EROSS E, DODICK D, EROSS M. THE SINUS, ALLERGY AND MIGRAINE STUDY (SAMS). HEADACHE 2007;47:213–224.

This paper reports a US study of 100 self-diagnosed "sinus headache" patients from the Phoenix area. While previous papers had shown that a very high percentage of such patients actually have some type of migraine headache disorder, this paper digs deeper into clinical phenotypes and reasons for diagnostic confusion. Using the 2004 criteria of the International Headache Society (IHS), the authors found that, of the 63 definite migraine patients, 52 clearly had migraine headaches with or without aura and 11 had migraine associated with medication overuse, while 23% had probable migraine. When the location of their pain was shown on facial maps, nearly two thirds of the 63 definite migraine subjects had pain above and below both eyes, while 16% said their pain was only above the eyes; most of the rest had facial pain as at least one of their pain sites (or their only pain site), so altogether 76% of those patients had pain in V-2 regions. Based on current indications, 92% would be candidates for treatment with triptans.

Several triggers commonly attributed to sinus headache were reported by these subjects, including weather changes, seasonal variations, and allergens. In addition, the presence of cranial autonomic symptoms (CAS), including nasal congestion, eyelid edema, and rhinorrhea, was high in this group (75%), with some patients reporting multiple symptoms. Only 3 patients had an underlying sinus infection that could be construed as a secondary cause for their headaches, while only 9 patients had an unclassifiable headache condition.

The authors emphasize the fact that the majority of undiagnosed migraineurs in the United States are misdiagnosed as having sinus headaches. This appears to be due in part to distractors such as environmental triggers, facial location of the pain, and the presence of CAS, cleverly described by the authors as "a case of guilt by provocation, location, and association." Because the IHS lacks specific classification criteria for these phenomena, the authors have proposed criteria for "Facial Migraine" (usual IHS 1.1 or 1.2 criteria, plus pain in 2 adjacent quadrants in the face), and also for "Migraine with CAS" (usual IHS 1.1 or 1.2 criteria, plus 1 or more typical CAS symptoms). They also attempt to create a new category for the 9 unclassifiable patients based on their common characteristics of bilateral maxillary pain associated with CAS but lacking all features of either migraine or tension-type headache; however, this proposal is not as persuasive as their proposals for facial migraine criteria. *(CSG)*

DIEPPE PA, LOHMANDER LS. PATHOGENESIS AND MANAGEMENT OF PAIN IN OSTEOARTHRITIS. LANCET 2005;365: 965–973.

This seminar paper presents an updated analysis of the heterogeneous group of articular disorders known as "osteoarthritis" (OA). Special emphasis is placed on separating the disease concepts of joint damage from the pain aspects of these conditions. Radiographic observations of the severity of OA in joints have never been well correlated with clinical complaints; this paper summarizes the recent discoveries that help explain this dichotomy. Thus, a section on risk factors for joint damage focuses on both predisposing factors (eg, age, genetics, obesity) and precipitating factors (injury, overloading, instability, etc). This is followed by a section on pathogenesis that deals with both mechanical and inflammatory factors that cause cartilage degradation and subchondral bony changes but not necessarily pain. While OA traditionally has been regarded as a noninflammatory form of arthritis, recent studies show that inflammatory pathways are upregulated in some stages of the disease, which contributes to joint tissue breakdown. The authors note that, in an effort to treat OA, compounds that target proteinases, inflammation pathways, and bone metabolism are being tested in animal studies, clinical trials, and laboratory experiments.

A separate section of the paper deals with risk factors for and pathogenesis of joint pain. Little is known about risk beyond the obvious role of overuse or injury as precipitators of pain, but most evidence points toward psychosocial factors (eg, anxiety, depression, hypochondriasis) as important elements. When pain does occur, aneural cartilage cannot be the cause. Findings of synovitis and subchondral bone changes have been correlated with pain. However, the main issue appears to be a combination of peripheral and central sensitization, with the result that normal stimuli become painful. This problem is further complicated by "reverse causality," in which neurogenic inflammation contributes to joint damage.

A full page is devoted to discussing the role of genetics in osteoarthritis. However, the phenotype question complicates matters (who has the disease—people whose joints appear abnormal in radiographs, or people with painful joints?). There appears to be some evidence for familial aggregation in OA cases. Some studies have identified several chromosomal loci and gene variations as risk factors. Genetic and environmental etiologic factors interact in a complex manner in OA; this interaction has yet to be clearly elucidated.

The final section of this paper discusses diagnosis and management of OA. The reader is cautioned against overreliance on radiographs, which can convince both the patient and the doctor that severe degenerative changes are responsible for the pain symptoms. The authors also note that other common causes of regional or generalized joint pain in older people include referred pain, periarticular (soft-tissue) conditions, and somatization. Gauging the severity of the problem requires both objective assessment of joint discomfort and dysfunction as well as a subjective assessment of the patient's suffering**.** Management of painful OA should be based on the large number of excellent review studies and treatment guidelines that have been published, many of which are referenced by the authors. A pyramidal diagram is presented to illustrate that most patients require only information and advice, self-help strategies, and simple nonsurgical interventions; only a minority need to receive injections, while even fewer should require any type of surgery.

Hopefully, all of the concepts discussed in this article should resonate with the temporomandibular disorders (TMD) therapist community, illustrating once again that most disorders of the temporomandibular joing should be managed within a conservative medical model. *(CSG)*

DODICK D, FREITAG F. EVIDENCE-BASED UNDERSTANDING OF MEDICATION-OVERUSE HEADACHE: CLINICAL IMPLICATIONS. HEADACHE 2006;46(SUPPL 4):S202–S211.

This paper covers the topic of medication-overuse headaches (MOH) in a very thorough manner. It begins with an explanation of this term, which replaces all older terminology such as analgesic rebound, drug-induced, medication-misuse, or withdrawal headaches. The new term has the advantage of being descriptive (because specific criteria defining overuse have been established for all medications) as well as avoiding any specific theory of pathophysiology. The arbitrary choice of 15 or more days per month as the cutoff for identifying MOH persists in the new International Classification of Headache Disorders-2 (ICHD-2, 2004) classification system.

Analgesics and just about every drug used to treat headaches have been associated with MOH, with the exception of dihydroergotamine (DHE) and, surprisingly, nonsteroidal anti-inflammatory drugs (NSAIDs). Studies have shown that simple analgesics or those with combination ingredients are by far the biggest offenders, followed by triptans and opioids. New criteria for MOH that were adopted by the International Headache Society in 2004 are already being modified by their classification committee; these proposed changes also are presented here. The prevalence of MOH in community headache patient samples is fairly high (18% for tension-type headache [TTH], 32%

for migraine), but it is much higher in tertiary-care headache centers. A large meta-analysis of 29 studies showed that MOH patients were using an average of 2.5 to 5.8 medications simultaneously.

The natural history of MOH is largely unknown. In studies of patients who underwent withdrawal therapies and then returned to the community, the relapse rate ranged from 38% to 70% within 4 years. However, 1 study utilizing repetitive intravenous administration of DHE showed much more positive outcomes; this may reflect the effectiveness of DHE in arresting central sensitization, which is a proposed mechanism for the pathogenesis of MOH. It is clear that not every chronic daily headache is related to medication overuse and that not every patient taking medications daily develops this condition. The development of MOH appears to require an as-yet-unknown predisposition factor.

A longitudinal study over an 11-year period of more than 32,000 patients showed that the degree of analgesic use at baseline predicted development of chronic migraine pain as well as chronic MOH pain. Several mechanisms for the development of MOH have been proposed, with the main focus on dysregulation of central pain-inhibitory as well as pain-facilitatory systems; this type of central sensitization is similar to that which occurs in migraine disorders. In the absence of specific drugs that abort or arrest central hypersensitivity, current treatment protocols for MOH include the following 4 steps: *(1)* discontinue overused medications; *(2)* terminate persistent headache (bridge therapy or transition to preventative medications); *(3)* provide behavioral therapy to address overuse patterns; and *(4)* provide instructions for appropriate acute headache control. For patients who present with MOH and intractable pain, the parenteral use of DHE and certain other drugs such as neuroleptics, steroids, diphenhydramine (DPH), valproic acid, and ketorolac has been shown to be effective for terminating intractable migraine.

However, the most important message of this paper is that doctors need to educate their patients about the dangers of MOH. Many over-the-counter medications can lead to these unpleasant headache conditions, because people can and do utilize many of them inappropriately. In addition, doctors need to be aware of this common form of drug abuse. The drugs they prescribe to help people relieve their pain are capable of having a paradoxical effect when improperly used. *(CSG)*

Svensson P. Muscle pain in the head: Overlap between temporomandibular disorders and tension-type headaches. Curr Opin Neurol 2007;20:320–325.

This literature review provides a timely update on this important topic. The fact that patients with myofascial temporomandibular disorders (TMD) are more likely to have a tension-type headache (and vice versa) has led some people to conclude that these are completely overlapping disorders. However, despite the similarities in *(1)* sensitization of nociceptive pathways, *(2)* dysfunction of pain-modulatory systems, and *(3)* contributing genetic factors, there still are a number of distinct differences between these 2 patient groups. In both cases it has been impossible so far to make a mechanism-based classification, so diagnoses are based on well-defined signs and symptoms. The most striking difference between the groups is the presence of spontaneous and/or functional pain upon jaw movement concentrated around the masseter muscle, which occurs only in the myofascial TMD group. This finding is strongly supported by experimental evidence (much of it produced by Svensson and his colleagues) that hypertonic saline injections into the masseter muscle can produce widespread head pain secondarily, while injections into the splenius muscle produce head pain but usually do not lead to pain in the lower part of the face.

The controversial issue of trigger points (TPs) as a major feature of myofascial pain disorders is handled very well in this paper. Both positive and negative experimental papers are reviewed. Topics covered include biochemical changes found (or not found) in the local microenvironment around TPs as well as the relatively poor response of TPs to botulinum toxin injections. Another section on the epidemiology of these 2 disorders describes the significant overlap between them as well as comorbid conditions such as fibromyalgia and chronic fatigue syndrome. It has been shown that widespread somatic pain is a significant risk factor for TMD pain, and a recent study suggests that the development of TMD pain in adolescence may reflect an underlying vulnerability to other types of musculoskeletal pain problems. Both TTH and TMD patients respond to experimental injections of hypertonic saline with significantly more widespread and longer-lasting pain than normal subjects, indicating the presence of both peripheral and central sensitization. It is clear that overlap between TTH and myofascial TMD is a significant issue. However, they are far from being completely similar disorders.

The role of genetic factors as risk factors for the development of painful conditions is discussed briefly, with emphasis on recent papers that have described coding of the catechol O-methyltransferase (COMT) enzyme, which metabolizes catecholamines and is involved in pain perception, cognitive function, and affective mood. Also, the possibility that chronic pain patients may have a dysfunctional endogenous pain-modulatory system is considered, but at present we do not know whether such factors are a cause of or a consequence of persistent myofascial TMD or T-TH problems. A very extensive set of references and recommended readings is presented at the conclusion of this article. *(CSG)*

McNeely ML, Armijo Olivo S, Magee DJ. A systematic review of the effectiveness of physical therapy interventions for temporomandibular disorders. Phys Ther 2006;86:710–725.

This paper describes a thorough analysis of the literature on the effectiveness of physical therapy interventions through 2005. The literature search performed by these authors resulted in a total of 1,138 articles. In 3 appendices at the end of the article, they describe the criteria for including or excluding studies, the Jadad scale for evaluating the quality and internal validity of each study, and a critical rating system developed by de Vet et al to evaluate study parameters such as blinding and outcome measures.

Thirty-six articles made the first cut based on their relevance, but after application of the aforementioned inclusion and exclusion criteria only 14 articles representing 12 clinical studies were left; of these, only 3 were considered methodologically strong.

There was considerable diversity among the diagnostic groups included in these 12 studies: 6 included myogenous TMD patients, 2 included arthrogenous TMD patients, and 3 included both. Half of the studies used the Research Diagnostic Criteria for Temporomandibular Disorders (RDC/TMD) to establish the diagnoses, while the others used TMD signs and symptoms to establish their own diagnoses. Four studies examined the effect of exercise interventions, with 2 considering posture training (which seemed to produce positive effects) and 2 examining oral exercises (which produced mixed results). Acupuncture was utilized in 2 studies. One group of investigators reported that acupuncture was superior to no treatment and equal to an occlusal splint, while the other reported a slightly positive outcome from both real and sham acupuncture. Studies that looked at electrophysical modalities varied considerably in group diagnosis, chosen modality versus comparison or control group, and treatment duration or frequency. Transcutaneous electrical nerve stimulation (TENS), radio-frequency energy, and low-level laser were no better than placebo versions of each for reducing pain, but both laser and biofeedback seemed to improve oral opening. Comparisons of these modalities with occlusal splints or muscle relaxation training produced very diverse outcomes.

The authors conclude by saying that both postural exercises and oral exercises should be considered potentially useful interventions for treating symptoms of TMD, but they acknowledge that much better studies are needed to establish ideal treatment regimens. They state that there is not enough information to support the use of either acupuncture or electrophysical modalities for reducing TMD pain but that oral opening may be improved by the use of biofeedback, relaxation techniques, and low-level lasers. A 3-page table at the end of the article summarizes all the information presented in the 14 selected studies. *(CSG)*