

SCIENTIFIC ARTICLE

Occlusal Dysesthesia and Temporomandibular Disorders: Is There a Link?

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Most dentists have experienced the occasional patient with a strong concern about how the teeth mesh. Often it is the patient who feels that the new crown or bridge that was just cemented is not comfortable or "the bite is off." There may be legitimate merit to the patient's complaint about the new restoration, but after the second or third time it is adjusted or replaced without successfully resolving the complaint, the merit of the complaint has to be questioned.

Such complaints can occur in isolation of any functional problem in the orofacial musculoskeletal system. However, the most common comorbid disorders are myofascial pain involving the masticatory muscles or temporomandibular joint (TMJ) inflam-

matory or arthritic disease. In this article, we speculate that these two diseases either alter the articulation of the mandible to the temporal bone (i.e., arthritis) or alter the muscle's proprioceptive information being fed to the brain and produce an aberrant sense of jaw position (i.e., myofascial pain) and a lack of bite comfort.

To illustrate how often these complaints are comorbid, Figure 1 presents the frequency of positive responses taken from a TMJ pain and dysfunction questionnaire (0-4 scale) by Gerstner and colleagues.¹ One particular item on the 13-item questionnaire asked, "Do you have a problem with your bite being uncomfortable?" These data were taken from 127



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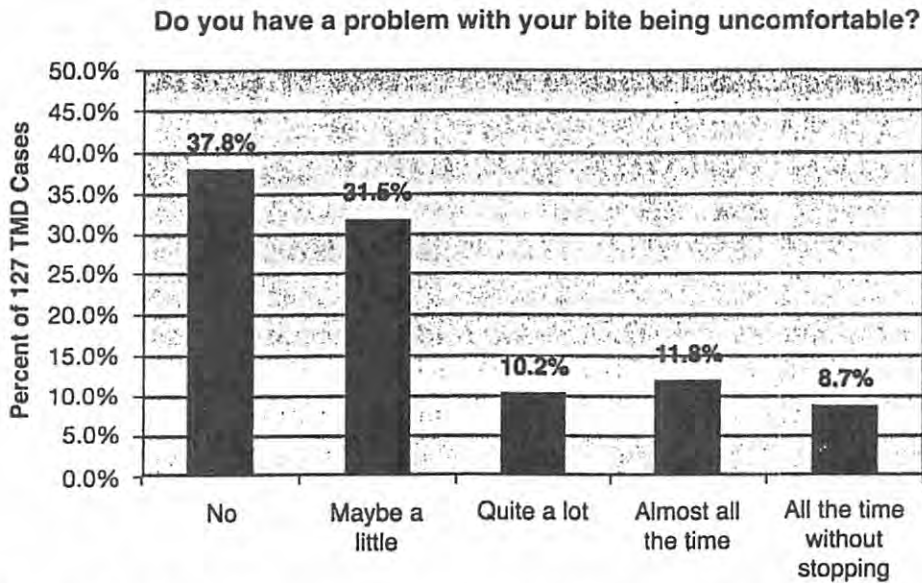


Figure 1. This figure shows the response to a question on bite comfort taken from a group of 127 consecutive clinic patients with temporomandibular disorder (TMD).

consecutive patients attending a university temporomandibular disorder (TMD) clinic for diagnosis and help with their jaw pain and dysfunction problems. The results showed that nearly 30% of the respondents scored 2 or higher on this question.

The three most common complaints by patients with a persistent uncomfortable bite are "my bite is not comfortable," "my bite is off" and "I don't know where my teeth belong anymore." When such complaints surface, it is necessary to distinguish between those patients with true bite discrepancies and those with no observable abnormality of the occlusion. When an observable bite discrepancy is discovered, it may have developed spontaneously, but more often than not it is found to have developed after an alteration in the occlusion occurring as a routine part of dental treatment. Occlusal contact changes can also develop in cases in which the TMJ is altered by either arthrotic destruction, synovitis or diskal displacement, although the latter joint problem is less likely to produce an obvious change in the occlusion than the other conditions. Regardless of the cause, the conscientious dentist will examine the patient for evidence of an improper jaw relationship or the suspected tooth or teeth for signs of occlusal trauma or dentoalveolar or TMJ pathology. Sometimes clear and demonstrable

abnormality of the teeth, TMJ or maxillomandibular relationship is present, and an immediate plan of correction can be made.

Another scenario to consider when assessing the occlusion for abnormality is that the observed occlusal abnormality might be a transient. Such cases are called an "acute malocclusion," and, in these cases, a well-intentioned dentist might perform a bite adjustment on a patient with an acute malocclusion that is caused by sustained lateral pterygoid contraction or TMJ synovitis in an attempt to correct the bite discrepancy. Unfortunately, the adjustment of an acute malocclusion may induce a true bite discrepancy when the sustained contraction or swelling subsides.

The final group of patients with an uncomfortable bite (and the focus of this article) are those patients without any observable occlusal anomaly or discrepancy. In these cases, the complaint of bite discomfort often develops spontaneously. When no obvious occlusal abnormality or dentoalveolar pathology is evident, the confusion begins, and there are more chances for misadventure. For example, a dentist may attempt to make a good occlusion even better by performing occlusal adjustment of the teeth or by replacing the new dental restoration in the suspected tooth or teeth, hoping that the problem will simply resolve

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with time. If this fails, increasingly more aggressive and often irreversible interventions may follow.

Unfortunately, these subsequent interventions may aggravate the patient's initial complaint. For example, in 1983, Remick and colleagues described 21 patients with atypical facial pain who had undergone 65 dental/surgical treatments.² The results of these treatments were that only one patient had less pain afterward. They suggested in their case series that frequently a psychological diagnosis was comorbid with the atypical facial pain, and patients with this diagnosis should receive conservative dental and medical treatment and a behavioral assessment before dental and surgical procedures are contemplated. Of course, these cases were characterized as pain cases, but the same basic observation for patients with persistent uncomfortable bite without an observable occlusal abnormality is equally applicable.

Prior Nomenclature for the Persistent Uncomfortable Bite without an Observable Occlusal Abnormality

Of course, if the patient's complaint can be clearly identified as being attributable to an observable bite discrepancy, then the correct terminology to describe the problem would be the phrase "persistent uncomfortable bite secondary to [fill in the etiologic agent]." However, when there is no observable bite discrepancy, the terminology used for this problem has varied over the years and includes several terms (Table 1).

The most common term understood by dentists is "occlusal neurosis," which appears to have been first cited by Boyens³ and Bodecker⁴ and is related to occlusal habit neuroses, a term coined by Tishler in

1928⁵ to describe bruxism or "bruxomania." Tishler speculated that occlusal interferences in combination with a neurotic individual would result in bruxism.

Ramfjord and Ash attributed the creation of an "occlusal neurotic" to poorly fitting teeth that were not corrected.⁶

Posselt used the term "positive occlusal sense" in his textbook and said that this problem was a contraindication to the performance of an occlusal adjustment.⁷ He wrote that when a patient whose masticatory system functions within the physiologic range of adaptation, their bite or occlusion will not come into the patient's consciousness. He referred to this as "negative occlusal sense." He continued by saying that after occlusal grinding or adjustment, some persons with a nervous predisposition may become too conscious of their own occlusion—hence they develop occlusal neurosis or positive occlusal sense.

Marbach introduced the terms "phantom bite" and "phantom bite syndrome" to the literature when he described a population of patients (equal male/female distribution) who presented with a self-perceived "need for bite correction," and he implied that this disorder had a strong psychological origin.⁸⁻¹¹ Marbach reported that patients with phantom bite generally have no apparent pathology on examination and most often no complaint of pain. He described them as having been evaluated by a multitude of dentists with many unsuccessful attempts at correction of the occlusion either with restoration or adjustment.

Although the chronic complaint of an "uncomfortable bite" is well known to dentists, the literature is surprisingly devoid of experimental research. More recently, Harris suggested the term "dysproprioception,"¹² and Harris and colleagues described patients with persistent uncomfortable occlusions.¹³

Occlusal Dysesthesia: What Is It?

In 1997, Clark and colleagues suggested the term "occlusal dysesthesia" (OD) as the best one to describe the complaint of a persistently uncomfortable bite when a bite discrepancy cannot be observed.¹⁴ Occlusion is defined in the dictionary as the act of closure or the state of being closed. The dental meaning of the term occlusion relates to the static intercuspal relationship of the teeth and also to

Table 1. Terms That Have Been Used to Describe a Persistent Uncomfortable Bite

Occlusal habit neurosis	Tishler, 1928 ⁵
Positive occlusal sense	Posselt, 1960
Occlusal neurosis	Ramfjord, 1961
Phantom bite syndrome	Marbach, 1978
Monosymptomatic hypochondriacal psychosis	Marbach, 1985
Dysproprioception (iatrogenic)	Harris, 1991
Persistent uncomfortable occlusion	Harris et al, 1993
Proprioceptive dysfunction	Greene and Gelb, 1994
Occlusal dysesthesia	Clark et al, 1997

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the act of closing the teeth together. Dysesthesia can be described as a disagreeable or impaired (abnormal) sensation, whether spontaneous or evoked.

Clark and colleagues suggested that when patients complain of an uncomfortable bite, it is possible that they are experiencing a kinesthetic abnormality, OD. Of course, this complaint can also be attributable to a true physical abnormality or "abnormal occlusal contact pattern." OD is therefore defined as a "persistent uncomfortable sense of maximum intercuspation after all pulpal, periodontal, muscle and TMJ pathologies have been ruled out and a physically obvious bite discrepancy can not be observed." Clark and colleagues proposed this as a more appropriate term than those used in the past because the core complaint of these patients is an altered occlusal sense producing a loss of comfortable position in full closure of the teeth.¹⁴ Persistent could describe a complaint continuing over a 6-month period.

Possible Explanatory Mechanism for OD

Two explanations of the patients diagnosed with OD (severe bite discomfort) are that they have either a seriously altered oral kinesthetic ability or a diagnosable psychiatric disorder. The kinesthetic ability of the jaw is defined as "the tested accuracy of subjects to discriminate the position of the mandible in the path from maximum opening to maximum intercuspation." At least one theory underlying this disorder is that there is a general loss of peripheral sensory receptor function in the jaw and teeth and, more specifically, the function of muscle spindles in the jaw closers are affected.

This theory is based on facts reported by Hellsing that distortion of kinesthesia was independent of the amplitude and frequency of vibration to confound muscle spindle sensory afferents and persisted during anesthesia of the TMJs and loading of the mandible.¹⁵ Hellsing also speculated that jaw muscle receptors may contribute to mandibular kinesthesia.

Morimoto and Kawamura also investigated mandibular kinesthesia and suggested that muscle spindles in jaw-closing muscles are mainly responsible for interdental thickness discrimination.¹⁶

Finally, van Duersen and colleagues showed that with aging and associated peripheral neuropathy, the

kinesthetic ability deteriorates.¹⁷ This group demonstrated that vibration had a greater effect on younger subjects, less effect on the elderly and even less effect on those with peripheral neuropathy. This diminishing effect was presumably owing to the fact that with aging and peripheral neuropathy, the muscle spindles become progressively dysfunctional and therefore less relied on by those subjects.

OD and Psychological Dysfunction

Usually, a psychological diagnosis is invoked only after all known physical explanations of pathology are exhausted. However, a psychologically based diagnosis is important to consider in most cases of persistent OD based on inclusion criteria.

As mentioned, Marbach implied that the phantom bite problem has a psychiatric etiology. Munroe referred to the term "monosymptomatic hypochondriacal psychosis," a psychotic disorder characterized by an unshakable belief in a distorted body image.¹⁸ Another example of this would include anorexia nervosa. Marbach suggested also that some subjects with phantom bite could instead have a neurotic disorder termed "dysmorphophobia." This is the belief that a body part is cosmetically defective in a person of normal appearance. Marbach also contended that patients with phantom bite will talk endlessly about the shape, color and size of crowns, and their standard complaints are the "bite is off" and the "teeth don't look right."⁸⁻¹¹ He does not support his theory with psychometric testing but rather with clinical observation.

In contrast, Greene and Gelb published a brief report on five subjects with what they term proprioceptive dysfunction and a range in duration of symptoms between 44 and 264 months.¹⁹ They found only one subject with a somatoform disorder, and the other four "did not qualify for a psychopathological diagnosis, and certainly were not delusional or psychotic." However, the actual data used to reach these conclusions were not provided in their short report.

Given this unresolved controversy, all OD cases warrant consideration for psychometric testing and psychological evaluation. The primary psychological disorder to be ruled out is somatoform disorder. This diagnostic condition is also the most frequent diagnosis associated with a subjective sensory abnormal-

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ity unconfirmed by objective signs and tests. A psychiatric diagnosis, *per se*, does not do much to help us understand the neuropsychological process involved. Actually, cognitive theory does a better job of explaining this process. According to cognitive theory, preexisting expectations and beliefs about appearance and somatic sensations could be connected to certain emotions (fears) that something is wrong. Such a mind-set then affects the patient's perceptions of their somatic condition.

Sensory-Kinesthetic Testing of the Jaw and Teeth

In addition to psychological assessments, it is also prudent to conduct somatosensory and somatomotor testing on patients to assess whether a true kinesthetic disturbance exists. Unfortunately, no commercial instrument or device has ever been produced to measure the sensory discrimination thresholds for individual teeth and the kinesthetic performance of the mandible itself.

Several testing procedures have been described in the literature. For example, occlusal position comfort is determined by three peripheral neural inputs: periodontal ligament mechanoreceptors of the teeth, TMJ mechanoreceptors and muscle spindle receptors in the jaw muscles.

The research on the sensory perceptive and discriminative abilities of subjects can be separated into two domains: the kinesthetic sense of the mandible and the tactile abilities of the teeth. Kinesthetic knowledge of where the jaw is at all times is important because we do not have visual feedback of our jaw's position, and the absence of this information would result in many more self-inflicted tooth, cheek and lip bites. As mentioned, the mandible receives sensory input from changes in muscle spindle lengths, mechanoreceptors of the TMJs and periodontal mechanoreceptors during the bolus-crushing phase of chewing.

The idea that jaw muscle spindles may be altered has been suggested by several scientists over the years. Clark and colleagues examined a group of patients with myofascial pain dysfunction and a control group with regard to their thickness discrimination abilities.²⁰ The patient population had primarily muscle tenderness but no specific TMJ abnormality, and the test involved giving the patients different size blocks and asking them if the current block was thinner or thicker than the last block. The patients were found to have diminished interdental thickness discrimination (ITD) compared with controls (Figure 2). The figure shows that within a range of ± 1.0 mm, the error rate for guess if the test block was thicker or thinner than

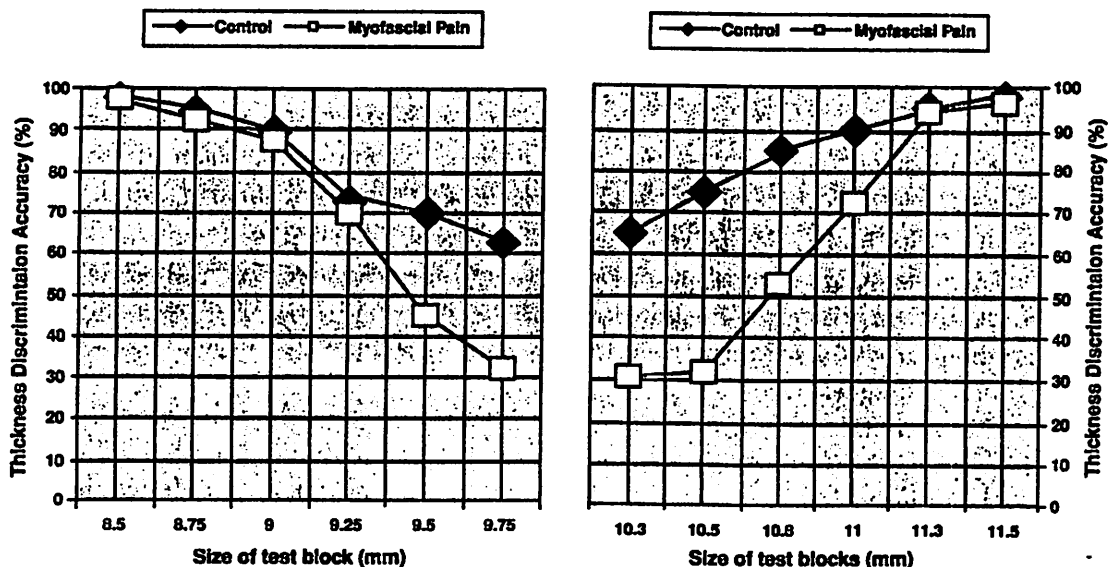


Figure 2. This figure shows the results of data on the accuracy of discriminating blocks of different thicknesses between the central incisors. Subjects were given a standard thickness block between each test block of 10 mm. The data are from a group of patients with myofascial pain in the jaw and a comparison group of matched control subjects.

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the reference block was much higher in the subjects with myofascial pain.

Morimoto and Kawamura tested the thickness discrimination (using the block method) with and without tendon vibratory stimulation applied to the jaw.¹⁶ This type of stimulation adversely affects the ability of the muscle spindles to accurately discriminate changes in muscle length. The fact that the ITD was worse as a result of the vibration also suggests the strong role that the muscle spindles play in jaw position monitoring.

The importance of periodontal receptor input to mandibular kinesthesia was shown to be low based on an experiment by Christensen and Morimoto.²¹ They reported that the dimension discrimination at two different degrees of mouth opening was mostly unaffected after local anesthesia was applied to the periodontal ligaments in normal subjects. Because of this, they concluded that muscle receptors were primarily responsible for interdental thickness discrimination.

Of course, other receptors in addition to muscle spindles contribute to the sum of data used by the brain to determine where the jaw is positioned in space. In the absence of periodontal feedback, the TMJ receptors, and probably the mucosal mechanoreceptors in the oral cavity, contribute to determining the position of the jaw in space.

In 1961, Thilander studied the repeatability of a jaw position in space (no tooth contact) with and without anesthesia applied to the TMJ.²² She found that normal young adults were able to reproduce an arbitrary position in space between rest and maximum opening ± 3.2 mm of the original position. If one TMJ was anesthetized, the range of this error increased to 9.4 mm. With both TMJs anesthetized, the mean was 9.3 mm. It must be noted, however, that the role of the TMJ as a major contributor to thickness discrimination was discounted by work done by Caffesse and colleagues, who found that in subjects with good discriminatory ability, local anesthesia of the TMJ capsules had little to no effect on thickness threshold discrimination.²³

It would be logical to conclude that muscle afferents appear to be primary contributors to the sense of thickness discrimination between the teeth. Muscle afferents are most likely activated only when very wide opening occurs (when there would be substan-

tial stretching of the tendons) and when a slight activation of the muscle occurs in association with touching or biting on an object. The link between the periodontal mechanoreceptor and the muscle afferents is shown by the fact that both provide sensory input directly to the mesencephalic nucleus and possibly to the cerebellum.

In spite of the research on the kinesthetic sense of the mandible and the tactile abilities of the teeth, to date, there are no specific reports in the literature that have proposed the use of these types of tests for detection of abnormalities of the occlusion, although this area appears to be a logical and potentially fruitful area for future research endeavors. No specific test(s) has yet been described to differentiate the patient with OD from the normal patient. Although there appears to be a trend toward psychological issues in these patients, it is likely that physical objective findings also explain some of the symptoms.

Conclusion and Approach to Management

The answer to the question, "Are TMD problems and an uncomfortable bite linked?" is sometimes yes and sometimes no. Certainly, OD can develop independently of obvious myofascial pain and TMJ arthritis problems, but the normal kinesthetic skills and abilities of patients can also be substantially diminished by arthritic disease and, certainly, chronic muscle pain. When these two problems (OD and TMD) are comorbid, the logical approach is to treat the TMD problem first and then see what degree of change has occurred in the bite comfort complaint. If a substantial suspicion exists that a somatoform disorder exists, obtain a psychiatric consultation.

Regarding treatment, the bad news is that we have no good direct treatment for idiopathic OD. The best form of symptomatic management for those cases in which the complaint is localized to a single tooth or teeth in one region of the mouth is to make the patient an occlusal disengagement device (e.g., an anterior bite plane). Such a device allows the patient to more or less avoid contact on the offending posterior teeth. In such cases, if the disengagement splint is helpful, it is logical to slowly reduce the thickness of the occlusal splint over several weeks to months until the posterior teeth come back into contact.

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No specific medication has been found to be helpful other than a partially sedating medication (e.g., clonazepam), which would more or less reduce anxiety and increase the tolerance to the symptoms.

The final critical component of treating any patient with OD is education. Specifically, it is necessary to fully explain that the patient's complaint of a persistent uncomfortable bite often is not a physical problem but a mechanosensory disorder and may best be characterized as the outward consequence or a pathologic alteration of the mandibular proprioceptive system. In these cases, mechanical therapies (e.g., occlusal adjustment or restoration replacement) are not helpful, but it is hoped that with time and symptomatic management, the severity of the problem will fade to a level of tolerance.

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