

SYSTEMATIC REVIEW

Prosthodontic planning in patients with temporomandibular disorders and/or bruxism: A systematic review

Daniele Manfredini, DDS, PhD^a and Carlo E. Poggio, DDS, PhD^b

Debates on the role of dental occlusion as a risk factor for temporomandibular disorders (TMDs) have inflamed the dental profession. Also, the possible relationship with bruxism has been a common topic of discussion.¹ Studies suggest that the cause of both TMDs and bruxism is less linked to occlusal morphology than believed in the past^{2,3} but that practitioners have yet to fully abandon some classic occlusion-oriented theories. Irreversible occlusal changes of prosthodontic or orthodontic rehabilitations cannot be recommended for the management or even the prevention of such conditions.⁴⁻⁶ Notwithstanding patients with TMD symptoms or with severe bruxism-related signs frequently present with indications for prosthetic treatment, such as partial edentulism, esthetic deficiencies, or functional problems. Those patients should be managed carefully, and the prosthodontist should be well versed in the current concepts of TMDs and bruxism.

ABSTRACT

Statement of problem. The presence of temporomandibular disorders (TMDs) and/or bruxism signs and symptoms may present multifaceted concerns for the prosthodontist.

Purpose. The purpose of this systematic review was to evaluate the relationship between prosthetic rehabilitation and TMDs and bruxism.

Material and methods. Three research questions were identified based on different clinical scenarios. Should prosthodontics be used to treat TMD and/or bruxism? Can prosthodontics cause TMDs and/or bruxism? How can prosthodontics be performed (for prosthetic reasons) in patients with TMDs and/or bruxism? A systematic search in the PubMed database was performed to identify all randomized clinical trials (RCTs) comparing the effectiveness of prosthodontics with that of other treatments in the management of TMDs and/or bruxism (question 1); clinical trials reporting the onset of TMDs and/or bruxism after the execution of prosthetic treatments in healthy individuals (question 2); and RCTs comparing the effectiveness of different prosthodontics strategies in the management of the prosthetic needs in patients with TMDs and/or bruxism (question 3).

Results. No clinical trials of the reviewed topics were found, and a comprehensive review relying on the best available evidence was provided. Bruxism is not linearly related to TMDs, and both of these conditions are multifaceted. Based on the diminished causal role of dental occlusion, prosthetic rehabilitation cannot be recommended as a treatment for the 2 conditions. In theory, they may increase the demand for adaptation beyond the stomatognathic system's tolerability. No evidence-based guidelines were available for the best strategy for managing prosthetic needs in patients with TMDs and/or bruxism.

Conclusions. This systematic review of publications revealed an absence of RCTs on the various topics concerning the relationship between TMD and bruxism and prosthodontics. Based on the best available evidence, prosthetic changes in dental occlusion are not yet acceptable as strategies for solving TMD symptoms or helping an individual stop bruxism. Clinicians should take care when performing irreversible occlusal changes in healthy individuals and in patients with TMD and/or bruxism. (*J Prosthet Dent* 2016;■:■-■)

Temporomandibular disorders are a heterogeneous group of conditions affecting the temporomandibular joints (TMJ), the jaw muscles, and the related structures.^{7,8} They have a multifactorial cause, with an

This article is based on D.M.'s presentation to the American Academy of Fixed Prosthodontics, Chicago, IL, February 2016.

^aAssistant Professor, School of Dentistry, University of Padova, Padova, Italy.

^bAssistant Professor, Department of Prosthodontics, Eastman Institute for Oral Health, University of Rochester, Rochester, NY; and Assistant Professor, Maxillofacial Surgery and Dentistry UOC, School of Dentistry, University of Milan, Fondazione IRCCS Ca' Granda, Milan, Italy.

Clinical Implications

The current body of evidence suggests that the concept of correcting occlusion should not be the reason for restorative treatment.

interaction of systemic (psychosocial, genetic, hormonal, neurological) and anatomic (facial morphology) factors. Factors also include the complex relationship with bruxism and are of much greater importance than anatomic factors in explaining the onset and perpetuation of symptoms and in predicting individual prognoses and treatment outcomes.⁹

Bruxism is "a repetitive jaw-muscle activity characterized by clenching or grinding of the teeth and/or by bracing or thrusting of the mandible. Bruxism has two distinct circadian manifestations: it can occur during sleep (indicated as sleep bruxism [SB]) or during wakefulness (indicated as awake bruxism [AB])."¹⁰ Such specifications of the different motor activities featuring bruxism highlight the need to consider their possible different causes and clinical consequences. However, as for TMDs, bruxism is centrally and not peripherally mediated.²

The relationship between TMDs and bruxism is controversial. A systematic review of publications on the topic yielded interesting findings as far as the contrasting results emerging from studies adopting different strategies to diagnose bruxism.¹¹ Generally, the findings supported an association between self-reported/questionnaire-diagnosed bruxism and TMD symptoms,^{12,13} with minor exceptions.¹⁴ Such results were consistent with studies of clinically diagnosed bruxism,^{15,16} whereas experimental investigations of forced, voluntary bruxism-like muscle activities elicited only transient muscle pain.^{17,18} Studies of tooth wear as a possible proxy for bruxism did not find any associations with TMDs.^{19,20} Finally, the few investigations that measured muscle activities during sleep by either polysomnography (PSG) or electromyography (EMG) provided hard-to-interpret results with inconsistent findings.^{21,22} Studies published later than 2010 drew the same contrasting results between investigations of PSG-based bruxism diagnosis, which showed no association with myofascial pain,²³ and studies adopting a self-reported approach to bruxism detection, most of which reported its association with TMDs.²⁴⁻²⁸ Thus, the research does not clearly support either the presence or the absence of a cause-and-effect relationship between the 2 conditions, probably because different conditions (SB versus AB; clenching versus grinding) have often been meshed together, regardless of the potentially different consequences in terms of muscle

fatigue and joint stress.²⁹⁻³¹ For future studies of the bruxism-TMD relationship, the different patterns of EMG activity characterizing the various bruxism phenomena and the observations that motor adaptations to pain are not always straightforward should be taken into account.³²⁻³⁵

As for dental occlusion, seminal papers adopting multiple variable models^{36,37} and comprehensive reviews of publications³⁸⁻⁴⁰ suggest that the relationship between both TMDs and bruxism and the natural dental occlusion is very weak, if it exists at all. Notwithstanding, for the prosthodontist, some interesting considerations came from studies introducing iatrogenic changes to dental occlusion. In healthy individuals, the placement of a restoration in supraocclusion can at worst cause local trauma; however, it rarely leads to transient dental and/or masticatory muscle pain and can be reversed by removing the interference.⁴¹ Concurrently, there is a reduction in the usual EMG activity of the masseter muscles⁴² and no effect on the pressure pain threshold.⁴³ This suggests the establishment of an avoidance adaptation pattern, as confirmed by empirical clinical observations that patients are not able to masticate on restorations in supraocclusion and try to avoid contacts with that tooth. Thus, bruxism cannot be triggered by iatrogenic changes to dental occlusion.³⁸

Interestingly, individuals with a history of TMD may have an increased risk for palpation-elicited muscle pain in response to artificially introduced occlusal interferences.⁴⁴ This should be borne in mind when carrying out rehabilitation treatments involving periods of occlusal instability due, for example, to interim restorations, increases in vertical dimension, or shifting of teeth. Clinically, the importance of these findings should not be overestimated or incorrectly translated to healthy individuals. Indeed, any transient muscle pain in response to the placement of an artificial interference cannot be equated with the presence of clinically relevant, treatment demanding TMDs; similarly, an acute, simulated occlusal alteration, cannot really be compared with a clinical situation characterized by the presence of a nonideal natural dentition to which the patient has gradually adapted over a period of years.

Within this complex framework of interactions, published studies of the relationship between prosthodontics and TMD and bruxism was systematically reviewed with a focus on the potential cause-and-effect implications and on the strategies for planning prosthetic treatments in patients with TMD and/or bruxism.

MATERIAL AND METHODS

The 3 following possible clinical questions based on different scenarios were identified for review purposes:

should prosthodontics be a treatment option for TMD or bruxism? Can prosthodontics cause TMD and/or bruxism? How should prosthodontics be performed (for prosthetic reasons) in patients with TMD and/or bruxism?

A systematic search in the PubMed database was performed on July 25, 2016, with the goal of identifying all clinical trials of the above topics. The search queries provided that the MeSH term "prosthodontics" be combined with either the MeSH term "temporomandibular joint disorders" or "bruxism." As a first step, the limit was set to clinical trials, and inclusion in the review was tentatively reserved for investigations assessing the role of prosthodontics as either a treatment (question 1) or a cause (question 2) for TMD or bruxism, as well as assessing the clinical effectiveness of different prosthodontic treatments/protocols to solve prosthetic problems in patients with TMD and/or bruxism (question 3). The following criteria were imposed for inclusion in the review for the specific questions: (1) randomized clinical trials (RCTs) comparing the effectiveness of prosthodontics with other treatments in the management of TMDs and/or bruxism; (2) clinical trials reporting the onset of TMDs and/or bruxism after the execution of prosthetic treatments in healthy individuals; and (3) RCTs comparing the effectiveness of different prosthodontic strategies concerning the management of vertical dimension of occlusion, intermaxillary relationship recordings, occlusal scheme design in the management of prosthetic needs in patients with TMDs and/or bruxism. Case reports, review articles, and letters to the editor were excluded from consideration. As a second step, the search was extended to the full citation lists identified by the above queries. In addition, the search was expanded to the related article lists and to author personal collections. Two authors (D.M., C.P.) screened the abstracts of the full list of citations to identify potential articles for full-text retrieval. Any decision about the inclusion/exclusion in the review was made by consensus.

RESULTS

The first search step identified only 11 citations with the query "prosthodontics" AND "temporomandibular joint disorders" and 10 citations with the query "prosthodontics" AND "bruxism." None of them satisfied the inclusion criteria. Thus, the search was extended to the full citation lists identified by the above queries, providing 622 and 350 citations, respectively. Examination of the titles and abstracts excluded all papers, none of which had a study design specifically aimed at addressing either of the above-described clinical scenarios. Because of the absence of relevant clinical trials,

the 3 following clinical scenarios were discussed based on the best available evidence.

DISCUSSION

Should prosthodontics be a treatment option for TMD or bruxism?

The first scenario concerns the possible role of prosthetic rehabilitation as a therapy for TMDs or bruxism. The concept that irreversible occlusal changes should be the end procedure of any TMD treatment implies that something is wrong with the occlusion and that such abnormality provokes TMD symptoms. Evidence in support of this approach is arguable.⁴⁵ Most arguments against occlusal finalization for TMD purposes came from orthodontic studies showing that orthodontic treatment neither reduces nor increases the risk for TMDs.⁴⁵ Interestingly, even if adolescents with reverse articulation have an increased risk of developing TMJ disk displacement during early adulthood, the presence of disk displacement in adult age is not reduced with correction of the reverse articulation during adolescence.⁴⁶ Another recent paper showed that the epidemiology of malocclusion in untreated adults is poorly known, but its presence is so diffused and overlapping with the prevalence of malocclusion in individuals with TMD that any further assumptions about correcting occlusion in patients with TMD should be discouraged from a straightforward epidemiological viewpoint.⁴⁷ Thus, correcting dental occlusion and/or repositioning the mandible for TMD treatment purposes is not medically necessary.^{6,48}

Given these premises, the general practitioner may be perplexed by the apparent occlusion-related effects of oral appliances (OA) on TMD symptoms.^{49,50} Oral appliances may favor reorganization of muscle fiber recruitment patterns^{51,52} and a shift in the area of highest joint loading.⁵³ These effects are unrelated to the correction of occlusion and are simply due to the thickness of the appliance. In addition, emerging evidence seems to minimize the true efficacy of OA as a treatment option.^{54,55} Prosthodontists should be conscious of the possible transient effects of OA, which are not miraculous devices that position the mandibles correctly but are instead responsible for transient shifts in joint and muscle loading.

As for bruxism, arguments in favor of a possible therapeutic role for prosthetic treatments are so weak that the recent studies show no traces.⁵⁶ Notwithstanding, recent knowledge of the relationship between bruxism and obstructive sleep apnea (OSA) may provide an interesting framework for a paradoxical scenario. Mandibular advancement devices are a option for OSA treatment, with several studies trying to refine their customized indications.^{57,58} Some investigations showed

that, parallel to OSA, mandibular advancement devices seem to reduce SB events.^{59,60} Such effects on SB are in part explained with the potential OSA-ending goal of some SB events, which should therefore disappear in the absence of apnea.⁶¹ Based on that, some unproven philosophies advocating the need for mandible repositioning for prosthodontic purposes and for controlling bruxism may actually reduce SB as a “side-effect” of OSA decrease induced by jaw advancement.⁶²

Can prosthodontics cause TMDs and/or bruxism?

A second possible scenario is the risk that prosthodontic treatment may even lead to TMDs or bruxism in otherwise asymptomatic patients. Because single-tooth interference may be considered an involuntary clinical mistake, and their effects have been discussed above, extensive rehabilitation should focus on voluntarily changing the interarch relationship. Increases in the occlusal vertical dimension (OVD) and mandible repositioning treatments are examples of such changes.

The biological plausibility that a centrally mediated phenomenon such as bruxism may be induced by a prosthetic treatment is nonexistent.³⁸ As for TMDs, the diminished importance of occlusion in the field of TMD does not mean ignoring it while planning prosthetic work. The masticatory system has extraordinary powers of adaptation, both to natural dental-skeletal abnormalities⁶³ and to iatrogenic modifications.⁶⁴ The recent orthodontic publications suggesting that even unsuccessful orthodontic treatments do not increase the risk for TMDs with respect to successful orthodontics or to absence of orthodontic treatment support this concept.⁵ Notwithstanding, with respect to orthodontics, prosthetic treatments often provoke a more immediate change in the interarch relationship, thus potentially requiring more adaptation. For this reason, the safest prosthodontic strategy against the possible onset of TMD symptoms is not to plan occlusal modifications that jeopardize the capacity for accommodation. Rehabilitations based on preconceived ideal occlusal schemes or interarch relations are not advisable, as they fail to account for the muscle engrams and the functional adaptation that the neuromuscular system of an asymptomatic patient has developed naturally.

In this regard, for decades, centric relation has been a controversial and much debated concept.⁶⁵ Its definition evolved from a mechanically determined to a physiologically acceptable position,⁶⁶ implying a considerable reduction in the importance as a key reference position and, to some extent, as a therapeutic position for TMJs.⁶⁷ This is also due to the absence of an ideal condylar position associated with a healthy TMJ or jaw muscle function.⁶⁸⁻⁷⁰ Therefore, no grounds exist for considering any single condyle position more appropriate than any other, and consequently, the pursuit of any condylar

repositioning strategy is not needed. Similarly, OVD has for years been considered a fundamental parameter for prosthodontics purposes.⁶⁶ Its maintenance is a result of the interaction of the genetic potential for craniofacial tissue growth with environmental factors and with the dynamic development of neuromuscular function during skeletal growth and aging.⁷¹ Even patients with the most severe vertical dental wear may be not associated with relevant OVD loss, thanks to the continuous dentoalveolar eruption that partly compensates for the loss of tooth substance. Based on the wide range of physiologically acceptable centric relation and OVD values at the inter- and intraindividual level, the habitual position of the interarch relationship should be used as a reference whenever possible, and prosthetic treatments required to change it should provide the minimum shift from that position. This straightforward guide may reduce the demand for adaptation to the new position while reducing the risk of discomfort, if not TMD symptoms.

In theory, any repositioning based on preconceived ideas of normal relationships between the components of the stomatognathic system can backfire, as there is no way to predict how the system reacts.^{72,73} As a general rule, changes must be carried out only for valid prosthetic reasons and be performed over the longest possible period by testing adaptation with interim restorations.⁷⁴

How should prosthodontics (for prosthetic reasons) be performed in patients with TMDs and/or bruxism?

The third, and probably most clinically relevant scenario, concerns the need to perform treatments for prosthetic reasons in patients who have ongoing TMD symptoms or signs of bruxism. Do these individuals need any special attention with respect to other situations a prosthodontist often faces? In routine clinical practice, the presence of clicking disk displacement sounds do not constitute contraindications to occlusal rehabilitations, even if investigation by a clinician experienced in TMD management is recommended. On the contrary, in patients with ongoing TMDs, their symptoms should be treated before starting any prosthetic treatment. In particular, the presence of TMJ and/or masticatory muscle pain and a limited range of joint movement are absolute contraindications to extensive prosthetic rehabilitation. Patients with TMDs are hypervigilant to stressors and may thus adapt less easily than healthy patients to the occlusal and psychological stress of a modification to their occlusal scheme because of their delicate psychophysiological equilibrium.⁷⁵⁻⁷⁷

As for bruxism, a recent review of complications with implant-supported restorations⁷⁸ suggests that it may be associated more with mechanical (screw loosening, ceramic chipping or fracture, fixture or abutment fracture) than biological (loss of marginal bone attachment, biological failure) causes, in line with suggestions that it is

unlikely to provoke biological complications on the periodontium.⁷⁹ Thus, we cannot hypothesize that the role of bruxism may be different in teeth- or implant-supported prostheses. The real difference for the prosthodontist is performing either complex, multiple-tooth restorations, or simple, single-or-few-tooth restorations. Indeed, although the latter can be easily managed by adopting simple strategies to reduce mechanical trauma, such as reducing cusp steepness, enlarging contact areas, and providing slight occlusal under-contact, the former require attention in the planning phases.

The 2 most frequent bruxism-related clinical scenarios are the restoration of severely worn dentitions or the execution of prosthetic treatments to replace or restore teeth in patients who have bruxism. The key issue in both conditions is the diagnosis. A self-reported approach, often performed with single-item evaluations during history taking,⁸⁰ is responsible for the lack of evidence-based knowledge on the topic of prosthodontic planning in bruxers.⁸¹ The current standard of reference for SB diagnosis is PSG recording,^{21,82} which is hard to introduce into the dental office routine and should thus be replaced with validated devices for in-home use.^{83,84} A definite AB diagnosis may be achieved with smartphone applications based on an ecological momentary assessment.⁸⁵ A thorough bruxism assessment in the preprosthetic stages also helps establish a solid doctor-patient relation and prevents potential medical litigation.

From a practical viewpoint, the clinician should adopt a safe, cautionary, sensible approach to a patient with bruxism who needs to undergo prosthetic treatment with strategies to reduce the potentially negative effects of bruxism. These include both surgical (number, size, location of implants) and prosthetic (occlusal design) aspects.

As for specific biomechanical suggestions, each implant or natural supporting element is proportionally less loaded with the increase in number.⁸⁶ In addition, as far as implants are concerned, their connection between each other may provide a better load distribution and reduce periimplant bone stress.⁸⁷ Long, wide implants may keep stress forces on surrounding tissues at minimum levels.⁸⁷ Cantilevers should be avoided as the lever arm on the distal abutment may be further enhanced in bruxism, whereas occlusal levers can be controlled with an adequate length of the prosthetic crown and not necessarily by placing the longest possible implant.^{88,89} The load-bearing capability may depend also on the bone structure and density, with high-density bone undergoing less deformation during functional load.^{90,91} As for load timing, based on suggestions of reduced success rates of immediately loaded implants,⁹² avoiding immediate-loading protocols is reasonable in patients with bruxism to minimize risk factors.⁸¹ Nonaxial loads

may be a source of periimplant bone stress and should therefore be avoided whenever the bone and antagonist occlusion allow.⁹³⁻⁹⁵ In addition, a certain freedom of movement is useful around the occlusal contact areas in maximum intercuspal position to create flatter cuspal planes to protect the prosthesis during eccentric movements. As for the restorative material, research does not support any clinical evidence.^{95,96} The long-debated dispute on this topic (high strength anatomic contour ceramics versus potentially chippable ceramics versus in-mouth restorable composite resins) has not yet been solved, and the choice of material for an extensive rehabilitation in patients with bruxism is often based on the clinician's predilections and patient expectations. In that respect, restorations with occlusal devices worn at night should be protected.

Notwithstanding those general remarks, the most interesting topic concerning the bruxism-TMD-prosthodontics relationship involves the frequent prosthetic need to increase the original OVD and to plan positional changes. Unfortunately, no evidence-based recommendations are available for how to perform those changes. None of the occlusal designs or interarch positional changes proposed over the years has ever been shown to be better than others or associated with a healthier stomatognathic system.⁹⁷⁻⁹⁹ Thus, positional changes of the mandible or an increase in OVD should be adopted only for prosthetic reasons. Examples include the presence of severe tooth wear preventing retentive crown preparations, insufficient interarch space to restore or replace missing teeth, and for esthetic reasons.^{96,100} Working with long-term interim restorations and using them as a template for the definitive rehabilitation, even if they seem to be based more on the clinician's experience than scientific evidence, is still likely the best option for managing any extensive prosthodontic rehabilitation safely.

The occlusal design of extensive rehabilitations in patients with TMDs or bruxism should be as simple as possible. Basic requisites such as a symmetrical distribution of interarch contacts, occlusal stability, and subjective comfort are generally enough to optimize function.^{73,81,98} Keeping this in mind, some technical schemes such as canine guidance for disclusion or some interarch recording techniques such as individually mounted articulators and facebows may help manage the clinical conditions of severe occlusal instability. However, the clinical reports¹⁰¹⁻¹⁰³ have never fully supported the reproducibility of any centric relation recording procedures. The plausible explanation for the purported clinical success of those pragmatic strategies is the high neuroplasticity-based adaptability of the system, rather than any real functional advantages.¹⁰⁴

Thus, after decades of working with the bias of considering occlusion as the main reason for treatment, a

new paradigm for dentistry has emerged: the correction of dental occlusion should not be the guiding principle or indication for a prosthetic treatment.

CONCLUSIONS

Based on a systematic review of the published research in 3 different TMD and bruxism-related clinical scenarios, that is the possible role of prosthetic treatment as therapy for TMDs and/or bruxism, its possible role as a cause/risk factor of TMDs and/or bruxism, and the planning of prosthetic rehabilitations in patients with TMDs and/or bruxism for prosthetic reasons, the following conclusions were drawn:

1. Randomized controlled trials and high-level evidence for any of the above topics are absent, thus leading to common sense rather than evidence-based recommendations.
2. Although prosthetic changes in dental occlusion are not yet acceptable as strategies to solve TMD symptoms or to help an individual stop bruxing, cautionary approaches must be recommended when planning irreversible occlusal changes in healthy individuals and in patients with TMDs and/or bruxism.

REFERENCES

1. Türp JC, Greene CS, Strub JR. Dental occlusion: a critical reflection on past, present and future concepts. *J Oral Rehabil* 2008;35:446-53.
2. Lobbezoo F, Naeije M. Bruxism is mainly regulated centrally, not peripherally. *J Oral Rehabil* 2001;28:1085-91.
3. Suvinen TI, Reade PC, Kemppainen P, Körönen M, Dworkin SF. Review of aetiological concepts of temporomandibular pain disorders: towards a biopsychosocial model for integration of physical disorder factors with psychological and psychosocial illness impact factors. *Eur J Pain* 2005;9: 613-33.
4. Luther F, Layton S, McDonald F. Orthodontics for treating temporomandibular joint (TMJ) disorders. *Cochrane Database Syst Rev* 2010;(7): CD006541.
5. Manfredini D, Stellini E, Gracco A, Lombardo L, Nardini LG, Siciliani G. Orthodontics is temporomandibular disorder-neutral. *Angle Orthod* 2016;86:649-54.
6. Greene CS, Obrez A. Treating temporomandibular disorders with permanent mandibular repositioning: is it medically necessary? *Oral Surg Oral Med Oral Pathol Oral Radiol* 2015;119:489-98.
7. Schiffman E, Ohrbach R, Truelove E, Look J, Anderson G, Goulet JP, et al; for the International RDC/TMD Consortium Network, International Association for Dental Research; Orofacial Pain Special Interest Group, International Association for the Study of Pain. Diagnostic criteria for temporomandibular disorders (DC/TMD) for clinical and research applications: recommendations of the International RDC/TMD Consortium Network and Orofacial Pain Special Interest Group. *J Oral Facial Pain Headache* 2014;28:6-27.
8. American Association for Dental Research. AADR policy statement on temporomandibular disorders. Available at: <http://www.aadronline.org/i4a/pages/index.cfm?pageid=3465#>. Accessed Month March 20, 2016.
9. Ohrbach R, Bair E, Fillingim RB, Gonzalez Y, Gordon SM, Lim PF, et al. Clinical orofacial characteristics associated with risk of first-onset TMD: the OPPERA prospective cohort study. *J Pain* 2013;14:T33-50.
10. Lobbezoo F, Ahlberg J, Glaros AG, Kato T, Koyano K, Lavigne GJ, et al. Bruxism defined and graded: an international consensus. *J Oral Rehabil* 2013;40:2-4.
11. Manfredini D, Lobbezoo F. Relationship between bruxism and temporomandibular disorders: a systematic review of literature from 1998 to 2008. *Oral Surg Oral Med Oral Pathol Oral Radiol Endod* 2010;109: e26-50.
12. Ciancaglini R, Gherlone E, Radaelli G. The relationship of bruxism with craniofacial pain and symptoms from the masticatory system in the adult population. *J Oral Rehabil* 2001;28:842-8.
13. Johansson A, Unell L, Carlsson GE, Soderfeldt B, Halling A. Risk factors associated with symptoms of temporomandibular disorders in a population of 50- and 60-year-old subjects. *J Oral Rehabil* 2006;33:473-81.
14. Van der Meulen MJ, Lobbezoo F, Aartman IH, Naeije M. Self-reported oral parafunctions and pain intensity in temporomandibular disorder patients. *J Orofac Pain* 2006;20:31-5.
15. Israel HA, Diamond B, Saed-Nejad F, Ratcliffe A. The relationship between parafunctional masticatory activity and arthroscopically diagnosed temporomandibular joint pathology. *J Oral Maxillofac Surg* 1999;57:1034-9.
16. Manfredini D, Cantini E, Romagnoli M, Bosco M. Prevalence of bruxism in patients with different research diagnostic criteria for temporomandibular disorders (RDC/TMD) diagnoses. *Cranio* 2003;21:279-85.
17. Arima T, Svensson P, Arendt-Nielsen L. Experimental grinding in healthy subjects: a model for post-exercise jaw muscle soreness. *J Orofac Pain* 1999;13:104-14.
18. Svensson P, Burgaard A, Schlosser S. Fatigue and pain in human jaw muscles during a sustained, low-intensity clenching task. *Arch Oral Biol* 2001;46:773-7.
19. John MT, Frank H, Lobbezoo F, Drangsholt M, Dette KE. No association between incisal tooth wear and temporomandibular disorders. *J Prosthet Dent* 2002;87:197-203.
20. Pergamalian A, Rudy TE, Zaki HS, Greco CM. The association between wear facets, bruxism, and severity of facial pain in patients with temporomandibular disorders. *J Prosthet Dent* 2003;90:194-200.
21. Romph PH, Daigle-Landy D, Guitard F, Montplaisir JY, Lavigne GJ. Identification of a sleep bruxism subgroup with a higher risk of pain. *J Dent Res* 2007;86:837-42.
22. Rossetti LM, Pereira de Araujo Cdos R, Rossetti PH, Conti PC. Association between rhythmic masticatory muscle activity during sleep and masticatory myofascial pain: a polysomnographic study. *J Orofac Pain* 2008;22:190-200.
23. Raphael KG, Sirois DA, Janal MN, Wigren PE, Dubrovsky B, Nemelovsky LV, et al. Sleep bruxism and myofascial temporomandibular disorders. A laboratory-based polysomnographic investigation. *J Am Dent Assoc* 2012;143:1223-31.
24. Michelotti A, Cioffi I, Festa P, Scala G, Farella M. Oral parafunctions as risk factors for diagnostic TMD subgroups. *J Oral Rehabil* 2010;37:157-62.
25. Fernandes G, Franco AL, Siqueira JT, Goncalves DA, Camparis CM. Sleep bruxism increases the risk for painful temporomandibular disorder, depression and non-specific physical symptoms. *J Oral Rehabil* 2012;39: 538-44.
26. Manfredini D, Winocur E, Guarda-Nardini L, Lobbezoo F. Self-reported bruxism and temporomandibular disorders: findings from two specialized centers. *J Oral Rehabil* 2012;39:319-25.
27. Van der Meulen MJ, Lobbezoo F, Aartman IH, Naeije M. Validity of the oral behaviours checklist: correlations between OBC scores and intensity of facial pain. *J Oral Rehabil* 2014;41:115-21.
28. Manfredini D, Peretta R, Guarda-Nardini L, Ferronato G. Predictive value of combined clinically diagnosed bruxism and occlusal features for TMJ pain. *J Craniomandib Pract* 2010;28:105-13.
29. Lavigne GJ, Huynh N, Kato T, Okura K, Adachi K, Yao D, et al. Genesis of sleep bruxism: motor and autonomic-cardiac interactions. *Arch Oral Biol* 2007;52:381-4.
30. Manfredini D, Lobbezoo F. Role of psychosocial factors in the etiology of bruxism. *J Orofac Pain* 2009;23:153-66.
31. Lund JP, Donga R, Widmer CG, Stohler CS. The pain-adaptation model: a discussion on the relationship between chronic musculoskeletal pain and motor activity. *Can J Physiol Pharmacol* 1991;69:683-94.
32. Murray GM, Peck CC. Orofacial pain and jaw muscle activity: a new model. *J Orofac Pain* 2007;21:263-78.
33. Minami I, Akhter R, Albersen I, Burger C, Whittle T, Lobbezoo F, et al. Masseter motor unit recruitment is altered in experimental jaw muscle pain. *J Dent Res* 2013;92:143-8.
34. Manfredini D, Cicilovo F, Stellini E, Favero L, Guarda-Nardini L. Surface electromyography findings in unilateral myofascial pain patients: comparison of painful vs non painful sides. *Pain Med* 2013;14:1848-53.
35. Paesani DA, Lobbezoo F, Gelos C, Guarda-Nardini L, Ahlberg J, Manfredini D. Correlation between self-reported and clinically based diagnoses of bruxism in temporomandibular disorders patients. *J Oral Rehabil* 2013;40:803-9.
36. Manfredini D, Castroflorio T, Perineti G, Guarda-Nardini L. Dental occlusion, body posture and temporomandibular disorders: where we are now and where we are heading for. *J Oral Rehabil* 2012;39:463-71.
37. Türp JC, Schindler H. The dental occlusion as a suspected cause for TMDs: epidemiological and etiological considerations. *J Oral Rehabil* 2012;39: 502-12.
38. Lobbezoo F, Ahlberg J, Manfredini D, Winocur E. Are bruxism and the bite causally related? *J Oral Rehabil* 2012;39:489-501.

39. Pullinger AG, Seligman DA. Quantification and validation of predictive values of occlusal variables in temporomandibular disorders using a multifactorial analysis. *J Prosthet Dent* 2000;83:66-75.
40. Manfredini D, Visscher C, Guarda-Nardini L, Lobbezoo F. Occlusal factors are not related with self-reported bruxism. *J Orofac Pain* 2012;26:163-7.
41. Clark GT, Tsukiyama Y, Baba K, Watanabe T. Sixty-eight years of experimental occlusal interference studies: what have we learned? *J Prosthet Dent* 1999;82:704-13.
42. Michelotti A, Farella M, Gallo LM, Veltri A, Palla S, Martina R. Effect of occlusal interference on habitual activity of human masseter. *J Dent Res* 2005;84:644-8.
43. Michelotti A, Farella M, Steenks MH, Gallo LM, Palla S. No effect of experimental occlusal interferences on pressure pain thresholds of the masseter muscles in healthy women. *Eur J Oral Sci* 2006;114:167-70.
44. Le Bell Y, Jämsä T, Korri S, Niemi PM, Alonen P. Effect of artificial occlusal interferences depends on previous experience of temporomandibular disorders. *Acta Odontol Scand* 2002;60:219-22.
45. Koh H, Robinson PG. Occlusal adjustment for treating and preventing temporomandibular joint disorders. *J Oral Rehabil* 2004;31:287-92.
46. Michelotti A, Iodice G, Piergentili M, Farella M, Martina R. Incidence of temporomandibular joint clicking in adolescents with and without unilateral posterior cross-bite: a 10-year follow-up study. *J Oral Rehabil* 2016;43:16-22.
47. Manfredini D, Perinetti G, Stellini E, Di Leonardo B, Guarda-Nardini L. Prevalence of static and dynamic dental malocclusion features in subgroups of temporomandibular disorder patients: Implications for the epidemiology of the TMD-occlusion association. *Quintessence Int* 2015;46:341-9.
48. Okeson JP. Evolution of occlusion and temporomandibular disorder in orthodontics: past, present, and future. *Am J Orthod Dentofacial Orthop* 2015;147:S216-23.
49. Dao TT, Lavigne GJ. Oral splints: the crutches for temporomandibular disorders and bruxism? *Crit Rev Oral Biol Med* 1998;9:345-61.
50. Klasser GD, Greene CS. Oral appliances in the management of temporomandibular disorders. *Oral Surg Oral Med Oral Pathol Oral Radiol Endod* 2009;107:212-23.
51. Van Eijden TMGV, Blanksma NG, Brugman P. Amplitude and timing of EMG activity in the human masseter muscle during selected motor tasks. *J Dent Res* 1993;72:599-606.
52. Terebesi S, Giannakopoulos NN, Brüstle F, Hellmann D, Türp JC, Schindler HJ. Small vertical changes in jaw relation affect motor unit recruitment in the masseter. *J Oral Rehabil* 2016;43:259-68.
53. Ettlin DA, Mang H, Colombo V, Palla S, Gallo LM. Stereometric assessment of TMJ space variation by occlusal splints. *J Dent Res* 2008;87:877-81.
54. Friction J, Look JO, Wright E, Alencar FG, Chen H, Lang M, et al. Systematic review and meta-analysis of randomized controlled trials evaluating intraoral orthopedic appliances for temporomandibular disorders. *J Orofac Pain* 2010;24:237-54.
55. Quintus V, Suominen AL, Huttunen J, Raustia A, Ylostalo P, Sipila K. Efficacy of stabilization splint treatment on facial pain-1-year follow-up. *J Oral Rehabil* 2015;42:439-46.
56. Manfredini D, Ahlberg J, Winocur E, Lobbezoo F. Management of sleep bruxism in adults: a qualitative systematic literature review. *J Oral Rehabil* 2015;42:862-74.
57. Aarab G, Lobbezoo F, Heymans MW, Hamburger HL, Naeije M. Long-term follow-up of a randomized controlled trial of oral appliance therapy in obstructive sleep apnea. *Respiration* 2011;82:162-8.
58. Marchese-Ragona R, Manfredini D, Mion M, Vianello A, Staffieri A, Guarda-Nardini L. Oral appliances for the treatment of obstructive sleep apnea in patients with low C-PAP compliance: a long-term case series. *J Craniomandib Sleep Pract* 2014;32:254-9.
59. Landry-Schönbeck A, de Grandmont P, Rompré PH, Lavigne GJ. Effect of an adjustable mandibular advancement appliance on sleep bruxism: a crossover sleep laboratory study. *Int J Prosthodont* 2009;22:251-9.
60. Mainieri VC, Saueressig AC, Fagondes SC, Teixeira ER, Rehm DD, Grossi ML. Analysis of the effects of a mandibular advancement device on sleep bruxism using polysomnography, the BiteStrip, the sleep assessment questionnaire, and occlusal force. *Int J Prosthodont* 2014;27:119-26.
61. Manfredini D, Guarda-Nardini L, Marchese-Ragona R, Lobbezoo F. Theories on possible temporal relationships between sleep bruxism and obstructive sleep apnea events. An expert opinion. *Sleep Breath* 2015;19:1459-65.
62. Sugimoto K, Yoshimi H, Sasaguri K, Sato S. Occlusal factors influencing the magnitude of sleep bruxism activity. *J Craniomandib Pract* 2011;29:127-37.
63. De Boever JA, De Laat A. Prosthetic rehabilitation in TMD patients. In: Manfredini D, editor. Current concepts on temporomandibular disorders. Berlin: Quintessence Publishing; 2010:417-28.
64. Türp JC, Strub JR. Prosthetic rehabilitation in patients with temporomandibular disorders. *J Prosthet Dent* 1996;76:418-23.
65. Pokorny PH, Wiens JP, Litvak H. Occlusion for fixed prosthodontics: a historical perspective of the gnathological influence. *J Prosthet Dent* 2008;99:299-313.
66. Academy of Prosthodontics. The glossary of prosthodontic terms: 8th ed. *J Prosthet Dent* 2005;94:10-92.
67. Rinchuse DJ, Kandasamy S. Centric relation: a historical and contemporary orthodontic perspective. *J Am Dent Assoc* 2006;137:494-501.
68. Pullinger AG, Seligman DA, John MT, Harkins S. Multifactorial modeling of temporomandibular anatomic and orthopedic relationships in normal versus undifferentiated disk displacement joints. *J Prosthet Dent* 2002;87:289-97.
69. Pullinger A. Establishing better biological models to understand occlusion. I: TM joint anatomic relationships. *J Oral Rehabil* 2013;40:296-318.
70. Lelis ÉR, Guimarães Henrques JC, Tavares M, de Mendonça MR, Fernandes Neto AJ, Almeida Gde A. Cone-beam tomography assessment of the condylar position in asymptomatic and symptomatic young individuals. *J Prosthet Dent* 2015;114:420-5.
71. Moyers RE, Wainright RL. Skeletal contributions to occlusal development. In: McNamara JA Jr, editor. The biology of occlusal development. Monograph 7. Craniofacial Growth ser. Ann Arbor, MI: University of Michigan Press; 1977:89-111.
72. Sessle BJ. Biological adaptation and normative values. *Int J Prosthodont* 2003;16(suppl):72-3.
73. Klineberg I, Stohler CS. Interface of occlusion. *Int J Prosthodont* 2003;16(suppl):89-90.
74. Celenza FV. The theory and clinical management of centric positions. II. Centric relation and centric relation occlusion. *Int J Periodontics Restorative Dent* 1984;4:62-86.
75. Sarlani E, Grace EG, Reynolds MA, Greenspan JD. Evidence for up-regulated central nociceptive processing in patients with masticatory myofascial pain. *J Orofac Pain* 2004;18:41-55.
76. Manfredini D, Bandettini di Poggio A, Cantini E, Dell'Osso L, Bosco M. Mood and anxiety psychopathology and temporomandibular disorder: a spectrum approach. *J Oral Rehabil* 2004;31:933-40.
77. Maixner W, Fillingim R, Sigurdsson A, Kincaid S, Silva S. Sensitivity of patients with painful temporomandibular disorders to experimentally evoked pain: evidence for altered temporal summation of pain. *Pain* 1998;76:71-81.
78. Manfredini D, Poggio CE, Lobbezoo F. Is bruxism a risk factor for dental implants? A systematic review of the literature. *Clin Implant Dent Relat Res* 2014;16:460-9.
79. Manfredini D, Ahlberg J, Mura R, Lobbezoo F. Bruxism is unlikely to cause damage to the periodontium: findings from a systematic literature assessment. *J Periodontol* 2015;86:546-55.
80. Manfredini D, Winocur E, Guarda-Nardini L, Paesani D, Lobbezoo F. Epidemiology of bruxism in adults: a systematic review of the literature. *J Orofac Pain* 2013;27:99-110.
81. Manfredini D, Bucci MB, Sabattini VB, Lobbezoo F. Bruxism: overview of current knowledge and suggestions for dental implants planning. *J Craniomandib Pract* 2011;29:304-12.
82. Lavigne GJ, Rompré PH, Montplaisir JY. Sleep bruxism: validity of clinical research diagnostic criteria in a controlled polysomnographic study. *J Dent Res* 1996;75:546-52.
83. Castroflorio T, Deregius A, Bargellini A, Debernardi C, Manfredini D. Detection of sleep bruxism: comparison between an electromyographic and electrocardiographic portable holter and polysomnography. *J Oral Rehabil* 2014;41:163-9.
84. Stuginski-Barbosa J, Porporatti AL, Costa YM, Svensson P, Conti PC. Diagnostic validity of the use of a portable single-channel electromyography device for sleep bruxism. *Sleep Breath* 2016;20:695-702.
85. Manfredini D, Bracci A, Djukic G. Bruxapp: the ecological momentary assessment of awake bruxism. *Minerva Stomatol* 2016;65:252-5.
86. Duyck J, Van Oosterwyck H, Vander Sloten J, De Cooman M, Puers R, Naert I. Magnitude and distribution of occlusal forces on oral implants supporting fixed prostheses: an in vivo study. *Clin Oral Implants Res* 2000;11:465-75.
87. Guichet DL, Yoshinobu D, Caputo AA. Effect of splinting and interproximal contact tightness on load transfer by implant restorations. *J Prosthet Dent* 2002;87:528-35.
88. Renouard F, Nisand D. Impact of implant length and diameter on survival rates. *Clin Oral Implants Res* 2006;17(suppl 2):35-51.
89. Malo P, de Araujo Nobre M, Rangert B. Implants placed in immediate function in periodontally compromised sites: a five-year retrospective and one-year prospective study. *J Prosthet Dent* 2007;97:S86-95.
90. Manz MC. Factors associated with radiographic vertical bone loss around implants placed in a clinical study. *Ann Periodontol* 2000;5:137-51.
91. Isidor F. Influence of forces on peri-implant bone. *Clin Oral Implant Res* 2006;17(suppl 2):8-18.
92. Esposito M, Grusovin MG, Willings M, Coulthard P, Worthington HV. Interventions for replacing missing teeth: different times for loading dental implants. *Cochrane Database Syst Rev* 2007;2:CD003878.
93. Barbier L, Schepers E. Adaptive bone remodeling around oral implants under axial and nonaxial loading conditions in the dog mandible. *Int J Oral Maxillofac Implants* 1997;12:215-23.
94. Kitamura E, Stegaroiu R, Nomura S, Miyakawa O. Influence of marginal bone resorption on stress around an implant - a three dimensional finite element analysis. *J Oral Rehabil* 2005;32:279-86.

95. Carlsson GE. Dental occlusion: modern concepts and their application in implant prosthodontics. *Odontology* 2009;97:8-17.
96. Johansson A, Johansson AK, Omar R, Carlsson GE. Rehabilitation of the worn dentition. *J Oral Rehabil* 2008;35:548-66.
97. Taylor DT, Wiens J, Carr A. Evidence-based considerations for removable prosthodontic and dental implant occlusion: a literature review. *J Prosthet Dent* 2005;94:555-60.
98. Klineberg I, Kingston D, Murray G. The bases for using a particular occlusal design in tooth and implant-borne reconstructions and complete dentures. *Clin Oral Impl Res* 2007;18:151-67.
99. Gross MD. Occlusion in implant dentistry. A review of the literature of prosthetic determinants and current concepts. *Aust Dent J* 2008;53:S60-8.
100. Fayz F, Eslami A. Determination of occlusal vertical dimension: a literature review. *J Prosthet Dent* 1988;59:321-3.
101. Utz KH, Müller F, Lückerath W, Fuss E, Koeck B. Accuracy of check-bite registration and centric condylar position. *J Oral Rehabil* 2002;29:458-66.
102. Keshvad A, Winstanley RB. Comparison of the replicability of routinely used centric relation registration techniques. *J Prosthodont* 2003;12:90-101.
103. Hellmann D, Etz E, Giannakopoulos NN, Rammelsberg P, Schmitter M, Schindler HJ. Accuracy of transfer of bite recording to simulated prosthetic reconstructions. *Clin Oral Investig* 2013;17:259-67.
104. Sessle BJ. Mechanisms of oral somatosensory and motor functions and their clinical correlates. *J Oral Rehabil* 2006;33:243-61.

Corresponding author:

Dr Daniele Manfredini
Via Ingolstadt 3
54033 Marina di Carrara (MS)
ITALY
Email: daniele.manfredini@tin.it

Copyright © 2016 by the Editorial Council for *The Journal of Prosthetic Dentistry*.