

# The Changing Field of Temporomandibular Disorders: What Dentists Need to Know

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## ABSTRACT

Diagnosis and treatment of temporomandibular disorders (TMDs) have been within the domain of dentistry for many decades. However, the field of TMDs and other causes of orofacial pain is undergoing a radical change, primarily because of an explosion of knowledge about pain management in general. As a result, etiological theories about TMDs are evolving toward a biopsychosocial medical model from the traditional dental framework. Conservative and reversible management approaches (especially of chronic pain conditions) are becoming the norm rather than the exception in treating TMD patients, and already certain biological and psychosocial factors are known to affect the outcomes. Current research in this field is focused on genetic and environmental susceptibility factors as well as individual adaptive potentials. To continue as the main providers of care for TMD patients, dentists will need to recognize and appreciate these important changes.

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Temporomandibular disorders (TMDs) are defined by the American Academy of Orofacial Pain as “a collective term that embraces a number of clinical problems that involve the masticatory muscles, the TMJ [temporomandibular joint], and the associated structures.”<sup>1</sup> Pain and dysfunctional symptoms or signs such as limitations in opening, asymmetric jaw movements and TMJ sounds are the most common findings (**Box 1**).

The concept of TMDs as part of the constellation of musculoskeletal disorders, rather than some special kind of dental condition, is relatively recent. In 1918, Prentiss<sup>2</sup> initiated interest in the dental community when he suggested that the development of “TMJ problems” was due to the following process: “When the teeth are extracted, the condyle is pulled upward by the powerful muscula-

ture and pressure on the meniscus results in atrophy.” This was soon followed by several articles from other dentists, who emphasized missing teeth and lost vertical dimension leading to displacement of the mandible as the cause of the signs and symptoms displayed by patients with TMD.<sup>3-5</sup>

It was not until 1934 that dentists were given ownership of this problem, when J.B. Costen, an otolaryngologist, pronounced that the TMJ was a separate source of facial pain and several other associated symptoms, due to nerve impingement from overclosure of bites, lack of molar support and malocclusion.<sup>6</sup> Over the next 5 years, he followed up with 11 more articles emphasizing these structural concepts as the etiology for TMDs and urging dentists to take responsibility for managing them.

**Box 1** Common signs and symptoms of temporomandibular disorders

- Pain or tenderness in the temporomandibular joint, muscles of mastication, facial areas, ear region, shoulder and neck
- A clicking, popping or grating sound when opening or closing the mouth or while chewing
- Catching or locking of the joint with deviations or deflections of the mandible on opening or closing the mouth
- Limitations in opening or closing the mouth
- Difficulty or discomfort while chewing
- Sensation of an uncomfortable bite

It was subsequently shown that Costen's explanation of the anatomic relations between the TMJ and ear and sinus structures was incorrect.<sup>7,8</sup> However, terms such as overclosed vertical dimension, condylar malposition, trapped mandibles, occlusal disharmony and neuromuscular imbalance developed from the initial conceptual framework, and treatments to correct these problems became the basis for a variety of invasive and irreversible dental therapies, including bite-opening, occlusal adjustments, major restorative dentistry, orthodontics and even surgeries. Whatever one may think of these concepts and interventions, it is clear that they were the basis for a mechanical, dentistry-oriented etiological viewpoint and that the related therapies were seen as being anti-etiological. In fact, the word *definitive* was often used to describe the *curative* value of these approaches to TMD treatment.

Over the next 7 decades, the field of TMDs experienced many taxonomic and conceptual changes. Various labels, such as TMJ syndrome, TMJ pain-dysfunction syndrome and myofascial pain-dysfunction syndrome, were applied to TMDs. Fortunately, single-disease concepts have been discarded because of their simplicity and naiveté, and the early dental mechanical theories of misaligned jaws or faulty occlusal relations have largely been discredited.<sup>9</sup> Today, TMDs are being studied and treated from a medical perspective that involves orthopedic principles, combined with a biopsychosocial understanding of how chronic pain disorders affect those who have them.<sup>10,11</sup> Furthermore, studies of patients with TMD have shown that many of them, especially females, experience a multitude of other functional (nonorganic) disorders, such as fibromyalgia, interstitial cystitis, irritable bowel syndrome and pelvic pain, while others have reported multiple sites of pain throughout their bodies.<sup>12</sup> These high levels of comorbidity with other conditions have led to hypotheses about centrally mediated dys-

regulatory problems producing multiple symptoms in susceptible patients.

The aim of this paper is to make dentists aware of the significant conceptual and practical changes that have already occurred or are in the process of emerging in the field of TMDs, so that they can continue to play an important role in the management of these disorders.

### Etiology of TMDs

Greene<sup>13</sup> defined etiology as the following: "We want to know *why* a particular patient began to have both the biology and the perception of his/her pain (in the absence of frank trauma)." It is within the context of this definition that the etiology of TMDs is discussed here.

In addition to the early views described above, various disciplines of dentistry and other areas of health care have proposed theories about the etiology of TMDs. For example, the field of orthodontics developed its own version of structural disharmony concepts and corrective treatments within an orthodontic framework.<sup>14</sup> Another structural concept of TMD etiology, proposed by some physical therapists, chiropractors and dentists, is based on the notion that "bad" craniocervical relations may be causing TMDs. Although this idea has enjoyed some popularity in the past (and is still popular in some regions of the world), several studies have demonstrated that there are no consistent postural findings that differentiate TMD patients from other people.<sup>15-18</sup> Although many patients complain of concomitant cervical pain and TMDs, this should be understood as comorbidity resulting from functional rather than structural relations. In addition, this common clinical finding may be a result of heterotopic (referred) pain in these areas, due to the neuroanatomic and neurophysiologic convergence of cervical and cranial sensory nerves in the brainstem nuclei.<sup>19,20</sup>

The theories of TMD etiology that have made the largest impact are related to various types of occlusal imperfection. Occlusion is a very important subject within the profession of dentistry, especially as it pertains to orthodontics, restorative dentistry and prosthodontics; however, its relevance to the etiology of TMDs is questionable, especially in chronic conditions. In a review of 57 epidemiological studies of the relation between occlusion and TMDs, Okeson<sup>21</sup> found that 35 suggested a relation compared with 22 studies that suggested no relation. The "positive" occlusal findings in the 35 studies varied so widely that no consistent feature could be identified. The occlusal disharmonies cited in these studies were also prevalent among many symptom-free people.

McNamara and others<sup>22</sup> reviewed the role of morphologic and functional occlusal factors with respect to development of TMDs and found only a weak relation between them. Koh and Robinson<sup>23</sup> systematically reviewed the literature pertaining to occlusal adjustments

**Table 1** Relations among diagnosis, etiology and treatment in TMDs

Standard	Diagnosis	Etiology	Treatment
Ideal	Correct Measurable Demonstrable	Specific Measurable Treatable	Anti-etiological Definitive/curative Successful
Acceptable	Presumptive Probably correct Universal labels	Unclear Complex Reversible	Validated response Matched to diagnosis Conservative
Wrong/bad	Personal label Technologic diagnosis Possibly correct	Experience based Morphofunctional analysis Mechanistic	Prolonged use of an oral appliance Bite-changing procedures Jaw-repositioning procedures
Fringe	Misdiagnosis of pain Neglect pathology Neglect chronicity	Guru/cult concepts Quackery concepts Specialty bias	Whole-body procedures Unorthodox treatments Extreme dental intervention

Adapted with permission from Greene.<sup>13</sup>

for treating and preventing TMD. After reviewing specific outcome measures, they concluded that there was no evidence for the use of occlusal adjustment procedures for either the treatment or prevention of TMD.

In addition to structure, other etiological factors<sup>24,25</sup> have been proposed and discussed as a result of large studies of patient populations. For example, trauma at both the macro and micro levels has been noted in the history of certain TMD patients, with a rather clear relation to onset of symptoms in many cases.<sup>13</sup> A psychophysiological theory of the etiology of TMDs was developed in the 1950s and 1960s, with particular emphasis on the category of myofascial pain and dysfunction.<sup>26–28</sup> Even though Laskin’s classic article about the etiology of myofascial pain and dysfunction<sup>26</sup> served as the basis for much of this work, eventually his psychophysiological theory proved to be incomplete as an explanation for the development of myofascial pain. Today, the importance of psychological factors in the onset, progression, treatment and persistence of various TMDs is well recognized as foundational knowledge in this field. However, the reasons why some patients exhibit TMD symptoms while others do not remains unexplained by the psychophysiological theory of etiology.

Currently the most popular theories regarding TMD etiology are based on the biopsychosocial model, which involves a combination of biological, psychological and social factors.<sup>10,29</sup> These 3 words provide an excellent descriptor of the world that most patients with pain (and especially patients with chronic pain) are living with on a daily basis. They have a *biological* problem (i.e., activation of pain pathways, with or without a demonstrable pathological condition) that may have *psychological* antecedents as well as behavioural consequences. This situation exists in a *social* framework that includes interpersonal relationships with friends, families and health care pro-

viders, which almost always produces major negative experiences for the patients as well as for their immediate families. Unlike the mechanistic dental theories of etiology, the biopsychosocial model encourages a rehabilitation–management approach rather than providing the unrealistic expectation of a permanent cure (which is even less likely in chronic conditions). Unfortunately, due to the limitations of current physical diagnostic procedures for assessing pain conditions, as well as the crude psychometric tools that are currently available, the biopsychosocial model lacks the ability to assess all of these variables at the individual patient level and, therefore, is useful only at the group level.

Dentists should appreciate and recognize that the inability to identify precise etiologies or the lack of a perfect theoretical model does not prevent the rendering of reasonable and effective treatment. It is acceptable, as occurs daily in the medical profession, to provide a presumptive diagnosis that is probably correct, then to deliver reversible, conservative, noninvasive and empirically validated targeted treatments (**Table 1**). For example, a painful TMJ that began to cause pain without any specific initiating event or cause can still be successfully treated using medications, appliances or physical therapy in various combinations. By following these foundational concepts, dentists can take a “low-tech and high-prudence” therapeutic approach to TMD patient care.<sup>30</sup>

### Future Directions in the Field of TMDs

The changes taking place in the field of TMDs are not driven purely by dental research, but are coming more from progress in the larger field of pain management. Multiple research projects around the world involving basic and clinical sciences as well as translational activities (the merging of basic and clinical activities) are greatly influencing our understanding of pain. TMDs are

currently being investigated in terms of orthopedic principles, neurophysiological aspects of pain, neuroanatomic regions of pain processing, molecular and cellular pathophysiology of muscle and joints and behavioural aspects of chronic pain. From these domains, 3 main areas of investigation have emerged.

### **Genetics**

Human genetic studies are providing us with a better understanding of inherent susceptibility to pain, variability in pain perception and responses and the factors that predict risk of chronification of pain.<sup>31-33</sup> Some investigators have looked at catechol-O-methyltransferase (COMT), an enzyme that is responsible for metabolizing catecholamine and is involved in pain perception, cognitive function and mood.<sup>34</sup> Studies have reported that carriers of the low-pain haplotype on the gene that codes for COMT appear to have 2.3 times less risk of developing myogenous TMD.<sup>35</sup> In another study, people who have genetic coding for certain levels of adrenergic receptor expression were shown to be about 10 times less likely to develop TMDs.<sup>36</sup> Numerous other genes code for the neurotransmitters and neuromodulators that influence pain sensitivity.<sup>37</sup> The implications of these findings for the management of patients with pain may ultimately be to tailor treatment approaches to the individual or provide pharmaceutical agents targeted at specific receptors.

### **Pathophysiology**

A plethora of information is erupting regarding the molecular chemistry and cellular biology of various types of pain. Understanding of the pathophysiology of conditions that affect the TMJ has been greatly enhanced by these discoveries. For example, inflammation in the synovial tissues of the TMJ is the main determinant of whether the joint becomes painful. Complex cellular processes such as activation of T cells, macrophages and plasma cells with the expression of a multitude of inflammatory mediators, such as prostaglandins, serotonin, proinflammatory cytokines and their antagonists, drive the inflammatory cascade.<sup>38</sup> It appears that both the absolute levels of this inflammatory “soup” and the balance between pro- and anti-inflammatory substances are important in the pain process and the propensity for chronification.<sup>39</sup> In addition, neurochemicals from sympathetic efferents (neuropeptide Y, norepinephrine and others) and neuroendocrine peptides (substance P, calcitonin gene-related peptides and others) are involved by having bidirectional communication with the immune system and, thus, contributing to TMJ pain.<sup>38</sup>

Currently, the pathophysiology of muscle pain is not as well understood. Numerous mechanisms have been considered as sources of muscle pain, yet the literature has not provided definitive answers. Localized factors, such as microtrauma, local ischemia or hypoperfusion

can produce structural or functional consequences, because of the release of endogenous algescic substances (glutamate, histamine and others) from tissue cells and afferent nerve fibres leading to excitation or sensitization of muscle nociceptors.<sup>40</sup> Central processes involving neuroendocrine factors (endogenous and exogenous hormones) as well as neurophysiological mechanisms (peripheral and central sensitization) also play a role in the pathophysiology of muscular pain.<sup>41</sup> Combinations of local and central factors must also be considered.

As more research is undertaken and new information emerges, dentists should be aware of it and recognize that treatments directed at the underlying pathophysiology of both arthrogenous and myogenous painful conditions will inevitably result in a more precise and targeted medical approach to treatment.

### **Predictive Factors**

Predicting responses to therapeutic interventions in pain patients (including those with TMDs) by identifying certain physical and psychological factors is currently being done with some success.<sup>12,42</sup> A major focus of current research is trying to prevent acute pain conditions from developing into chronic ones. This requires good early intervention and treatment strategies as well as better predictors of who is most likely to develop such problems. The discovery of more predictors should enhance the ability of dentists to develop appropriate treatment plans tailored to the individual patient.

### **Conclusions**

The field of TMDs is undergoing a major transformation as a result of research findings about pain in general, as well as specific advances within the field. As a result, TMDs are currently recognized as a subset of musculoskeletal pain conditions, and this requires a medical perspective to understand and manage TMD patients. For the dental profession, the implications of this information are profound and serious in most TMD cases, but especially in chronic conditions. Essentially, it means that dentists should try to avoid invasive, irreversible and aggressive treatments that are intended to “cure” these problems. Instead, more reversible and conservative medically based management strategies are recommended to reduce pain and improve function, an approach that has been shown to be successful for most TMD patients.<sup>1</sup>

In the future, treatment modalities directed at the pathophysiological processes of joint and muscle pain as well as the psychosocial aspects of chronic pain will need to be tailored to each patient’s individual problems. For now, the cautious approach recommended by Stohler and Zarb<sup>30</sup> (low-tech and high-prudence) must be understood and followed so that dentists can continue to serve as the primary providers of care for TMD patients. If not, then it seems inevitable, as scientific discovery continues and

provides us with a deeper understanding of these patients, that “ownership” of this group of disorders will be lost to other medically oriented health practitioners. ♦

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## References

- Temporomandibular disorders. In: de Leeuw R, editor. American Academy of Orofacial Pain. Orofacial pain: guidelines for assessment, diagnosis and management. 4th ed. Chicago: Quintessence; 2008. p. 131.
- Prentiss HJ. A preliminary report upon the temporomandibular articulation in the human type. *Dent Cosmos* 1918; 60(6):505–12.
- Monson GS. Impaired function as a result of closed bite. *J Natl Dent Assoc* 1920; 7(5):399–404.
- Wright WH. Deafness as influenced by malposition of the jaws. *J Natl Dent Assoc* 1920; 7(12):979–92.
- Goodfriend DJ. Dysarthrosis and sub-arthritis of the mandibular articulation. *Dent Cosmos* 1932; 74(6):523–35.
- Costen JB. A syndrome of ear and sinus symptoms dependent upon disturbed function of the temporomandibular joint. *Ann Otol Rhinol Laryngol* 1934; 43(1):1–15.
- Sicher H. Temporomandibular articulation in mandibular overclosure. *J Am Dent Assoc* 1948; 36(2):131–9.
- Zimmermann AA. An evaluation of Costen's syndrome from an anatomical point of view. In: Sarnat BG, editor. The temporomandibular joint. Springfield: Charles C. Thomas; 1951. p. 82–110.
- Greene CS, Laskin DM. Temporomandibular disorders: moving from a dentally based to a medically based model. *J Dent Res* 2000; 79(10):1736–9.
- Dworkin SF, Massoth DL. Temporomandibular disorders and chronic pain: disease or illness? *J Prosthet Dent* 1994; 72(1):29–38.
- Dworkin SF, LeResche L. Research diagnostic criteria for temporomandibular disorders: review, criteria, examinations and specifications, critique. *J Craniomandib Disord* 1992; 6(4):301–55.
- de Leeuw R, Klasser GD, Albuquerque RJ. Are female patients with orofacial pain medically compromised? *J Am Dent Assoc* 2005; 136(4):459–68.
- Greene CS. The etiology of temporomandibular disorders: implications for treatment. *J Orofac Pain* 2001; 15(2):93–105.
- Greene CS. Orthodontics and temporomandibular disorders. *Dent Clin North Am* 1988; 32(3):529–38.
- Hackney J, Bade D, Clawson A. Relationship between forward head posture and diagnosed internal derangement of the temporomandibular joint. *J Orofac Pain* 1993; 7(4):386–90.
- Darlow LA, Pesco J, Greenberg MS. The relationship of posture to myofascial pain dysfunction syndrome. *J Am Dent Assoc* 1987; 114(1):73–5.
- Olivo SA, Bravo J, Magee DJ, Thie NM, Major PW, Flores-Mir C. The association between head and cervical posture and temporomandibular disorders: a systematic review. *J Orofac Pain* 2006; 20(1):9–23.
- Armijo Olivo S, Magee DJ, Parfitt M, Thie NM. The association between the cervical spine, the stomatognathic system, and craniofacial pain: a critical review. *J Orofac Pain* 2006; 20(4):271–87.
- Sessle BJ. Sensory and motor neurophysiology of the TMJ. In: Laskin DM, Greene CS, Hylander WL, editors. Temporomandibular disorders: an evidence-based approach to diagnosis and treatment. Chicago: Quintessence; 2006. p. 69–88.
- Tal M, Devor M. Anatomy and neurophysiology of orofacial pain. In: Sharav Y, Benoliel R, editors. Orofacial pain and headache. Edinburgh: Elsevier; 2008. p. 19–44.
- Okeson JP. Etiology of functional disturbances in the masticatory system. In: Management of temporomandibular disorders and occlusion. St. Louis: Mosby; 2008. p. 130–63.
- McNamara JA Jr, Seligman DA, Okeson JP. Occlusion, orthodontic treatment, and temporomandibular disorders: a review. *J Orofac Pain* 1995; 9(1):73–90.
- Koh H, Robinson PG. Occlusal adjustment for treating and preventing temporomandibular joint disorders. *Cochrane Database Syst Rev* 2003; (1):CD003812.
- Seligman DA, Pullinger AG. The role of functional occlusal relationships in temporomandibular disorders: a review. *J Craniomandib Disord* 1991; 5(4):265–79.
- Gesch D, Bernhardt O, Kirbschus A. Association of malocclusion and functional occlusion with temporomandibular disorders (TMD) in adults: a systematic review of population-based studies. *Quintessence Int* 2004; 35(3):211–21.
- Laskin DM. Etiology of the pain-dysfunction syndrome. *J Am Dent Assoc* 1969; 79(1):147–53.
- Schwartz L. Conclusions of the temporomandibular joint clinic at Columbia. *J Periodontol* 1958; 29:210–2.
- Moulton RE. Emotional factors in non-organic temporomandibular joint pain. *Dent Clin North Am* 1966; Nov: 609–20.
- Engel GL. The need for a new medical model: a challenge for biomedicine. *Science* 1977; 196(4286):129–36.
- Stohler CS, Zarb GA. On the management of temporomandibular disorders: a plea for a low-tech, high-prudence therapeutic approach. *J Orofac Pain* 1999; 13(4):255–61.
- Lötsch J, Geisslinger G. Current evidence for a modulation of nociception by human genetic polymorphisms. *Pain* 2007; 132(1–2):18–22.
- Diatchenko L, Nackley AG, Tchivileva IE, Shabalina SA, Maixner W. Genetic architecture of human pain perception. *Trends Genet* 2007; 23(12):605–13.
- Diatchenko L, Nackley AG, Slade GD, Bhalang K, Belfer I, Max MB, and others. Catechol-O-methyltransferase gene polymorphisms are associated with multiple pain-evoking stimuli. *Pain* 2006; 125(3):216–24.
- Nackley AG, Shabalina SA, Tchivileva IE, Satterfield K, Korchynskiy O, Makarov SS, and others. Human catechol-O-methyltransferase haplotypes modulate protein expression by altering mRNA secondary structure. *Science* 2006; 314(5807):1930–3.
- Diatchenko L, Slade GD, Nackley AG, Bhalang K, Sigurdsson A, Belfer I, and others. Genetic basis for individual variations in pain perception and the development of a chronic pain condition. *Hum Mol Genet* 2005; 14(1):135–43.
- Diatchenko L, Anderson AD, Slade GD, Fillingim RB, Shabalina SA, Higgins TJ, and others. 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(5):449–62.
- Kim H, Mittal DP, Iadarola MJ, Dionne RA. Genetic predictors for acute experimental cold and heat pain sensitivity in humans. *J Med Genet* 2006; 43(8):e40.
- Kopp S. Neuroendocrine, immune, and local responses related to temporomandibular disorders. *J Orofac Pain* 2001; 15(1):9–28.
- Uceyler N, Valenza R, Stock M, Schedel R, Sprotte G, Sommer C. Reduced levels of antiinflammatory cytokines in patients with chronic widespread pain. *Arthritis Rheum* 2006; 54(8):2656–64.
- Sessle BJ. The neural basis of temporomandibular joint and masticatory muscle pain. *J Orofac Pain* 1999; 13(4):238–45.
- Svensson P, Graven-Nielsen T. Craniofacial muscle pain: review of mechanisms and clinical manifestations. *J Orofac Pain* 2001; 15(2):117–45.
- Grossi ML, Goldberg MB, Locker D, Tenenbaum HC. Reduced neuro-psychologic measures as predictors of treatment outcome in patients with temporomandibular disorders. *J Orofac Pain* 2001; 15(4):329–39.