Do Bruxism and Temporomandibular Disorders Have a Cause-and-Effect Relationship?

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Controversy continues to exist over the putative role of bruxism in the etiology of temporomandibular disorders. A commonly held concept is that bruxism leads to signs and symptoms characteristic of one or more of the subdiagnoses of temporomandibular disorders, while another hypothesis suggests that bruxism is a temporomandibular disorder itself that sometimes coexists with other forms of temporomandibular disorders. Following a thorough review of the literature in this article, it is concluded that the relationship between bruxism and temporomandibular disorders is still unclear. Future research should examine longitudinal epidemiologic and clinical/experimental data to establish or refute a cause-and-effect relationship. In doing so, the existence of various subgroups of temporomandibular disorders should be taken into account, and sleep-related bruxism should be discriminated from its daytime variant.
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In the management of functional disturbances of the orofacial region, bruxism is often identified as a contributing or causative factor, and treatment is designed to decrease bruxism activity or to decrease its purported injurious effects. However, much controversy exists over the interrelationship between bruxism and other disturbances of the masticatory system.[1] One view suggests that a true cause-and-effect relationship exists,[2] while others suggest only a mutual coexistence.[3] The purpose of the present study was to review the literature concerning the relationship between bruxism and temporomandibular disorders in an attempt to further clarify this issue.

Temporomandibular Disorders: A Multifarious Problem

Temporomandibular disorders (TMD) is a collective term embracing a number of musculoskeletal problems of the masticatory system.[4] According to the Task Force on Taxonomy of the International Association for the Study of Pain (IASP), TMD is characterized by pain and tenderness of the masticatory muscles and/or temporomandibular joints and is often associated with temporomandibular joint (TMJ) sounds and a restricted range of mandibular movements.[5]

Large differences in prevalence rates of TMD can be found in the literature.[6,7] Among others, these discrepancies may be because many studies did not use representative randomized samples from the general population.[8] In a randomized stratified sample from the general adult population of the province of Quebec, Canada, the prevalence of frequent jaw pain of moderate to severe intensity has recently been estimated to be approximately 5%; for frequent joint noises and difficulty in mouth opening, prevalence rates of 4% and 9%, respectively, were found.[8] While the highest prevalence of frequent TMD symptoms occurs between the ages of 18 and 45, a decrease in prevalence can be noted with increasing age in people older than age 45 years.[9] The study of Goulet et al[8] confirms these findings. In general, the prevalences of signs and symptoms of TMD are lower in children than in adults, and the younger a child, the lower the prevalence.[7] Finally, for all symptoms, the overall prevalence rate among women is about two times higher than that reported by men.[8] The review[7] of the epidemiology of TMD by Carlsson and LeResche is recommended for further reading.

Temporomandibular disorders represents several diagnostic subgroups with different clinical profiles that have some features in common.[4,10–14] Because a universally accepted and validated classification system for TMD has been lacking, it is difficult to interpret and compare many previous studies. To overcome this problem, an international project team proposed the Research Diagnostic Criteria (RDC), a dual-axis classification system that places myofascial pain, disc displacement, and the arthritides (ie, the physical diagnosis) on one axis, and an assessment of pain-related disability and psychologic status on a second axis.[15] According to Lund,[3] a major strength of the RDC is that they use operational definitions only and that they are not based on any etiologic theory. This makes it possible to use the RDC in a clinical setting without any bias in favor of one theory over another, thus maintaining a certain open-mindedness on the etiology of TMD. The validity of the RDC is currently being tested.

Basically, the cause of TMD is still unknown,[5] although most authors support a multifactorial etiology. An interplay of structural (occlusion, anatomy of TM joints and skeleton), psychologic, and functional (neuromuscular) factors is thought to be involved in the predisposition, initiation, and perpetuation of TMD.[4,16–19] In 1969, Laskin[20] proposed a psychophysiologic theory of TMD etiology based on the work of Schwartz,[21] and he suggests that muscle spasm is the primary factor responsible for the signs and symptoms of TMD. The most common cause for myospasm was believed to be muscle fatigue resulting from tooth grinding or clenching. Hence, a functional problem (ie, bruxism) caused an organic disease (ie, TMD). From there, Laskin[20] hypothesized that the "condition" would become self-perpetuating with abnormal patterns of muscle activity (altered chewing patterns) reinforcing the original myospasm and pain, thus setting up a chronic vicious cycle like the one proposed by Travell et al.[22] However, there is no experimental proof that the entire chain of events that constitutes the vicious cycle exists.[23] Actually, there is evidence that muscle pain does not cause hyperactivity in people.[24] Because no proof to support the vicious cycle model can be found in the clinical literature, some authors have challenged the validity of this model.[25,26]

Does Bruxism Represent Normal or Abnormal Orofacial Motor Behavior?

Before the role of bruxism in TMD can be assessed, some up-to-date insight into definition, epidemiology, etiology, and evaluation of bruxism is imperative. The American Academy of Orofacial Pain (AAOP) has defined bruxism as diurnal or nocturnal parafunctional activity including clenching, bracing, gnashing, and grinding of the teeth.[4] A more operational definition, offered by the American Sleep Disorders Association (ASDA), refers to bruxism as a periodic, stereotyped movement disorder of the masticatory system that involves tooth grinding or clenching during sleep.[27] Although this definition includes bruxism that occurs during daytime naps, it disregards other diurnal parafunctional activities. However, because of its operational nature, the ASDA definition is considered the best currently available description for both clinical and research purposes.[28,29]

Awareness of bruxism, with either grinding or clenching, is reported by 6% to 20% of adults. These figures are derived from epidemiologic surveys in student populations,[30] general dental practices,[31] and general populations.[32,33] In 11-year-old Quebec children, a prevalence of about 14% was found.[34] Widmalm et al[35] noted bruxism in about 20% of 4- to 6-year-old black and white children. The incidence of bruxism diminishes with age, especially after the age of 50.[33] Women report clenching about 22% more frequently than do men, but no gender predilection has been observed for grinding.[32]

Historically, occlusal interferences were considered major causes for bruxism by "triggering" parafunctional activity via a proprioceptive feedback mechanism.[36] However, Rugh et al[37] demonstrated that experimentally placed deflective occlusal contacts in bruxism patients tend to reduce masticatory muscle activity during sleep rather than to enhance it. In addition, it has been shown that occlusal adjustments do not stop bruxism.[38] Moreover, the occlusal relationships of bruxism patients do not differ significantly from those of asymptomatic control subjects.[39] In their review on occlusal therapy, Clark and Adler[40] concluded that no reliable evidence has been presented to demonstrate that occlusal interferences can cause bruxism. Nevertheless, the role of occlusion in bruxism is still debated.[41,42]

Besides dental occlusion, psychologic stress has been implicated in the etiology of bruxism.[43] Levels of bruxism were reported to increase with stress during a 31-day period in one patient.[44] In addition, if subjects are exposed to stressful experimental stimuli, an increased masticatory muscle activity is commonly found.[1] However, it is not clear how such experimentally induced muscle activity relates to bruxism. Moreover, Goulet et al[32] demonstrated only a weak association between reported life stress and bruxism awareness ($\gamma < .4$), while Pierce et al[45] found no overall relationship between electromyography (EMG) measures and self-reported stress in bruxers. Hence, the stress hypothesis needs further testing in future studies.

Current polysomnographic and clinical studies have linked sleep-related bruxism to the field of sleep disorders. Factors that indicate a lightening of sleep, such as electroencephalographic K-α complexes, rapid and transient muscle activities of legs and body, short increases in heart rate, and frequent sleep stage shifts were found in association with bruxism.[28,29,46–48] These observations are consistent with the hypothesis that bruxism is part of an arousal response.[27,49]

Finally, an altered brain chemistry (eg, an asymmetric nigrostriatal dopaminergic function) has recently been associated with bruxism.[50] More research is needed to establish the specificity of these latter findings.

One of the major confounding factors is how to diagnose bruxism. Numerous techniques are currently used for the diagnosis of bruxism.[29] One of them is the evaluation of dental attrition, either from direct visual observations in the mouth,[51] from occlusal appliances,[52] or from dental study casts.[53–55] However, since dental attrition should be considered a cumulative record of both functional and parafunctional wear, it does not provide evidence of current bruxism. Seligman and Pullinger[56] concluded that besides parafunctional activity, a significant part of observed wear is attributable to factors such as age and the geometry of the contact relationships of the dentition. In addition, tooth wear measures reflect contributions of tooth grinding only, and they disregard clenching activity. This suggests that tooth wear is a less reliable and valid measure of bruxism than would be desired. Techniques that use intraoral appliances to assess tooth wear are especially problematic because such devices may affect the behavior to be measured.[52,57,58]

The measurement of masticatory muscle activities by means of EMG in a sleep laboratory or in the patient's own home is another commonly used technique to evaluate bruxism.[27–29,44] Through EMG techniques, both grinding and clenching will be detected. In addition, EMG yields the identification of nonbruxism oromandibular motor activity, such as myoclonus, somniloquy, and tics.[59] However, this holds true only if audio and video recordings are obtained in parallel with all-night polygraphic recordings.[29,60] Such an experimental setup is almost impossible using an ambulatory (home) recording system, leaving the sleep laboratory as an expensive and time-consuming alternative. The advantages of using an ambulatory system, on the other hand, are the low costs, the direct monitoring in the natural environment, and a better patient compliance during long-term recordings.[44]

Polysomnographic observations made in the Center for Sleep Research of the Sacré-Coeur Hospital in Montreal indicate that so-called rhythmic masticatory muscle activities (RMMAs; phasic jaw muscle activities characterized by lower EMG activity than in bruxism and an absence of tooth grinding) are present in about 56% of the general sleep laboratory population.[28,47] Therefore, it is hypothesized that bruxism may represent basically normal orofacial motor behavior in which certain factors have strengthened and increased normal jaw-muscle activity, thus pushing it into a pathologic range.[28] Consequently, Lavigne et al[61] proposed the following polysomnographic cut-off criteria for a bruxism diagnosis:

1. At least 2 RMMA episodes with grinding sounds
2. One or more of the following: more than 4 RMMA episodes per hour of sleep; more than 25 RMMA bursts per hour of sleep; more than 6 RMMA bursts per episode

With these criteria, the clinically established presence or absence of bruxism was correctly predicted in 83% of bruxers and in 81% of asymptomatic control subjects, respectively. However, since bruxism is known to show a high night-to-night variability,[44] it is obvious that sleep laboratory or ambulatory recordings alone, ie, without additional information from measurement techniques such as self-report questionnaires, oral history taking, and clinical examination, cannot be considered on their own.

Cause-and-Effect Relationships Are Difficult to Establish

Feinstein[62] characterized the evaluation of cause-and-effect relationships as one of the most difficult challenges in biomedical research. Cause-and-effect relationships may increase our understanding of the etiology and pathogenesis of diseases in general. Therefore, insight into cause and effect is imperative for comprehension of the role that bruxism may or may not play in TMD.

Several different types of associations may be observed between two conditions.[63] Besides direct and indirect cause-and-effect relationships, the observed association between two conditions may result from an influence that should not be present, yielding a biased relationship. In addition, both conditions may coexist totally independently, which means that no relationship is actually present. Finally, the association between two conditions may be observed by chance. To establish which type of association best describes the relationship between bruxism and TMD, factors such as bias, chance, and confounding influences should be eliminated. Because this is not easy to accomplish, their presence may be left undetected. Therefore, it is not uncommon that previously accepted associations turn out to be flawed when new or different evaluation techniques are used.

It is impossible to establish a cause-and-effect relationship with 100% certainty. Therefore, a high degree of probability is sought. The probability of a cause-and-effect relationship strongly depends on the study design. Double-blind, randomized clinical trials are to be preferred to any other type of trial (eg, uncontrolled series of patients, case reports) because randomization and blinding eliminate most of the bias in any part of the trial, including the final assessment of causality.[63,64] In addition, a true association should be reproducible, ie, the finding should be consistent from trial to trial. The probability of validating a cause-and-effect relationship can be further increased by collecting data at multiple time points that are associated with the condition under examination. Since it is essential for the cause to precede the effect in a valid cause-and-effect relationship, single measurements are not sufficient because of their inability to demonstrate a temporal relationship between two conditions. The presence of a "dose-response gradient" (ie, a stronger cause leads to a greater effect) makes causality more probable as well. Furthermore, an association should make epidemiologic sense. The more specific an association (ie, the effect does not occur without the claimed cause having occurred previously), the more convincing the evidence for a cause-and-effect relationship. The criteria for establishing causation (modified from Spilker[63]) are summarized as follows:

1. Bias, chance, and confounding influences are absent.
2. The association should be consistent.
3. The cause must precede the effect.
4. A dose-response gradient is present.
5. The association should make epidemiologic sense.
6. The association must be specific.

The more criteria are met, the more convincing the evidence for causality.

Finally, it is important to realize that a single direct cause leading to an effect is exceptional in most clinical disorders.[63] Usually, multiple direct (proximate) and indirect (distant) causes are involved. Unfortunately, it is impossible to study more than a few direct and indirect causes in a single trial. Therefore, it is considered optimal to compare groups that are as similar as possible except in one or a few differences. These differences are the factors (causes) being studied. Alternatively, the factors under examination may be varied systematically with each other. If the effect changes in parallel with the cause, this is indicative of a cause-and-effect relationship. The former approach is referred to as the method of difference, whereas the latter is called the method of concomitant variation.[65]

The Role of Bruxism in TMD Still Needs to Be Defined

The belief that bruxism is involved in the predisposition, initiation, and perpetuation of TMD is commonly held, the more so because clinically defined bruxism is more prevalent in TMD patients

than in the general population.[20,66] Consequently, intense bruxism has been hypothesized to result in, among others, myofascial pain, TMJ noises (eg, clicking, crepitus), limitation of jaw movements, and tension-type headaches.[44]

Most evidence for the existence of a cause-and-effect relationship between bruxism and TMD is derived from epidemiologic surveys in both children and adult populations, in which positive correlations between reported awareness of parafunctional activities (eg, tooth grinding and clenching, lip/cheek biting, nail biting) and signs and symptoms of one or more subgroups of TMD have been found.[2,35,67–70]

Magnusson et al[2] stated that the positive correlation found in their study, which included up to three evaluations during a 10-year period, indicates the existence of a causal relationship between parafunctions and signs of TMD. However, since both bruxism and TMD are known to fluctuate over time at a rate faster than once every 5 years,[44,71] the number of evaluations in this study can hardly be considered sufficient to increase the probability of this statement (cause-and-effect criterion 3). The statement of Magnusson et al[2] is supported by Vanderas,[72] who concluded on the basis of a review of the literature on this subject that both the strength and the consistency of a finding of significant associations between parafunctions and dysfunction supports causality. Indeed, if an association is observed in multiple studies and under a variety of circumstances, the case for causality is strengthened (cause-and-effect criterion 2). However, more convincing evidence is needed to accept the bruxism-TMD association as a real cause-and-effect relationship.

Widmalm et al[35] are a bit more cautious about the interpretation of their results: they recognize that the presence of a significant association does not prove causation but that significant associations may point to possible common risk factors. Locker and Slade,[9] who found positive correlations between reported "hypothesized risk factors" such as bruxism and signs of TMD as well, stated that such findings need to be treated with caution, because a temporal sequence of cause and effect can never be established on the basis of a cross-sectional study design. Indeed, as mentioned in the previous section, data should be collected at multiple time points that respect the natural course of the disorders in determining whether the temporal relationship is correct (cause-and-effect criterion 3). Longitudinal epidemiologic surveys with a prospective design and a sufficient number of evaluations may be more suitable to determine the exact role of bruxism in the etiology of TMD (cause-and-effect criterion 1).

A major methodologic problem that makes the interpretation of epidemiologic surveys difficult is the use of self-report for bruxism.[43,73–75] Self-reports of any behavior have typically been unreliable[58,76]; at the least, they run the risk of bias toward either overreporting or underreporting.[73] In addition to the patient's awareness of the presence or absence of bruxism,[2] the clinician's support of the theory that bruxism plays a role in the etiology of TMD might influence patients' self-reports.[43,73] Therefore, it is important for researchers to determine how patients know that they brux.[74] When self-report measures are used with children, words familiar to the child should be used and parents should be asked to assist, especially for children younger than 6 years.[77] Preferably, however, multiple measurement techniques should be used to assess bruxism.

Droukas et al[78] reported positive correlations between reported awareness of bruxism and pain or fatigue in the face or jaws, but they found a negative correlation between bruxism and an impaired function of the TMJ in the same study. This suggests that at the least, different subgroups of TMD may relate differently to bruxism. In this context, it is appropriate to mention the recommendation of Clark[17] that research should focus on the correct identification and measurement of specific etiologic factors that produce a certain subdiagnosis of TMD.

It is significant to recognize that pain associated with bruxism is not a compulsory finding: many patients who appear to brux nightly have no masticatory muscle pain at all.[26,79,80] This implies that the degree of specificity of the association between bruxism and muscle pain is low, which reduces the probability of establishing a valid cause-and-effect relationship (cause-and-effect criterion 6). As an alternative, it is suggested that pain associated with bruxism may be a form of postexercise muscle soreness (PEMS), a condition characterized by muscle fiber microtrauma induced by an excessive loading, and by pain and dysfunction that develop gradually over several hours following the exercise.[25,26] Indeed, Christensen[81] demonstrated the occurrence of muscle pain that peaked about 2 hours after the completion of a 30-minute experimental (ie, purposeful) tooth grinding task; during the days following experimental jaw muscle contraction, no significantly increased pain levels were found to be present.[82] In addition, support for Lund's suggestion can be found in a recent study by Hutchins et al,[83] who showed that contraction

injuries can be induced in the masticatory muscles of mice by "forced lengthening contraction," a technique that simulates the eccentric contraction pattern of parafunctional activities. The PEMS hypothesis is further supported by the observation that 83.3% of bruxers with associated pain reported that their pain was worst upon awakening in the morning, ie, following the exercise.[80] In contrast, only 19.7% of myofascial pain patients without any clinical evidence or report of bruxism had their highest pain in the morning.[80] This indicates that pain associated with bruxism and with myofascial pain may be two different entities.

Interestingly, it has recently been demonstrated that the number of bruxism episodes per hour of sleep is lower in bruxers with reports of concomitant localized jaw-muscle pain than in those without pain.[84] In one study,[84] 66.7% of bruxers with pain reported their highest level of pain intensity in the morning, which is in accordance with the aforementioned findings of Dao et al.[80] On average, however, a nonsignificant increase in pain intensity of only 19.9% overnight was found in the bruxers with associated pain. This suggests that bruxism may not be the primary cause of jaw-muscle pain and, by inference, that pain modulates the pattern of bruxism by reducing its hourly number of episodes. It remains to be demonstrated whether this modulation is because of the masticatory muscle pain per se or secondary to a putative influence of pain on sleep.

A final piece of evidence that reduces the probability of a valid cause-and-effect relationship between bruxism and TMD is the difference between some epidemiologic characteristics of both disorders. While the prevalence of bruxism is high in children and decreases into adulthood, the prevalence of TMD is low in children and in people older than age 45 years and peaks between the ages of 18 and 45. In addition, while both conditions are reported more frequently by women than by men, this finding is much more prominent for TMD than for bruxism. In other words, the association does not make epidemiologic sense, although this is one of the criteria for establishing a cause-and-effect relationship (cause-and-effect criterion 5).

Conclusions and Recommendations for Future Research

Although many epidemiologic surveys indicate positive correlations between bruxism and TMD, they should be interpreted with caution because causality is extremely difficult to establish. Bias, chance, and confounding influences may lead to faulty conclusions. In addition, it is probable that multiple direct and indirect causes are involved in TMD. Thus, a simple, direct causal relationship is likely to result in an incomplete picture of the etiology of TMD. Longitudinal epidemiologic surveys and clinical/experimental studies designed to establish or refute a cause-and-effect relationship seem more appropriate to identify if bruxism (and other direct and indirect factors) may actually produce TMD. When such a design is adopted, the existence of diagnostic subgroups of TMD should be taken into account because different subgroups may relate differently to bruxism. In addition, it is important to establish the presence of bruxism along multiple axes: any single, currently used method to assess bruxism (eg, self-report questionnaires, sleep laboratory recordings) is difficult to consider on its own. The use of polysomnographic cut-off criteria to discriminate bruxism from "normal" RMMAs should be an integral part of the diagnostic procedure for sleep bruxism. Since daytime clenching shows a stronger association with TM pain than does sleep-related grinding,[85] sleep bruxism should be discriminated from its daytime variant.

It can be concluded that the nature of a putative functional relationship between bruxism and TMD is not clear at present. As yet, it is not at all unlikely that bruxism and TMD are simply coexisting entities in a considerable number of patients. As stated by Marbach,[74] any treatment of TMD assuming bruxism as an intermediate variable remains speculative.

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Resumen

Tienen el Bruxismo y los Desórdenes Temporomandibulares una Relación de Causa y Efecto?

La controversia sobre el papel putativo del bruxismo en la etiología de los desórdenes temporomandibulares (DTM) continúa. Una idea que se considera comúnmente es el que el bruxismo conduce a signos y síntomas característicos de uno o mas de los subdiagnósticos de DTM, mientras que otra hipótesis indica que el bruxismo es en sí un DTM que algunas veces coexiste con otros tipos de DTM. Luego de una revisión minuciosa de la literatura en este artículo, se concluye que la relación entre el bruxismo y los DTM no es clara todavía. La investigación futura debería examinar la epidemiología longitudinal y los datos clínicos y experimentales para establecer o refutar una relación de causa y efecto. Al hacer esto, se debería tener en cuenta, la existencia de varios subgrupos de DTM, y el bruxismo relacionado al sueño debería diferenciarse de su variante diurna.

Zusammenfassung

Haben Bruxismus und temporomandibuläre Erkrankungen eine Ursache-und-Wirkung Beziehung?

Kotroversen bleiben bestehen über die vermeintliche Rolle von Bruxismus in der Aetiologie von temporomandibulären Erkrankungen. Ein gemeinhin aufrechterhaltenes Konzept ist, dass Bruxismus zu Zeichen und Symptomen führt, welche charakteristisch sind für eine oder mehrere Subdiagnosen von temporomandibulären Erkrankungen, während eine andere Hypothese nahelegt, dass Bruxismus selbst eine temporomandibuläre Erkrankung darstellt und manchmal zusammen vorkommt mit anderen Formen von temporomandibulären Erkrankungen. Gemäss einem gründlichen Literaturrückblick in diesem Artikel wird gefolgert, dass die Beziehung zwischen Bruxismus und temporomandibulären Erkrankungen immer noch unklar ist. Zukünftige Forschung sollte longitudinale epidemiologische und klinische/experimentelle Daten untersuchen, um eine Ursachen-und-Wirkung Beziehung zu bestätigen oder zu verweisen. Dabei sollte die Existenz von verschiedenen Untergruppen der temporomandibulären Erkrankungen in Betracht gezogen werden, und schlafverbundener Bruxismus sollte von der Variante tagsüber unterschieden werden.