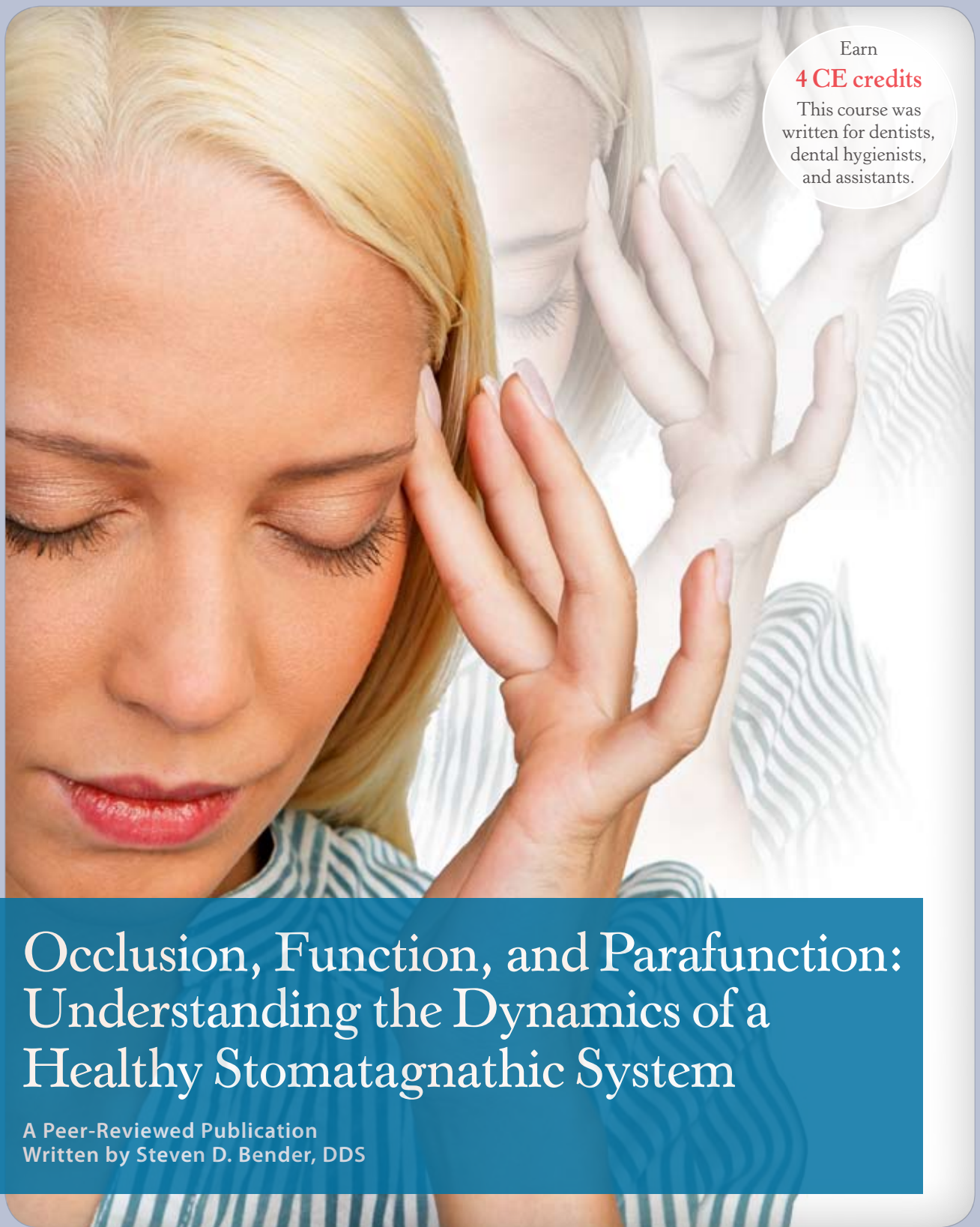

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Occlusion, Function, and Parafunction: Understanding the Dynamics of a Healthy Stomatagnathic System

A Peer-Reviewed Publication
Written by Steven D. Bender, DDS

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Educational Objectives

Upon completion of this course, the clinician will be able to do the following:

1. Define parafunction and the activities associated with this
2. Identify the signs and symptoms of parafunctional activity
3. Know the considerations and steps involved in diagnosing parafunctional activity
4. Identify the types of appliances that can be used to manage parafunction, their advantages and disadvantages, and considerations in selecting an appliance for individual patients

Abstract

Parafunctional activities associated with the stomatagnathic system include lip and cheek chewing, fingernail biting, and teeth clenching. Bruxism can be classified as awake or sleep bruxism. Patients with sleep bruxism are three to four times more likely to experience jaw pain and limitation of movement than people who do not experience sleep bruxism. Signs and symptoms of bruxism and parafunctional activity include hypertrophied masseter and temporalis muscles, myocitis of these same muscles, morning jaw stiffness, and sensitivity in a tooth or teeth. Additionally, migraine is associated with parafunctional activity. Dental professionals are responsible for diagnosing and managing parafunction. Patients can be managed with oral appliances of various designs.

Introduction

Understanding the relationship between how the upper and lower teeth meet in function, along with the orthopedic condition of the temporomandibular joints, is critical to successful dental therapy. Occlusion has been defined as "any contact between biting or chewing surfaces of maxillary (upper) and mandibular (lower) teeth."[1] Okeson describes occlusion as the "static relationship of teeth … basic to all aspects of dentistry."[2] According to the Glossary of Prosthodontic Terms, occlusion is "the act or process of closure or of being closed. It is the static relationship between the incising or masticating surfaces of the maxillary or mandibular teeth or tooth."[3] The functions of chewing, swallowing, and speaking are largely dependent on the manner in which the upper and lower teeth come together. As dentists, we are presented daily with the opportunity to affect these relationships.

Parafunction

Parafunction in dentistry refers to those activities of the stomatagnathic system that would be considered to fall outside of functional activities. Lip and cheek chewing, fingernail biting, and teeth clenching are examples of parafunctional activity. Much focus in dentistry has been given to teeth grinding, or bruxism, as a parafunctional activity. Bruxism can be classified as awake or sleep bruxism. Awake bruxism involves tooth clenching or tapping and jaw bracing, with or without tooth contact. Grinding is rarely noted during waking hours. Sleep bruxism involves tooth grinding with phasic (rhythmic), tonic (sustained), or mixed (both types) jaw muscle contractions.[4] The overall prevalence of awake clenching is about 20 percent in the adult population, with more women reporting clenching awareness than men.[5,6]

Since it is probable that sleep bruxism differs in terms of etiology from daytime parafunctional jaw muscle activity, it should be distinguished from teeth clenching, bracing, or grinding while awake.[7,8] It has been estimated that 8 percent of adults in the general population are aware of teeth grinding during sleep, usually as reported by their sleep partners or roommates.[9] According to parental reports, the incidence of teeth grinding noises during sleep in children younger than 11 years of age is between 14 and 20 percent.[10,11] Dental signs of bruxism can be seen in approximately 10 to 20 percent of children.[12] Studies have shown that approximately 60 percent of "normal" sleepers exhibit rhythmic masticatory muscle activity (RMMA) during sleep. RMMA is defined as three masseter muscle bursts or contractions within an episode in the absence of teeth grinding.[13] Bruxism episodes tend to occur primarily in light sleep (stages 1 and 2). In pediatric bruxers, 5–10 percent of episodes will occur during rapid eye movement (REM) sleep. An estimated 15–20 percent of sleep bruxism episodes are reported to occur during the transition from deep sleep (stages 3 and 4) to REM sleep.[14,15]

Table 1. Tooth grinding and bruxism statistics

Awake clenching	20% of adult population
Aware of grinding during sleep	8% of adults
Noise from tooth grinding during sleep	14–20% of children under age 11
Dental signs of bruxism in children	10–20%
Rhythmic masticatory muscle activity during sleep	60% of 'normal' sleepers

Etiology and Pathophysiology

The reported causes of sleep bruxism have ranged from stress and anxiety disorders to tooth relationship discrepancies. In the 1960s, Ramfjord utilized physiologic electromyogram (EMG) recordings of awake subjects to suggest that occlusal interferences were responsible for teeth grinding.[16] More recent research has demonstrated that the notion of peripheral causes for sleep bruxism is doubtful.[17,18,19,20,21,22] In a 24-hour period, it is estimated that tooth contact occurs for 17.5 minutes.[23] During a 7- to 8-hour sleep cycle, bruxism-related muscle activity occurs for approximately eight minutes and does not always occur with tooth contact. Grinding noises are reported to occur in approximately 44 percent of sleep bruxism/RMMA events.[24,25] It appears that sleep bruxism is probably a manifestation of a normal, naturally occurring event during sleep. Teeth grinding has been demonstrated to occur subsequent to a sequence of physiological events that consists of an increase in sympathetic nervous system activity; cortex activation; heart rate increase; and, ultimately, an increase in jaw depressor muscle activity.[26] 60–80 percent of the time, sleep bruxism episodes tend to occur along with cyclic alternating patterns (CAPs).[27] CAPs consist of activation of electroencephalogram (EEG) and electrocardiogram (EKG or ECG) patterns and occur approximately every 20–60 seconds during non-REM sleep. These events are thought to be physiologic events that support sleep quality. Sleep bruxism episodes will also occur in clusters (within 100 seconds), adding support to the association between CAPs and sleep bruxism.

Signs, Symptoms, and Sequelae

Patients with sleep bruxism are three to four times more likely to experience jaw pain and limitation of movement than people who do not experience sleep bruxism.[28,29] Up to 65 percent of sleep bruxism patients report frequent headaches.[30] The sustained contraction of the masseter muscles seen with sleep bruxism can affect parotid salivary flow. This is known as parotid-masseter syndrome,[31] and reportedly mimics the sensation felt when biting into a tart or sour food. Usually the patient will report pain with the first bite of food, which then dissipates as the meal progresses. Some studies have reported the average sleep bruxing event to generate up to 60 percent of the force generated during voluntary maximum clenching prior to sleep.[32] The greatest maximum voluntary clenching force has been measured to be 975 pounds per square inch.[33] It is of little wonder that we witness tooth destruction in the parafunctioning population. Sensitive and sore teeth, abfractions, fractures, and unexplained tooth mobility can be apparent in patients with parafunctional occlusion. Sleep bruxism may also be responsible for stomatagnathic muscle nociceptive signaling into the trigeminal nervous system. Sleep clenching/grinding episodes may last as long as 20–40 seconds.[34] Muscle pain has been elicited in individuals who voluntarily clenched for 20–30 seconds.[35] Inflammatory mechanisms have been shown to be involved in temporomandibular joint pain and dysfunction.[36] Milam proposed a possible etiology for inflammatory mechanism of the temporomandibular joint structures via a hypoxic-reperfusion injury model. This injury may occur when the capsular pressure of the temporomandibular joint exceeds the end-capillary perfusion blood pressure of the feeding vasculature.[37] The tissues then undergo reperfusion via mouth opening or relaxation of the elevator muscles. Capsular nociceptive fibers triggered by pathologic loading of the highly innervated synovial tissues may also stimulate the release of the nociceptive transmitters, calcitonin gene-related peptides (CGRP) and Substance P, leading to further inflammatory processes. Not only do the inflammatory mediators contribute to the joint degenerative process, they also increase nociceptive excitation at the second-order neurons of the trigeminal vascular system – the system thought to be responsible for headache disorders. Patients are usually unaware of sleep bruxism activity and will often deny its occurrence. Many sleep bruxism subjects will present with indentations or scalloping of the lateral tongue borders.[38] These patients may also display ridging of the buccal mucosa,[39] as well as exostosis present in the mandible and maxilla.

Figure 1. Wear and abfractions

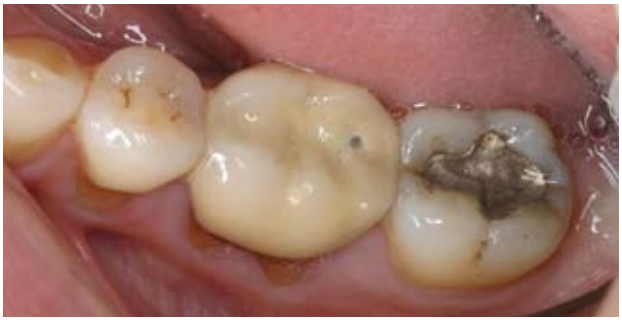


Figure 2. Scalloping of the lateral border of the tongue

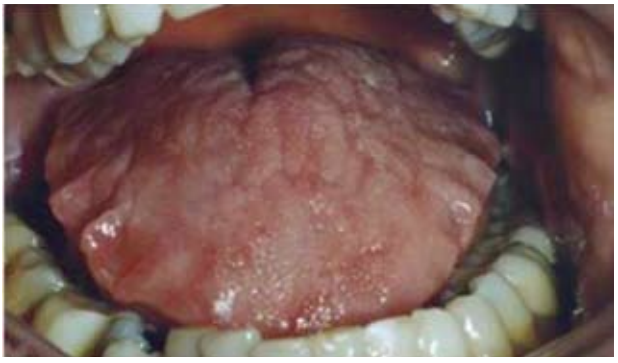


Figure 3. Ridging of the buccal mucosa

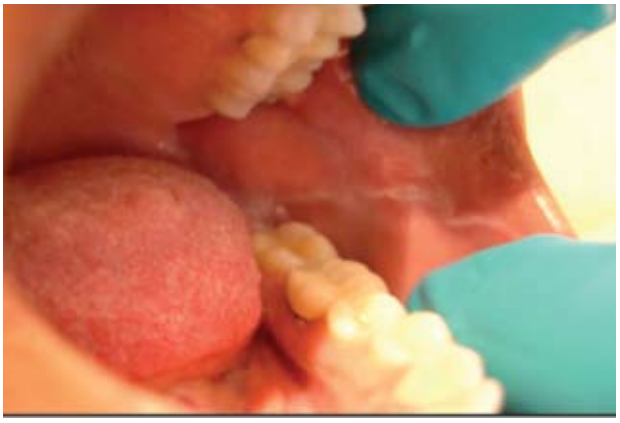
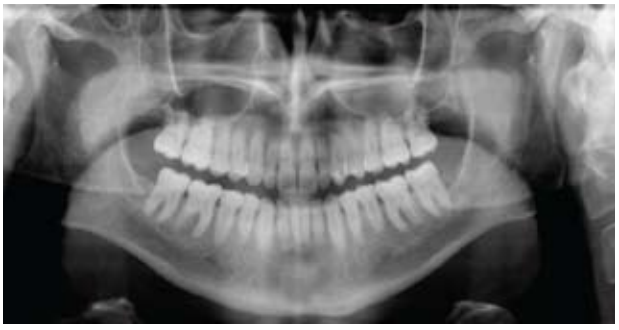


Figure 4. Radiographic changes



Flattening of the condyle and enlargement of the posterior mandibular ramus angle

Other indicators will include hypertrophied masseter and temporalis muscles, myocitis of these same muscles, radiographic changes of the mandibular condyles and articular fossas, and the appearance of intrusion of the posterior dentition. Tooth wear is traditionally thought to be a diagnostic presentation of sleep parafunction, but can often prove to be an unreliable indicator. Many sleep bruxism patients will complain of morning jaw stiffness but may also report a worsening of symptoms as the day progresses. Tooth or teeth sensitivity can also be an indicator of sleep bruxism. The physical damage and pain of parafunctional occlusion are related to the intensity, frequency, and duration of the parafunctional activity and the position of the stomatagnathic system.

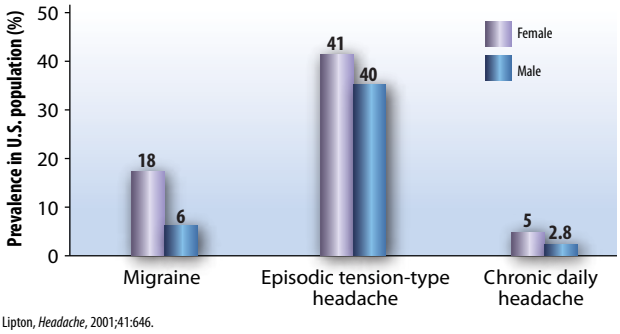
Table 2. Signs and symptoms of parafunction

Tooth wear
Tooth sensitivity or pain
Abfractions
Tooth mobility
Indentations of lateral border of tongue
Ridging of buccal mucosa
Maxillary and mandibular exostoses
Jaw pain or stiffness
Parotid-masseter syndrome
Radiographic changes of the condylar region
Hypertrophy and/or myocitis of muscles (masseter and temporalis)
Headaches
Neck pain
Back pain

The Migraine Connection

According to Silberstein, migraine is a chronic neurologic condition with intermittent, episodic attacks.[40] There appears to be a strong genetic component (up to 50 percent), with women being afflicted more than men.[41,42,43] Two major subtypes of migraine exist: with aura (classical) and without aura (common).[44] The prevalence of migraine in the U.S. general population has been estimated to be 12 percent, with 18 percent of women and 6 percent of men affected.[45,46]

Figure 5. Prevalence of primary headaches



Lipton, *Headache*, 2001;41:646.
Schwartz. *JAMA*, 1998;279:381.

Migraine is now considered one of the 20 most disabling diseases according to the World Health Organization.[47] Its characteristics vary among patients and often among attacks in a single patient.

Diagnosis of Parafunction and Migraine

A diagnosis of parafunction can be made after reviewing the patient's signs and symptoms. Symptoms may include generalized nonspecific jaw pain, masseter muscle stiffness, neck pain, back pain, headaches, dental pain, and tooth sensitivity. Signs can include abnormal tooth wear, abfractions, tongue scalloping, and exostoses. It is important to perform a thorough examination, taking all signs and symptoms into account before determining a diagnosis of parafunction. In the case of migraines, it is essential to consider both primary headaches (due to migraine, episodic stress, and chronic daily headaches that may or may not be associated with vision problems) and secondary headaches due to other underlying causes (in the extreme, as a result of an intracranial pathology). Imaging should be avoided if it will not lead to a change in the management strategy. Exceptions should be considered when a patient is exceptionally fearful of serious pathology. Red flags signaling the need for imaging studies would include systemic symptoms such as fever or weight loss; neurologic test abnormalities; sudden, abrupt onset; new onset headache in a patient >50 years of age; and a change from a previous headache history.

A diagnosis of migraine can be made based on the following criteria:

A. At least five attacks fulfilling criteria B through D
B. Headache attacks: 4 to 72 hours
C. Headache: at least two of the following characteristics
   - Unilateral
   - Pulsating
   - Moderate or severe pain intensity
   - Aggravation by or causing avoidance of routine physical activity
D. During headache, at least one of the following
   - Nausea and/or vomiting
   - Photophobia and phonophobia
E. Not attributed to another disorder[48]

The five phases of a migraine attack include the premonitory phase, commonly referred to as a prodrome; the aura (occurring in approximately 10–25 percent of migraine patients); the headache phase (lasting 4 to 72 hours); the resolution; and the postdrome. All five phases do not necessarily occur, and there is no distinct onset or end of each stage, with the possible exception of the aura. It is generally accepted that the development of migraine headache pain depends on the activation of sensory afferent fibers of the ophthalmic division (V1) of the trigeminal nerve. Experimental evidence suggests that the cerebral cortex of patients with migraine is more easily excitable than in non-migraneures.[49,50] A cortical spreading depression (CSD) is thought to lead to the activation of the trigeminovascular afferents. CSD is a slowly propagating (2–6 mm/min) wave of sustained neuronal depolarization creating a transient spike in activity as it progresses into the tissue, followed by a long-lasting neural suppression. The depolarization phase is associated with an increase in regional cerebral blood flow. Neurotransmitters such as K^+ ions, H^+ ions, NO, and arachidonic acid are released during CSD and appear to be responsible for activation of the perivascular trigeminal terminals in the meninges. This activity appears to cause activation of the caudal portion of the trigeminal nucleus caudalis (TNC) in the brainstem. The perception of pain is mediated by higher-order projections from the TNC.

This evidence disputes the once popular "vascular theory" of migraine. The exact mechanisms involved in CSD and subsequent activation of the trigeminal ganglion vascular system (TGVS) remain unclear. The activation of the TGVS leads to neuropeptide release resulting in neurogenic inflammation in the dura. The sustained firing of the sensitized meningeal nociceptors leads to the activation and sensitization of second-order central trigeminovascular neurons, resulting in what has been termed central sensitization. This central sensitization appears to be responsible for the prolonged duration of the migraine attack.[51] Thalakoti et al. discussed how activation of any of the three branches of the trigeminal nociceptive system has the potential to initiate a migraine event in the susceptible individual.[52] Bender further elucidated the putative role of facial pain in migraine pathophysiology, and the need for all practitioners to involve the stomatagnathic system in the headache patient examination process.[53]

Management Strategies

Currently, there is no evidence-based, curative treatment for sleep bruxism. The dentist's role is to diagnose the parafunction (sleep bruxism) and manage it, with the primary goals of preventing damage to the orofacial structures and reducing pain complaints. In addition to signs and symptoms assessed during a diagnosis, oral appliances may be used as diagnostic tools. In this situation, the appliance is worn by the patient to determine its effect on his or her presenting complaints. If the appliance helps, this can lead to a differential diagnosis of parafunction. Therapeutic options include palliative strategies to manage pain. These strategies could include pharmacologic therapies, behavior modification, physical therapy, counseling, and use of intraoral appliances. This discussion will focus on oral appliances; other modalities are beyond the scope of this article.

Oral Appliances

An oral appliance, covering a full or partial dental arch, has been the traditional option for patients who are frequent and severe grinders or clenchers.[54] The first report of oral appliance use was by Karolyi in 1901.[55] To date, little is known as to the mechanism by which oral appliances produce their therapeutic effects. Oral appliances can protect the orofacial structures from damage caused by bruxism. They have also been shown to decrease stomatagnathic muscle activity, as measured by EMG recordings.[56] There are basically two different materials utilized in the fabrication of intraoral appliances: hard appliances in which acrylic resins are either chemically cured or heat and pressure processed, forming a rigid appliance on the internal and external aspects, and, alternatively, soft or semi-soft appliances made from plastics or polymers resulting in an appliance that is somewhat flexible and pliable throughout.[57] A soft mouth guard is usually only recommended for use on a short-term basis. Studies have shown that soft appliances may increase bruxism in some individuals.[58,59,60]

Figure 6. Full coverage soft appliance

In one crossover, controlled, polysomnagraphic study comparing full maxillary tooth coverage appliances with a palatal splint without tooth coverage, a 40–50 percent reduction in the number of sleep bruxism/RMMA teeth grinding episodes was observed.[61] One study, however, found that maxillary oral appliances may aggravate breathing disorders.[62]

Mandibular appliances may be full arch coverage or partial arch coverage – either of the posterior or of the anterior teeth. Full arch coverage appliances are designed to spread the load across the teeth more evenly and require more bulk in the posterior area to prevent fracturing of the material during clenching or grinding with molar teeth.

Figure 7. Full coverage hard appliance

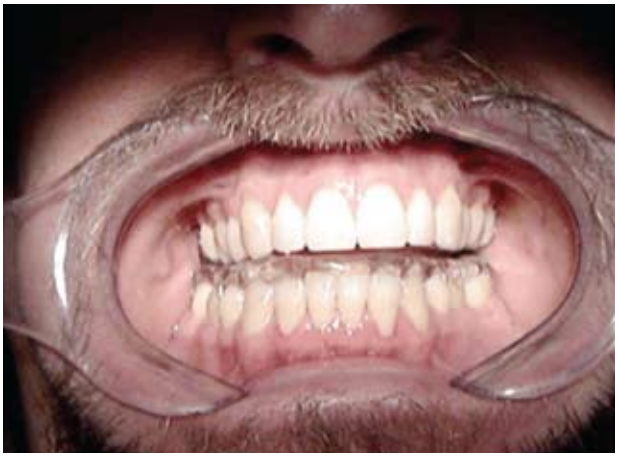


Figure 8. Partial posterior coverage custom hard appliance

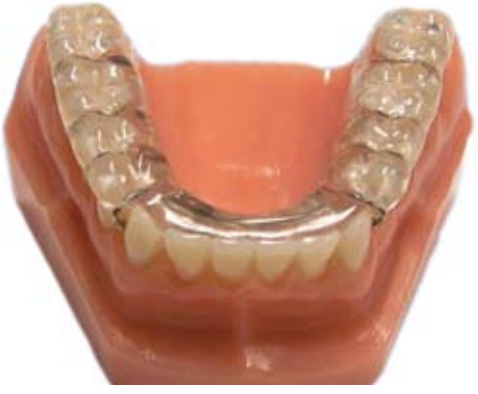


Figure 9. Partial posterior coverage soft OTC appliance

Figure 10. NTI-tss device

According to Hattori et al., clenching on a full arch orthotic does not significantly reduce the degree of muscle contraction as compared to clenching of the natural dentition.[63] Studies by Baad-Hansen et al., as well as Becker et al., have demonstrated a decrease in the EMG activity of the elevator muscle group when clenching on an anterior-contact-only appliance.[64,65] Other studies have demonstrated an increase in bite force with the use of appliances that allow posterior contact.[66,67] The rationale, then, for the anterior device would appear to be consistent with the observed physiology.[68]

Anterior Coverage Design

A number of anterior coverage designs are available, including appliances that cover all the anterior teeth and appliances designed to meet and cover only the central incisors. The concept of an oral appliance that provides contact on only anterior teeth was first introduced in the mid 1900s as the Lucia jig. Since that time, the idea has taken on different designs. In July 1998, the U.S. Food and Drug Administration (FDA) granted approval for an appliance known as the "NTI Clenching Suppression System" (now known as the "Nociceptive Trigeminal Inhibition Tension Suppression System" or NTI-tss). The NTI-tss provides anterior coverage of the incisors only. The manufacturer suggests that this device is indicated for the treatment and prevention of medically diagnosed migraine pain, tension-type headache, and temporomandibular joint disorders.[69] For the majority of cases, it is designed to be worn only at night, and can be constructed using indirect or direct techniques. Utilizing a direct technique involves a prefabricated polycarbonate matrix, customized chair side using acrylic or a thermoplastic reline to precisely fit the patient's teeth. Adjustments of the occluding surface are made by the dentist to ensure that with jaw closure and during all mandibular excursive movements, tooth contact occurs only between the device and the incisal edges of the opposing central incisors. The NTI appears to be the first device to only allow for incisor contact on the occluding surface. The periodontal ligament and the periodontal bone contain a rich network of low-threshold mechanoreceptor afferents that provide information about tooth loads.[70] The density of these fibers surrounding the tooth increases the more anterior in the arch the tooth is.[71] Elevator muscle activity is decreased while clenching on an anterior–contact–only appliance compared to maximum voluntary clenching of the natural dentition and on posterior coverage oral appliances.[72]

One common assumption is that the use of this appliance may lead to an increase in intra-articular pressures as well as loading of the TMJ articular surfaces.[73,74,75,76] A frequently cited report by Nitzan points to her findings of an average 81.2 percent reduction in intra-articular pressures when an awake and anesthetized individual clenches on a uniformly adjusted oral appliance that allows for even contact of the opposing dentition in the posterior segments.[77] It is of significance that no other type of appliance was utilized in this study for comparison. Kuboki et al. utilized tomographic radiographs to measure TM joint space while clenching on two different types of oral appliances. A flat or stabilization appliance that allows for posterior tooth contact did not produce any changes in the joint space as observed radiographically. The second appliance, known as an anterior repositioning appliance due to its effect of holding the mandible in a slightly forward posture, caused a reduction in the anterior portion of the joint space. It could be interpreted then, that neither appliance tested produced an "unloading" effect of the articular structures.[78] May et al. demonstrated a decrease in condylar compression utilizing an anterior-contact-only type of intraoral appliance. While there has been concern that these smaller appliances can be aspirated and that this is more likely than with larger appliances,[79] cases of aspirated NTI-tss devices could not be identified in a recent systematic review.[80]

A second appliance, the 'B splint', utilizes the same principle with the central incisors as the only contacting teeth in an upper or lower appliance with coverage of more teeth in the arch. It is important that distalizing forces are not placed on the mandible by the contacting surfaces. It has been suggested that this appliance design also works to decrease elevator muscle activity during sleep parafunction.

Selecting and Monitoring Appliances

The selection of an appliance depends on clinical parameters, the anticipated therapy, clinician preference, and whether the appliance is required during waking hours or during sleep hours only. Anterior coverage appliances for sleep are generally less bulky than posterior or full-coverage appliances, reportedly reduce the intensity of muscular activity,[81] and can be easier to adjust. Partial coverage designs with posterior coverage are designed in part to reduce the bulk of the appliance in the anterior segment and relieve the load of parafunctional activity on the anterior teeth. Bulky appliances are uncomfortable

for patients, and are unsightly (a factor for day appliances), and therefore impact patient compliance. The less bulky and comfortable an appliance is, the more likely the patient is to comply with treatment recommendations.

Adverse events may occur when any oral appliance is used. The potential for tooth movement and occlusal changes would appear to be the main concerns when wearing an NTI appliance. Kinoshita et al. demonstrated that it took approximately eight days for an unopposed tooth to begin to extrude in the dental arch.[82] Miles et al. demonstrated that it requires approximately 8–14 hours of continuous force to cause tooth movement.[83] Trenouth reported that bruxism patients spend about 38.7 minutes with their teeth together during an 8-hour period.[84] One frequently cited study concerning tooth movement involved 30 subjects, 15 wearing an NTI appliance and 15 wearing what was termed a "stabilization" appliance.[85] In this report, one subject in the NTI group displayed a decrease of one millimeter of overbite in the anterior region as well as a reduction in the occluding pairs of teeth from 10 to 5. Okeson explains how the effects of muscular relaxation and condylar seating can affect how the teeth come together.[86] This potentially explains the results observed by Magnusson.[87] Irrespective of the type of appliance selected, it is important that regular monitoring be carried out to ensure that the appliance is comfortable and is functioning appropriately, and that no unwanted issues such as irregular tooth wear, tooth movement, or occlusal changes are occurring. Due to the potential for negative outcomes with any oral appliance usage, great care should be taken to only prescribe therapy for patients who are willing to engage in supervised observation and follow-up appointments.

Direct and Indirect Appliances

As previously stated, an oral appliance can be fabricated using a direct or indirect technique. While the direct technique is fast, typically involving one visit, and may be less expensive since a laboratory fee is not involved, the indirect technique offers the opportunity for greater precision and strength, true custom fabrication, reduced chair time, and potentially improved patient comfort. In addition, should the clinician wish, a lab consultation is available and can prove helpful during treatment planning and appliance selection. Appliances for bruxism using the indirect technique include the BiteSoft Anterior Splint. The case below demonstrates the use of an indirect technique and the key steps and measurements that must be performed for indirect fabrication of a device, in this case the NTI-tss appliance.

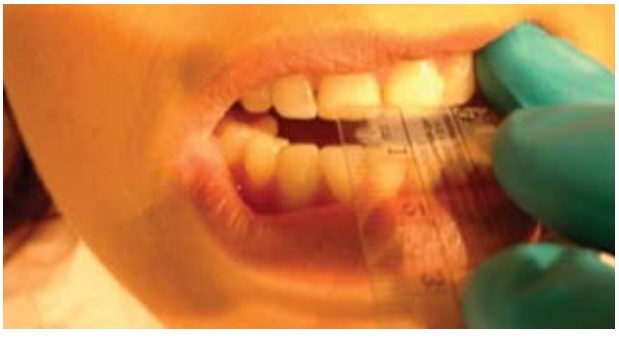
Treatment Planning and Indirect Appliance Design

Once a definitive diagnosis has been made and it has been determined that the patient would benefit from therapeutic intervention, it is key to first be assured that the patient will be compliant with the prescribed therapy and follow-up care required. Only then should an appliance design be selected and fabricated for the patient. To fabricate an indirect appliance, the laboratory must be provided with an impression, bite registration, and key measurements: the protrusive measurement and vertical measurement (leaf gauge). In addition, in the case of an NTI-tss appliance, the maximum protrusion bite must be provided. An impression of the mandibular arch should be taken using a suitable impression material, and a bite registration taken. It is essential to measure the minimum vertical height required that will ensure that the molars, bicuspids, and canines are discluded (i.e., do not meet or occlude) during lateral and protrusive movements once the appliance is seated in position. This can be assessed easily using a leaf gauge.

Figure 11. Use of a leaf gauge to determine vertical height requirements

The maximum protrusive measurement can be determined using a TMJ Triangle or other millimeter ruler to determine the maximum achievable distance between the edge of the maxillary incisors and the mandibular incisors while the mandible is fully protruded. (The same type of measurement can also be made with the patient in maximum retrusion.) Fabrication must ensure that the two central incisors bear the load equally, both to prevent untoward unilateral changes and to avoid tooth sensitivity. The maximum protrusive length is measured so that the device can be fabricated without permitting the maxillary central incisors to "fall off" the posterior aspect of the occluding table.

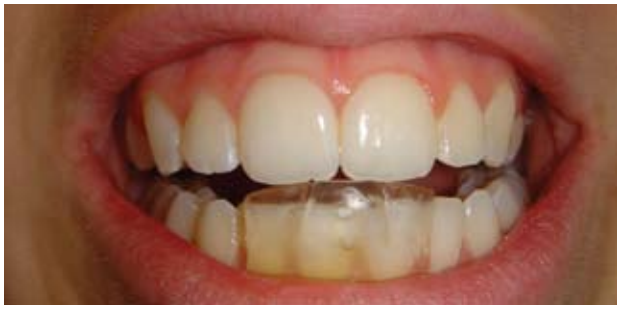
Figure 12. Protrusive measurement



At the delivery appointment, it is crucial to check that the appliance seats and fits properly, and is retentive, comfortable, and centered. A check should be performed to ensure that with all excursive movements the incisors remain in contact with the occluding table and that translation of the condyle in the fossa is minimized. There should be no canine or posterior contact in these excursions. Once these have been ascertained, the patient

should be given instructions on the wearing of the appliance, and cleaning and storage of the appliance.

Figure 13. Patient wearing completed appliance



Re-evaluation appointments should be scheduled to ensure that progress is appropriate and that the patient's symptoms are lessening or have disappeared. The patient should also be counseled as to realistic expectations and outcomes based on known scientific outcomes.

Summary

Parafunctional activity may result in various pain presentations and have deleterious effects on the dentition and other oral structures. It is important to identify signs and symptoms of parafunction and to investigate the patient's health complaints that may be related to it. A thorough examination must be performed to exclude secondary causes of the presenting symptoms associated with parafunction, such as intracranial pathology mimicking primary headaches. Oral appliances can be used as an aid in reaching a diagnosis of parafunction, as well as be utilized in the management and prevention of the dental sequelae associated with these disorders. Many types of appliances are available. Case-appropriate selection should consider ease of fabrication, ease of use, and comfort for the patient. Consideration should also be given to the most current and relevant scientific evidence available as well as the reported reliability to perform as intended. By providing patients with an appropriate oral appliance, compliance with therapy will be improved, ultimately resulting in an improved quality of life.

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Author Profile

Dr. Steven D. Bender received his Bachelor of Science degree in 1981 from Stephen F. Austin State University in Nacogdoches, Texas and earned his Doctorate of Dental Surgery from Baylor College of Dentistry in 1986. He practiced general and restorative dentistry in Plano, Texas until 2000. In 1999, he began studying in a mini-residency program at the Parker E. Mahan Facial Pain Center, University of Florida College of Dentistry where he received advanced training in orofacial pain and temporomandibular joint disorders. He also completed post-graduate dental education training at the L.D. Pankey Institute and the Dawson Center for Advanced Dental Study. In 2001 he received a certificate of completion in Orofacial Pain from the University of Medicine and Dentistry of New Jersey. He also completed a mini-residency program in Headaches, sponsored by Columbia University College of Physicians and Surgeons in 2003. Since 2001, Dr. Bender has maintained a private practice devoted to pain management of the head, face, oral cavity and TMJ. He has earned fellowship in the American Academy of Orofacial Pain and the International Academy of Oral Oncology. He serves as a reviewer for three journals: Headache, Cephalalgia, and The Journal of Musculoskeletal Pain and General Dentistry. He also currently serves as an evidence reviewer for the American Dental Association. Dr. Bender's current areas of research and study include difficult headaches, neuropathic pain mechanisms, and sleep disorders.

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Acknowledgment

Figure 13 is courtesy of Dr. Jim Boyd.

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Questions

1. The functions of chewing, swallowing, and speaking are largely dependent on the manner in which the upper and lower teeth come together.
   a. True
   b. False

2. Parafunction in dentistry refers to those activities of the stomatagnathic system that would be considered to _____.
   a. fall inside of functional activities
   b. fall outside of functional activities
   c. fall outside of parafunctional activities
   d. all of the above

3. Bruxism can be classified as _____ .
   a. the act of chewing aggressively
   b. comatic bruxism
   c. awake or sleep bruxism
   d. all of the above

4. The overall prevalence of awake clenching is about _____ in the adult population.
   a. 10 percent
   b. 15 percent
   c. 20 percent
   d. 25 percent

5. Dental signs of bruxism can be seen in approximately 10 to 20 percent of children.
   a. True
   b. False

6. During a 7- to 8-hour sleep cycle, bruxism-related muscle activity occurs for approximately ten minutes and always occurs with tooth contact.
   a. True
   b. False

7. Patients with sleep bruxism are_____ times more likely to experience jaw pain and limitation of movement than people who do not experience sleep bruxism.
   a. two to three
   b. three to four
   c. four to five
   d. none of the above

8. The sustained contraction of the masseter muscles that can affect parotid salivary flow is known as the _____.
   a. parotid-sternohyoid syndrome
   b. parotid-temporalis syndrome
   c. parotid-masseter syndrome
   d. none of the above

9. The greatest maximum voluntary clenching force has been measured to be 275 pounds per square inch.
   a. True
   b. False

10. Inflammatory mechanisms have been shown to be involved in temporomandibular joint pain and dysfunction.
    a. True
    b. False

11. Inflammatory mediators contribute to the _____ , and increase _____ at the second-order neurons of the trigeminal vascular system.
    a. joint accretive process; nociceptive excitation
    b. joint degenerative process; periceptive excitation
    c. joint degenerative process; nociceptive excitation
    d. none of the above

12. Sleep bruxism subjects may present with _____.
    a. scalloping of the lateral tongue borders
    b. ridging of the buccal mucosa
    c. exostosis in the mandible or maxilla
    d. all of the above

13. Hypertrophied masseter and temporalis muscles may be present in patients with bruxism.
    a. True
    b. False

14. The physical damage and pain of parafunctional occlusion are related to the _____ of the parafunctional activity and the position of the stomatagnathic system.
    a. intensity
    b. duration
    c. frequency
    d. all of the above

15. The prevalence of migraine in the U.S. general population has been estimated to be _____.
    a. 6 percent
    b. 12 percent
    c. 18 percent
    d. none of the above

16. In patients with migraine, it is essential to consider both primary headaches and secondary headaches due to other underlying causes (in the extreme, as a result of an intracranial pathology).
    a. True
    b. False

17. It is generally accepted that the development of migraine headache pain depends on the activation of sensory afferent fibers of the ophthalmic division (V1) of the trigeminal nerve.
    a. True
    b. False

18. The putative role of facial pain in migraine pathophysiology has been described in the literature.
    a. True
    b. False

19. A primary goal with respect to parafunction is to _____.
    a. prevent damage to orofacial structures
    b. cure the condition
    c. reduce pain complaints
    d. a and c

20. Options for the treatment of parafunction include_____.
    a. behavior modification decreased duration of anesthesia
    b. the use of intraoral appliances
    c. pharmacologic therapies
    d. all of the above

21. The first report of oral appliance use for parafunction was by _____ in 1901.
    a. Barolyi
    b. von Tripp
    c. Karolyi
    d. none of the above

22. A soft mouth guard is usually only recommended for use on a short-term basis.
    a. True
    b. False

23. Mandibular appliances may be _____.
    a. full arch coverage or partial arch coverage
    b. anterior or posterior coverage
    c. made of soft or hard material
    d. all of the above

24. One study found that maxillary oral appliances may aggravate breathing disorders.
    a. True
    b. False

25. The concept of an oral appliance that provides contact on only anterior teeth was first introduced in the mid 1900s.
    a. True
    b. False

26. It has been found that elevator muscle activity is decreased while clenching on an anterior–contact–only appliance compared to maximum voluntary clenching of the natural dentition and on posterior coverage oral appliances.
    a. True
    b. False

27. The selection of an appliance depends on _____.
    a. clinical parameters
    b. the anticipated therapy
    c. clinician preference
    d. all of the above

28. Appliances providing anterior coverage of the incisors only have been suggested for the treatment and prevention of _____.
    a. medically diagnosed migraine pain
    b. tension-type headache
    c. temporomandibular joint disorders
    d. all of the above

29. Irrespective of the type of appliance selected, it is important that regular monitoring be carried out.
    a. True
    b. False

30. By providing patients with an appropriate oral appliance, compliance with therapy will be improved, ultimately resulting in an improved quality of life.
    a. True
    b. False

ANSWER SHEET

Occlusion, Function, and Parafunction: Understanding the Dynamics of a Healthy Stomatagnathic System

Name: Title: Specialty:

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Requirements for successful completion of the course and to obtain dental continuing education credits: 1) Read the entire course. 2) Complete all information above. 3) Complete answer sheets in either pen or pencil. 4) Mark only one answer for each question. 5) A score of 70% on this test will earn you 4 CE credits. 6) Complete the Course Evaluation below. 7) Make check payable to PennWell Corp.

Educational Objectives

1. Define parafunction and the activities associated with this
2. Identify the signs and symptoms of parafunctional activity
3. Know the considerations and steps involved in diagnosing parafunctional activity
4. Identify the types of appliances that can be used to manage parafunction, their advantages and disadvantages, and considerations in selecting an appliance for individual patients

Course Evaluation

Please evaluate this course by responding to the following statements, using a scale of Excellent = 5 to Poor = 0.

1. Were the individual course objectives met? Objective #1: Yes No Objective #3: Yes No Objective #2: Yes No Objective #4: Yes No

2. To what extent were the course objectives accomplished overall?	5	4	3	2	1	0
3. Please rate your personal mastery of the course objectives.	5	4	3	2	1	0
4. How would you rate the objectives and educational methods?	5	4	3	2	1	0
5. How do you rate the author's grasp of the topic?	5	4	3	2	1	0
6. Please rate the instructor's effectiveness.	5	4	3	2	1	0
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8. Do you feel that the references were adequate? Yes No

9. Would you participate in a similar program on a different topic? Yes No

10. If any of the continuing education questions were unclear or ambiguous, please list them.

_____

11. Was there any subject matter you found confusing? Please describe.

_____

_____

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_____

_____

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4.	Ⓐ	Ⓑ	Ⓒ	Ⓓ	19.	Ⓐ	Ⓑ	Ⓒ	Ⓓ
5.	Ⓐ	Ⓑ	Ⓒ	Ⓓ	20.	Ⓐ	Ⓑ	Ⓒ	Ⓓ
6.	Ⓐ	Ⓑ	Ⓒ	Ⓓ	21.	Ⓐ	Ⓑ	Ⓒ	Ⓓ
7.	Ⓐ	Ⓑ	Ⓒ	Ⓓ	22.	Ⓐ	Ⓑ	Ⓒ	Ⓓ
8.	Ⓐ	Ⓑ	Ⓒ	Ⓓ	23.	Ⓐ	Ⓑ	Ⓒ	Ⓓ
9.	Ⓐ	Ⓑ	Ⓒ	Ⓓ	24.	Ⓐ	Ⓑ	Ⓒ	Ⓓ
10.	Ⓐ	Ⓑ	Ⓒ	Ⓓ	25.	Ⓐ	Ⓑ	Ⓒ	Ⓓ
11.	Ⓐ	Ⓑ	Ⓒ	Ⓓ	26.	Ⓐ	Ⓑ	Ⓒ	Ⓓ
12.	Ⓐ	Ⓑ	Ⓒ	Ⓓ	27.	Ⓐ	Ⓑ	Ⓒ	Ⓓ
13.	Ⓐ	Ⓑ	Ⓒ	Ⓓ	28.	Ⓐ	Ⓑ	Ⓒ	Ⓓ
14.	Ⓐ	Ⓑ	Ⓒ	Ⓓ	29.	Ⓐ	Ⓑ	Ⓒ	Ⓓ
15.	Ⓐ	Ⓑ	Ⓒ	Ⓓ	30.	Ⓐ	Ⓑ	Ⓒ	Ⓓ

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PLEASE PHOTOCOPY ANSWER SHEET FOR ADDITIONAL PARTICIPANTS.

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