarthritis. The natural course of the disease suggests that the pain and limitation may "burn themselves out" after as little as several months in some patients.9,92,93 The majority can be kept comfortable with the use of noninvasive techniques until remission. In the acute phase, patients may require intra-articular injection of a long-acting corticosteroid such as beclomethasone or hvaluronic acid.94 Neither corticosteroids nor hyaluronic acid are recommended for long-term use, and the data on their effectiveness remains equivocal. These injection treatments are generally reserved for older patients and are limited to two or three injections separated by 4 to 6 weeks. When these techniques are not effective, surgery may be indicated to remove the loose fragments of bone (so-called joint mice) and reshape the condyle.

#### RHEUMATOID ARTHRITIS

There may be involvement of the TMJ in adults and children with rheumatoid arthritis. Among children with juvenile idiopathic arthritis (also known as juvenile rheumatoid arthritis), 50% pre-

### sent with pain, swelling, or limitation of motion in the TMJ.97 There may be associated growth restriction of the jaw resulting in micrognathia and ankylosis. In adults with long-standing rheumatoid arthritis, symptoms may develop in the TMJ late in the course of the disease, and these patients may report discomfort only when they have marked limitation of jaw motion. Other signs of rheumatoid arthritis will be evident. Imaging of the TMJ varies depending on the stage of the disease, but ultimately there is resorption of the condyle, with shortening of the mandibular ramus-condyle unit and potential reduction of joint space and hypomobility. Medical management, along with altering the biomechanics of the TMJ with the approaches mentioned earlier (physical medicine, jaw appliance, and biobehavioral therapy), may be helpful initially. If medical management is not effective, surgical treatment may be necessary, as it is for other joints in the body.

#### CONCLUSIONS

Temporomandibular disorders remain a frequent cause of visits to primary care physicians, internists, and pediatricians. Some approaches to understanding the basic causes of these conditions may prove to be promising, as much of the fundamental pathophysiology remains poorly understood. Substantial improvements have been made in our diagnostic and imaging capabilities, and some treatment advances have been helpful in the long-term management of these common disorders. Future efforts in the fields of genetics, pain research, and arthritis offer the possibility of better defining this heterogeneous group of disorders and providing more focused and effective treatment strategies.

Dr. Scrivani reports receiving grant support from Glaxo-SmithKline and Endo Pharmaceuticals. Dr. Kaban reports receiving grant support for fellows in his unit who hold AO–Sythes– Massachusetts General Hospital Pediatric Oral and Maxillofacial Surgery Fellowship Grants. No other potential conflict of interest relevant to this article was reported.

#### REFERENCES

1. Guralnick W, Kaban LB, Merrill RG. Temporomandibular-joint afflictions. N Engl J Med 1978;299:123-9.

2. Laskin DM. Temporomandibular disorders: a term past its time? J Am Dent Assoc 2008;139:124-8.

**3.** Okeson JP. Bell's orofacial pains: the clinical management of orofacial pain.

6th ed. Chicago: Quintessence Publishing, 2004.

**4.** Dworkin SF, Burgess JA. Orofacial pain of psychogenic origin: current concepts and classification. J Am Dent Assoc 1987;115:565-71.

**5.** Fordyce WE. Pain and suffering: a reappraisal. Am Psychol 1988;43:276-83.

**6.** Parker MW, Holmes EK, Terezhalmy GT. Personality characteristics of patients with temporomandibular disorders: diagnostic and therapeutic implications. J Orofac Pain 1993;7:337-44.

**7.** Solberg WK. Epidemiology, incidence and prevalence of temporomandibular disorders: a review. In: The President's Con-

ference on the Examination, Diagnosis and Management of Temporomandibular Disorders. Chicago: American Dental Association, 1983:30-9.

**8.** Oleson J. The international classification of headache disorders: 2nd ed. Cephalalgia 2004;24:Suppl 1:24-160.

**9.** de Leeuw R. Orofacial pain: guidelines for assessment, diagnosis and management. 4th ed. Chicago: Quintessence Publishing, 2008.

**10.** Schiffman E, Fricton JR. Epidemiology of TMJ and craniofacial pains: an unrecognized societal problem. In: Fricton JR, Kroening RJ, Hathaway KM, eds. TMJ and craniofacial pain: diagnosis and management. St. Louis: Ishiyaku EuroAmerica, 1988:1-10.

**11.** Dworkin SF, Huggins KH, LeResche L, et al. Epidemiology of signs and symptoms of temporomandibular disorders: clinical signs in cases and controls. J Am Dent Assoc 1990;120:273-81.

**12.** Wabeke KB, Spruijt RJ. On temporomandibular joint sounds: dental and psychological studies. (Ph.D. thesis. Amsterdam: University of Amsterdam, 1994: 91-103.)

**13.** Huber NU, Hall EH. A comparison of the signs of temporomandibular joint dysfunction and occlusal discrepancies in a symptom-free population of men and women. Oral Surg Oral Med Oral Pathol 1990;70:180-3.

**14.** Levitt SR, McKinney MW. Validating the TMJ scale in a national sample of 10,000 patients: demographic and epidemiologic characteristics. J Orofac Pain 1994:8:25-35.

15. Costen JB. A syndrome of ear and sinus symptoms dependent upon disturbed function of the temporomandibular joint. Ann Otol Rhinol Laryngol 1934;43:1-15.
16. Milam SB, Zardeneta G, Schmitz JP. Oxidative stress and degenerative temporomandibular joint disease: a proposed hypothesis. J Oral Maxillofac Surg 1998; 56:214-23.

Milam SB, Schmitz JP. Molecular biology of temporomandibular joint disorders: proposed mechanisms of disease.
 J Oral Maxillofac Surg 1995;53:1448-54.
 Zardeneta G, Milam SB, Schmitz JP. Presence of denatured hemoglobin deposits in diseased temporomandibular joints.
 J Oral Maxillofac Surg 1997;55:1242-9.

**19.** Israel HA, Langevin CJ, Singer MD, Behrman DA. The relationship between temporomandibular joint synovitis and adhesions: pathogenic mechanisms and clinical implications for surgical management. J Oral Maxillofac Surg 2006;64:1066-74.

**20.** Ratcliffe A, Israel HA, Saed-Nejad F, Diamond B. Proteoglycans in the synovial fluid of the temporomandibular joint as an indicator of changes in cartilage metabolism during primary and secondary osteoarthritis. J Oral Maxillofac Surg 1998;56:204-8.

**21.** Nackley AG, Tan KS, Fecho K, Flood P, Diatchenko L, Maixner W. Catechol-O-methyltransferase inhibition increases pain sensitivity through activation of both beta2- and beta3-adrenergic receptors. Pain 2007;128:199-208.

**22.** Diatchenko L, Nackley AG, Slade GD, et al. Catechol-O-methyltransferase gene polymorphisms are associated with multiple pain-evoking stimuli. Pain 2006;125: 216-24.

**23.** Diatchenko L, Anderson AD, Slade GD, et al. Three major haplotypes of the beta2 adrenergic receptor define psychological profile, blood pressure, and the risk for development of a common musculoskeletal pain disorder. Am J Med Genet B Neuropsychiatr Genet 2006;141B: 449-62.

**24.** Bhalang K, Sigurdsson A, Slade GD, Maixner W. Associations among four modalities of experimental pain in women. J Pain 2005;6:604-11.

**25.** Diatchenko L, Slade GD, Nackley AG, et al. Genetic basis for individual variations in pain perception and the development of a chronic pain condition. Hum Mol Genet 2005;14:135-43.

**26.** Bragdon EE, Light KC, Costello NL, et al. Group differences in pain modulation: pain-free women compared to pain-free men and to women with TMD. Pain 2002;96:227-37.

**27.** de Leeuw R, Albuquerque RJ, Andersen AH, Carlson CR. Influence of estrogen on brain activation during stimulation with painful heat. J Oral Maxillofac Surg 2006;64:158-66.

28. de Leeuw R, Albuquerque R, Okeson J, Carlson C. The contribution of neuroimaging techniques to the understanding of supraspinal pain circuits: implications for orofacial pain. Oral Surg Oral Med Oral Pathol Oral Radiol Endod 2005;100:308-14.
29. de Leeuw R, Bertoli E, Schmidt JE, Carlson CR. Prevalence of post-traumatic stress disorder symptoms in orofacial pain patients. Oral Surg Oral Med Oral Pathol Oral Radiol Endod 2005;99:558-68.

**30.** Ferrando M, Andreu Y, Galdón MJ, Durá E, Poveda R, Bagán JV. Psychological variables and temporomandibular disorders: distress, coping, and personality. Oral Surg Oral Med Oral Pathol Oral Radiol Endod 2004;98:153-60.

**31.** Manfredini D, di Poggio AB, Romagnoli M, Dell'Osso L, Bosco M. Mood spectrum in patients with different painful temporomandibular disorders. Cranio 2004;22:234-40.

**32.** Dworkin SF, Sherman J, Mancl L, Ohrbach R, LeResche L, Truelove E. Reliability, validity, and clinical utility of the research diagnostic criteria for Temporomandibular Disorders Axis II Scales: depression, non-specific physical symptoms, and graded chronic pain. J Orofac Pain 2002;16:207-20.

33. Auerbach SM, Laskin DM, Frantsve

LM, Orr T. Depression, pain, exposure to stressful life events, and long-term outcomes in temporomandibular disorder patients. J Oral Maxillofac Surg 2001;59: 628-34.

**34.** Turner JA, Dworkin SF, Mancl L, Huggins KH, Truelove EL. The roles of beliefs, catastrophizing, and coping in the functioning of patients with temporomandibular disorders. Pain 2001;92:41-51.

**35.** Campbell LC, Riley JL III, Kashikar-Zuck S, Gremillion H, Robinson ME. Somatic, affective, and pain characteristics of chronic TMD patients with sexual versus physical abuse histories. J Orofac Pain 2000;14:112-9.

36. Fillingim RB, Maixner W, Sigurdsson A, Kincaid S. Sexual and physical abuse history in subjects with temporomandibular disorders: relationship to clinical variables, pain sensitivity, and psychologic factors. J Orofac Pain 1997;11:48-57.
37. Patel H, Rosengren A, Ekman I. Symptoms in acute coronary syndromes: does sex make a difference? Am Heart J 2004;148:27-33. [Erratum, Am Heart J 2005;149:743.]

**38.** Hashimoto K, Kawashima S, Kameoka S, et al. Comparison of image validity between cone beam computed tomography for dental use and multidetector row helical computed tomography. Dentomaxillofac Radiol 2007;36:465-71.

**39.** Israel HA, Diamond B, Saed-Nejad F, Ratcliffe A. The relationship between parafunctional masticatory activity and arthroscopically diagnosed temporomandibular joint pathology. J Oral Maxillofac Surg 1999;57:1034-9.

**40.** Cisneros GJ, Kaban LB. Computerized skeletal scintigraphy for assessment of mandibular asymmetry. J Oral Maxillofac Surg 1984;42:513-20.

**41.** Kaban LB, Cisneros GJ, Heyman S, Treves S. Assessment of mandibular growth by skeletal scintigraphy. J Oral Maxillofac Surg 1982;40:18-22.

**42.** Pogrel MA. Quantitative assessment of isotope activity in the temporomandibular joint regions as a means of assessing unilateral condylar hypertrophy. Oral Surg Oral Med Oral Pathol 1985;60:15-7.

**43.** McCain JP, de la Rua H, Le Blanc WG. Correlation of clinical, radiographic, and arthroscopic findings in internal derangements of the TMJ. J Oral Maxillofac Surg 1989;47:913-21.

**44.** Perrott DH, Alborzi A, Kaban LB, Helms CA. A prospective evaluation of the effectiveness of temporomandibular joint arthroscopy. J Oral Maxillofac Surg 1990; 48:1029-32.

**45.** Nitzan DW, Dolwick MF, Heft MW. Arthroscopic lavage and lysis of the temporomandibular joint: a change in perspective. J Oral Maxillofac Surg 1990;48: 798-802.

**46.** McCain JP, Sanders B, Koslin MG, Quinn JH, Peters PB, Indresano AT. Tem-

poromandibular joint arthroscopy: a 6-year multicenter retrospective study of 4,831 joints. J Oral Maxillofac Surg 1992;50: 926-30. [Erratum, J Oral Maxillofac Surg 1992;50:1349.]

**47.** Israel HA. The use of arthroscopic surgery for treatment of temporomandibular joint disorders. J Oral Maxillofac Surg 1999;57:579-82.

**48.** Truelove E, Huggins KH, Mancl L, Dworkin SF. The efficacy of traditional, low-cost and nonsplint therapies for temporomandibular disorder: a randomized controlled trial. J Am Dent Assoc 2006; 137:1099-107.

**49.** De Laat A, Stappaerts K, Papy S. Counseling and physical therapy as treatment for myofascial pain of the masticatory system. J Orofac Pain 2003;17:42-9.

**50.** Dionne RA. Pharmacologic treatments for temporomandibular disorders. Oral Surg Oral Med Oral Pathol Oral Radiol Endod 1997;83:134-42.

**51.** Schütz TC, Andersen ML, Tufik S. Effects of COX-2 inhibitor in temporomandibular joint acute inflammation. J Dent Res 2007;86:475-9.

**52.** Ta LE, Dionne RA. Treatment of painful temporomandibular joints with a cyclooxygenase-2 inhibitor: a randomized placebo-controlled comparison of celecoxib to naproxen. Pain 2004;111:13-21.

**53.** Kerins C, Carlson D, McIntosh J, Bellinger L. A role for cyclooxygenase II inhibitors in modulating temporomandibular joint inflammation from a meal pattern analysis perspective. J Oral Maxillofac Surg 2004;62:989-95.

**54.** List T, Axelsson S, Leijon G. Pharmacologic interventions in the treatment of temporomandibular disorders, atypical facial pain, and burning mouth syndrome: a qualitative systematic review. J Orofac Pain 2003;17:301-10.

**55.** Rizzatti-Barbosa CM, Martinelli DA, Ambrosano GM, de Albergaria-Barbosa JR. Therapeutic response of benzodiazepine, orphenadrine citrate and occlusal splint association in TMD pain. Cranio 2003;21:116-20.

**56.** Herman CR, Schiffman EL, Look JO, Rindal DB. The effectiveness of adding pharmacologic treatment with clonazepam or cyclobenzaprine to patient education and self-care for the treatment of jaw pain upon awakening: a randomized clinical trial. J Orofac Pain 2002;16:64-70.

**57.** Onghena P, Van Houdenhove B. Antidepressant-induced analgesia in chronic non-malignant pain: a meta-analysis of 39 placebo-controlled studies. Pain 1992; 49:205-19.

**58.** Max MB, Lynch SA, Muir J, et al. Effects of desiprimine, amitriptyline and fluoxetine on pain in diabetic neruopathy. N Engl J Med 1992;326:1250-6.

**59.** Sullivan MD, Robinson JP. Antidepressant and anticonvulsant medication

for chronic pain. Phys Med Rehabil Clin N Am 2006;17:381-400.

**60.** Rowbotham MC, Goli V, Kunz NR, Lei D. Venlafaxine extended release in the treatment of painful diabetic neuropathy: a double-blind, placebo-contolled study. Pain 2004;110:697-706. [Erratum, Pain 2005;113:248.]

**61.** Sindrup SH, Bach FW, Madsen C, Gram LF, Jensen TS. Venlafaxine versus imipramine in painful polyneuropathy: a randomized, controlled trial. Neurology 2003;60:1284-9.

**62.** Goldstein DJ, Lu Y, Detke MJ, Lee TC, Jyengar S. Duloxetine vs. placebo in patients with painful diabetic neuropathy. Pain 2005;116:109-18.

**63.** Denucci DJ, Dionne RA, Dubner R. Identifying a neurobiologic basis for drug therapy in TMDs. J Am Dent Assoc 1996; 127:581-93.

**64.** Kishi Y. Paroxetine-induced bruxism effectively treated with tandospirone. J Neuropsychiatry Clin Neurosci 2007;19: 90-1.

**65.** Malki GA, Zawawi KH, Melis M, Hughes CV. Prevalence of bruxism in children receiving treatment for attention deficit hyperactivity disorder: a pilot study. J Clin Pediatr Dent 2004;29:63-7.

**66.** Lobbezoo F, van Denderen RJ, Verheij JG, Naeije M. Reports of SSRI-associated bruxism in the family physician's office. J Orofac Pain 2001;15:340-6.

**67.** Romanelli F, Adler DA, Bungay KM. Possible paroxetine-induced bruxism. Ann Pharmacother 1996;30:1246-8.

**68.** Singer E, Dionne R. A controlled evaluation of ibuprofen and diazepam for chronic orofacial muscle pain. J Orofac Pain 1997;11:139-46.

**69.** Dao TT, Lavigne GJ. Oral splints: the crutches for temporomandibular disorders and bruxism? Crit Rev Oral Biol Med 1998;9:345-61.

**70.** Fricton J. Myogenous temporomandibular disorders: diagnostic and management considerations. Dent Clin North Am 2007;51:61-83.

**71.** *Idem.* Current evidence providing clarity in management of temporomandibular disorders: summary of a systematic review of randomized clinical trials for intra-oral appliances and occlusal therapies. J Evid Based Dent Pract 2006;6: 48-52.

**72.** Al-Ani Z, Davies SJ, Gray RJ, Sloan P, Glenny AM. Stabilisation splint therapy for temporomandibular pain dysfunction syndrome. Cochrane Database Syst Rev 2004;1:CD002778.

**73.** Ekberg E, Nilner M. Treatment outcome of appliance therapy in temporomandibular disorder patients with myofascial pain after 6 and 12 months. Acta Odontol Scand 2004;62:343-9.

74. Wassell RW, Adams N, Kelly PJ. The treatment of temporomandibular disor-

ders with stabilizing splints in general dental practice: one-year follow-up. J Am Dent Assoc 2006;137:1089-98, 1168-9.

**75.** Al-Ani Z, Gray RJ, Davies SJ, Sloan P, Glenny AM. Stabilisation splint therapy for the treatment of temporomandibular myofascial pain: a systematic review. J Dent Educ 2005;69:1242-50.

**76.** Seligman DA, Pullinger AG. Analysis of occlusal variables, dental attrition, and age for distinguishing healthy controls from female patients with intracapsular temporomandibular disorders. J Prosthet Dent 2000;83:76-82.

**77.** Pullinger AG, Seligman DA. Quantification and validation of predictive values of occlusal variables in temporomandibular disorders using a multifactorial analysis. J Prosthet Dent 2000;83:66-75.

**78.** Tsukiyama Y, Baba K, Clark GT. An evidence-based assessment of occlusal adjustment as a treatment for temporomandibular disorders. J Prosthet Dent 2001;86: 57-66.

**79.** Koh H, Robinson PG. Occlusal adjustment for treating and preventing temporomandibular joint disorders. Cochrane Database Syst Rev 2003;1:CD003812.

**80.** Dworkin SF. The case for incorporating biobehavioral treatment into TMD management. J Am Dent Assoc 1996;127: 1607-10.

**81.** Turk DC. Psychosocial and behavioral assessment of patients with temporomandibular disorders: diagnostic and treatment implications. Oral Surg Oral Med Oral Pathol Oral Radiol Endod 1997;83:65-71.

**82.** Raphael KG, Klausner JJ, Nayak S, Marbach JJ. Complementary and alternative therapy use by patients with myofascial temporomandibular disorders. J Orofac Pain 2003;17:36-41.

**83.** Crider A, Glaros AG, Gevirtz RN. Efficacy of biofeedback-based treatments for temporomandibular disorders. Appl Psychophysiol Biofeedback 2005;30:333-45.

**84.** Stowell AW, Gatchel RJ, Wildenstein L. Cost-effectiveness of treatments for temporomandibular disorders: biopsychosocial intervention versus treatment as usual. J Am Dent Assoc 2007;138:202-8.

**85.** NIH Technology Assessment Panel on Integration of Behavioral and Relaxation Approaches into the Treatment of Chronic Pain and Insomnia. Integration of behavioral and relaxation approaches into the treatment of chronic pain and insomnia. JAMA 1996;276:313-8.

**86.** Venancio Rde A, Camparis CM, Lizarelli Rde F. Low intensity laser therapy in the treatment of temporomandibular disorders: a double-blind study. J Oral Rehabil 2005;32:800-7.

**87.** McNeely ML, Armijo Olivo S, Magee DJ. A systematic review of the effectiveness of physical therapy interventions for temporomandibular disorders. Phys Ther 2006;86:710-25. **88.** Medlicott MS, Harris SR. A systematic review of the effectiveness of exercise, manual therapy, electrotherapy, relaxation training, and biofeedback in the management of temporomandibular disorder. Phys Ther 2006;86:955-73.

**89.** Craane B, De Laat A, Dijkstra PU, Stappaerts K, Stegenga B. Physical therapy for the management of patients with temporomandibular disorders and related pain. Cochrane Database Syst Rev 2008;4: CD005621.

**90.** Israel HA, Syrop SB. The important role of motion in the rehabilitation of patients with mandibular hypomobility: a review of the literature. Cranio 1997;15: 74-83.

**91.** Horrell BM, Vogel LD, Israel HA. Passive motion therapy in temporomandibular joint disorders: the use of a new hy-

draulic device and case reports. Compend Contin Educ Dent 1997;18:73-6, 78, 80, passim.

**92.** Israel HA, Diamond B, Saed-Nejad F, Ratcliffe A. Osteoarthritis and synovitis as major pathoses of the temporomandibular joint: comparison of clinical diagnosis with arthroscopic morphology. J Oral Maxillofac Surg 1998;56:1023-7.

**93.** Dimitroulis G. The prevalence of osteoarthrosis in cases of advanced internal derangement of the temporomandibular joint: a clinical, surgical and histological study. Int J Oral Maxillofac Surg 2005;34: 345-9.

**94.** Bjørnland T, Gjaerum AA, Møystad O. Osteoarthritis of the temporomandibular joint: an evaluation of the effects and complications of corticosteroid injections compared with injection with sodium hyaluronate. J Oral Rehabil 2007; 34:583-9.

**95.** Koslin MG. Advanced arthroscopic surgery. Oral Maxillofac Surg Clin North Am 2006;18:329-43.

**96.** Limchaichana N, Petersson A, Rohlin M. The efficacy of magnetic resonance imaging in the diagnosis of degenerative and inflammatory temporomandibular joint disorders: a systematic literature review. Oral Surg Oral Med Oral Pathol Oral Radiol Endod 2006;102:521-36.

**97.** Kaban LB. Acquired abnormalities of the temporomandibular joint, juvenile rheumatoid arthritis 2nd ed. In: Kaban LB, Troulis MJ, eds. Pediatric oral and maxillofacial surgery. Philadelphia: Saunders, 2004:372-5.

Copyright © 2008 Massachusetts Medical Society.

VIEW CURRENT JOB POSTINGS AT THE NEJM CAREERCENTER

Visit our online CareerCenter for physicians at **www.nejmjobs.org** to see the expanded features and services available. Physicians can conduct a quick search of the public database by specialty and view hundreds of current openings that are updated daily online at the CareerCenter.