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Review
Bruxism is mainly regulated centrally, not peripherally

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SUMMARY **Bruxism is a controversial phenomenon. Both its definition and the diagnostic procedure contribute to the fact that the literature about the aetiology of this disorder is difficult to interpret. There is, however, consensus about the multifactorial nature of the aetiology. Besides peripheral (morphological) factors, central (pathophysiological and psychological) factors can be distinguished. In the past, morphological factors, like occlusal discrepancies and the anatomy of the bony structures of the orofacial region, have been considered the main causative factors for bruxism. Nowadays, these factors play only a small role, if any. Recent focus is more on the pathophysiological factors. For example, bruxism has been suggested to be part of a sleep arousal response. In addition, bruxism appears to be modulated by various neurotransmitters in the central nervous system. More specifically, disturbances in the central dopaminergic system have been linked to bruxism. Further, factors like smoking, alcohol, drugs, diseases and trauma may be involved in the bruxism aetiology. Psychological factors like stress and personality are frequently mentioned in relation to bruxism as well. However, research to these factors comes to equivocal results and needs further attention. Taken all evidence together, bruxism appears to be mainly regulated centrally, not peripherally.**

KEYWORDS: bruxism, aetiology, morphology, pathophysiology, psychology, review

Introduction

The number of studies that aim to unravel the mechanisms behind bruxism is increasing rapidly. Nevertheless, this clinical problem (Lavigne *et al.*, 1999) remains difficult to grasp and controversial. For example, there is still no agreement regarding the definition and diagnosis of bruxism (Lavigne & Manzini, 2000). As a consequence, the available articles on the aetiology of bruxism are difficult to compare and therefore hard to interpret unequivocally. The interpretation of the literature is further hampered by the fact that the effects of factors like gender and race on the prevalence of the disorder are not yet clarified (Lavigne *et al.*, 1995). This makes the generalization of certain findings difficult. Moreover, insight into study design has improved considerably during the last decade. For instance, control subjects are frequently absent in previous studies and regularly, the phenomenon of interest, bruxism, was quantified with indirect measures.

Notwithstanding these difficulties, many aetiological theories to explain bruxism have been formulated over the years. Although these theories are hard to confirm or refute because of the controversial character of the disorder, most suggest a multifactorial aetiology (Attanasio, 1997; Lobbezoo & Lavigne, 1997; Bader & Lavigne, 2000; Lavigne & Manzini, 2000). Basically, two groups of etiological factors can be distinguished, viz., peripheral (morphological) factors and central (pathophysiological and psychological) factors. Below, both groups will be reviewed and the current view on their relative contribution to the aetiology of bruxism will be discussed.

Some authors stress the importance to discriminate between sleep-related bruxism and bruxism during wakefulness, because both types may have a different aetiology (Ramfjord, 1961; Glaros, 1981; Rugh & Harlan, 1988). In this review, bruxism will be considered the combination of all parafunctional clenching and grinding activities, exerted both during sleep and

while awake, because as yet both phenomena are not or only inadequately distinguished in most related articles. However, most data regarding the aetiology of bruxism come from studies to sleep-related bruxism, because this type is better suited for a reliable diagnosis in a scientific research setting (polysomnography; see Lavigne & Manzini, 2000).

The purpose of this paper is to review the literature on the aetiology of bruxism so as to establish which group of etiological factors, viz., peripheral or central ones, can be considered most implicated in the disorder. Insight into the aetiology of bruxism is also clinically important, because it enables the clinician to choose a treatment that might influence or even eliminate one or more of the factors that perpetuate the disorder.

Morphological factors

Within the group of morphological factors, anomalies in dental occlusion and articulation as well as in the (bony) anatomy of the oro-facial region can be distinguished. Seen in the historical perspective reviewed below, these factors were formerly considered the most important initiating and perpetuating aetiological factors for bruxism. More recently, the role of occlusal-anatomical factors is believed to be much smaller, if at all present. This shift is illustrated by the fact that the term 'occlusal disturbance' is more and more being replaced by the term 'occlusal characteristic'. A disturbance usually indicates that something needs to be corrected, while a characteristic only suggests that something more or less noteworthy is present without there being a need for any corrections whatsoever. Nowadays, there is growing agreement upon the insight that it is not really important as to how someone's occlusion looks like, but that it matters how one 'copes' with a certain type of occlusion. It's the dental profession's task to prevent the creation of 'occlusal neurotics', which might easily occur when occlusal adjustment procedures are even discussed with the patient (Greene *et al.*, 1998).

A frequently cited study regarding bruxism is the classical one by Ramfjord (1961) in which for one of the first times, the clinical phenomenon 'bruxism' is being studied with electromyographic (EMG) techniques. Although Ramfjord also saw a role for 'neurotic tensions' in the aetiology of bruxism, he held certain occlusal characteristics mainly responsible for the initiation of the disorder. Especially discrepancies between retruded contact position and intercuspal position, and also the presence of mediotrusive (balancing side) contacts during articulation were thought to be involved in the aetiology of bruxism. Ramfjord reported that occlusal adjustments (grinding procedures) always led to a disappearance of bruxism. The presence or absence of bruxism was determined during a 45–60 min EMG protocol, during which the patient performed a couple of movement tasks. It is, however, doubtful whether these tasks were indicative of bruxism, because no direct measures of actual parafunctional activities were performed. Ramfjord supported his results with his own (unpublished) observations in rhesus monkeys, which only stopped their bruxism activities when they completed the elimination of the overfilled part of a restoration in the first molar. Bruxism would thus be an instrument, with which an individual tries to eliminate occlusal interferences. The interferences were believed to cause a reflexly mediated excitation of the jaw closing muscles through the stimulation of periodontal mechanoreceptors.

Although in Ramfjord's EMG study (1961) no controls were included, and although the use of indirect measures for bruxism renders the results impossible to interpret according to more recent insights into study design, the conclusions of this study have had a major impact on clinical dentistry for many decades. Research is still going on as to the role of occlusion and articulation in the aetiology of bruxism (e.g. Yustin *et al.*, 1993), although these factors are already being put aside by the outcomes of better controlled studies. For example, Rugh *et al.* (1984) studied the influences of artificial occlusal interferences, incorporated in crowns in the molar region, on masticatory muscle activity (MMA) during sleep. The MMA was quantified by means of EMG recordings from the sleeping patient. In contrast with the findings of Ramfjord (1961), artificial interferences caused a significant decrease of sleep-related MMA in 90% of the cases. This result sheds some serious doubt on the role of occlusion in the aetiology of bruxism. The other side of the medal is, that artificial interferences cannot directly be compared with natural ones, these latter possibly being the effect of bruxism rather than its cause.

In better controlled studies, the elimination of interferences in occlusion and articulation was shown to have no influence on bruxism activities (Kardachi *et al.*, 1978; Bailey & Rugh, 1980). Moreover, not every bruxer has occlusal interferences and not every person with such interferences is a bruxer (Greene & Marbach,

1982). Therefore, the conclusion is justified that although occlusal schemes are relevant to the distribution of the forces that go with bruxism activities (Rugh & Harlan, 1988), there is no scientific proof for a role of occlusion and articulation in the aetiology of bruxism (Clark & Adler, 1985).

Two recent studies have examined the possible relationship between bruxism and the anatomy of the oro-facial region. Miller *et al*. (1998) found a more pronounced asymmetry in condylar height in bruxers as compared with non-bruxers; Young *et al*. (1999) observed larger bizygomatic and cranial widths in bruxers. In neither of these studies, however, the presence or absence of bruxism, as assessed by self-report and a clinical examination, was confirmed polysomnographically, which hampers the interpretation of their results. The absence of a polysomnographic confirmation of the (non) bruxer status also hampers the interpretation of the study by Menapace *et al*. (1994), although these authors found no differences in the dentofacial morphology between bruxers and non-bruxers. In another study that primarily focused on tooth wear in relation to the morphology of the craniofacial structures (Waltimo *et al*., 1994), a more rectangular form of the maxillary dental arch was found in patients with severe dental attrition than in control subjects. In addition, these authors found that patients with severe attrition had a more rectangular facial morphology than controls, in combination with an anteriorly rotated mandible, a small anterior facial height and a large bimaxillary interincisal angle. Again, unfortunately, Waltimo *et al*. (1994) did not use polysomnography to classify their patients.

So far, only one controlled study to the relationship between bruxism and morphological factors was performed with the use of polysomnography to confirm or refute the presence of bruxism. In that study, Lobbezoo *et al*. (2001a) compared 26 occlusal variables and 25 cephalometric variables between bruxers and non-bruxers and found no differences between both groups. As for the interferences in dental occlusion and articulation, we therefore conclude that there is no proof for a role of factors related to the anatomy of the oro-facial skeleton in the aetiology of bruxism.

Pathophysiological factors

More and more, pathophysiological factors are suggested to be involved in the precipitation of bruxism (Bader & Lavigne, 2000; Lavigne & Manzini, 2000). For example, bruxism has been linked to sleep disturbances, an altered brain chemistry, the use of certain medications and illicit drugs, smoking, the consumption of alcohol, and certain traumata and diseases. In this review, genetic factors are included among the pathophysiological factors as well.

As bruxism often occurs during sleep, the physiology of sleep has been studied extensively in the search for possible causes of the disorder. Especially the so-called 'arousal response' has been the subject of many studies (e.g. Wrubble *et al*., 1989). An arousal response is a sudden change in the depth of sleep, during which the individual either arrives in a lighter sleep stage or actually wakes up (Thorpy, 1990a). Such a response is accompanied by gross body movements (e.g. turning), the appearance of K complexes in the electroencephalograph (EEG) (single, biphasic potentials with a relatively large amplitude), an increased heart rate, respiratory changes, peripheral vasoconstrictions and increased muscle activities. Recently, Macaluso *et al*. (1998) showed that in 86% of cases, bruxism episodes were associated with an arousal response. Besides the above-mentioned characteristics of an arousal response, involuntary leg movements were present in association with about 80% of the bruxism episodes. These observations suggest that bruxism is part of an arousal response indeed. Therefore, bruxism can be classified among the parasomnias, a group of sleep disturbances that also includes sleepwalking, nightmares, sleep talking and enuresis (Thorpy, 1990b).

From a recent series of papers, it can be derived that certain disturbances in the central neurotransmitter system may be involved in the aetiology of bruxism (Lobbezoo *et al*., 1996, 1997a,b). From these papers, it can be hypothesized that the balance between the direct and indirect pathways of the basal ganglia, a group of five subcortical nuclei that are involved in the coordination of movements, is disturbed in bruxers. The direct output pathway goes directly from the striatum (one of the five basal ganglia) to the thalamus, from where afferent signals project to the cerebral cortex. The indirect pathway, on the other hand, passes by several other nuclei before the thalamus is being reached. If there is an imbalance between both pathways, movement disorders are the result, like Parkinson's disease (Strange, 1993). The cause of such an imbalance can be found in the so-called 'nigrostriatal projection', a feedback loop within the complex of

nuclei that constitute the basal ganglia. The imbalance goes with disturbances in the dopamine-mediated transmission of action potentials. In case of actual nigrostiatal degeneration, Parkinson's disease emerges because of a lack of endogenous dopamine, which can be influenced by pharmacological therapy (e.g. dopamine precursors, dopamine agonists). In case of bruxism, there may be an imbalance between both output pathways as well, however, without signs of degeneration of the nigrostriatal feedback loop. The acute (short-term) use of L-dopa, a dopamine precursor (Lobbezoo *et al*., 1997a), and of bromocriptine, a D2 receptor agonist (Lobbezoo *et al*., 1997b), inhibits bruxism activity in controlled polysomnographical studies. The chronic (long-term) use of L-dopa by Parkinson patients is known to cause bruxism (Magee, 1970). Similarly, the chronic use of neuroleptics by psychiatric patients gives rise to bruxism during wakefulness (Micheli *et al*., 1993). Also medications that exert an indirect influence on the dopaminergic system, like selective serotonine reuptake inhibitors (SSRIs; for a review, see Lobbezoo *et al*., 2001b), may cause bruxism after long-term usage. This is corroborated by the observation (Amir *et al*., 1997) – the β-blocker propranolol relieves bruxism secondary to antipsychotic drug exposure in two cases. It appears that there may be two types of bruxism: an idiopathic type that can be suppressed by a short-term treatment with dopamine agonists, and an iatrogenic type that is caused by the long-term application of several dopaminergical medicines. The only study so far that complicates this view is a recent one by Lavigne *et al*. (2001), in which bruxism could not be influenced by the acute use of bromocriptine. A possible explanation for this deviant finding may be the fact that bromocriptine was combined with domperidone to suppress peripheral side-effects.

Tooth grinding that can be observed in relation to the abuse of amphetamine (Ashcroft *et al*., 1965) – a substance that increases the dopamine concentration by facilitating its release – can be classified as iatrogenic bruxism in association with dopamine as well. The drug XTC, an amphetamine-like substance, has recently been associated with excessive tooth wear (Milosevic *et al*., 1999). Also nicotine stimulates the central dopaminergic activities, which might explain the finding that smokers of cigarettes report bruxism almost two times more than non-smokers (Lavigne *et al*., 1997; Madrid *et al*., 1998) and that smokers show about five times more bruxism episodes per night than non-smokers (Lavigne *et al*., 1997). Hartman (1994) found that alcohol could lead to bruxism; more than four consumptions per day would then be necessary. Also this observation fits well into the view that bruxism is mainly a centrally mediated disorder.

In an attempt to establish a link between the role of occlusal disharmonies and that of alterations in central dopaminergic neurotransmission in experimentally induced bruxism in the rat, Gomez *et al*. (1998) and Areso *et al*. (1999) found that an acrylic cap on the mandibular central incisors, that was worn for a prolonged period of time, resulted in an imbalance between hemispheres in dopa accumulation in the basal ganglia. However, Lobbezoo *et al*. (2001a) were not able demonstrate a similar phenomenon in man: no significant correlations were found between morphological factors and asymmetries in striatal D2 receptor expression in a group of patients with polysomnographically confirmed sleep-related bruxism. It should be stated, however, that it is difficult to compare artificial occlusal interferences in experimentally bruxing rats with 'natural' ones in patients with sleep-related bruxism.

Many clinicians have the impression that bruxism runs in families. Hublin *et al*. (1998) demonstrated in a large-scale questionnaire study with about 4000 twin pairs that the contribution of heredity to bruxism varies from 39 to 64%. In contrast, Michalowicz *et al*. (2000) concluded on the basis of a combined questionnaire – clinical study with almost 250 pairs of twins, that there is no such contribution. Hence, whether or not bruxism is more or less genetically determined remains unclear.

Bruxism has been described as part of a number of neurological and psychiatric disorders as well as a consequence of certain traumata. These aspects are reviewed extensively by Lavigne and Montplaisir (1995).

On the basis of the above overview, it can be concluded that bruxism can be associated with several pathophysiological factors.

Psychological factors

As mentioned before, Ramfjord (1961) already saw a role for 'neurotic tensions' in the aetiology of bruxism. Besides, in this classical study, stress and personality have been already implicated in the aetiology of bruxism for many years. However, the exact contribution of these (and other) psychological factors to this

aetiology remains a subject of debate. A major problem is the fact that psychological factors are difficult to make operational. The combination of such factors with an equivocally defined disorder like bruxism (see above) obviously yields a difficult area of research. Nevertheless, a large number of studies have been performed to possible interactions between psychological factors and bruxism, mostly using questionnaires.

A controlled questionnaire study by Olkinuora (1972) demonstrated that bruxers can be considered emotionally out of balance and that they tend to develop more psychosomatic disorders. Their personalities would be characterized by perfectionism and an increased tendency towards anger and aggression. These findings were later confirmed by Kampe *et al.* (1997), who also demonstrated more anxiety in a group of bruxers. The use of a specific questionnaire for the amount of psychological disturbance, the Minnesota Multiphasic Personality Inventory (MMPI), however, indicated that bruxers do not differ from non-bruxers within a population of patients with facial pain (Harness & Peltier, 1992). Interestingly, an increased amount of aggression and somatization can already be found in bruxing 5- and 6-year olds (Kuch *et al.*, 1979).

Only rarely, EMG or polysomnography has been used in the study as to the role of psychological factors in the aetiology of bruxism. A unique 'EMG case' was described by Rugh and Robbins (1982). During a 6-month period, they continuously recorded the masticatory EMG activity of a young woman. In times of increased stress caused by, for example, exams and fights with her partner, she developed an increase in her sleep-related MMA. Although interesting, it is still a case, and this relationship is less obvious in groups of bruxers (see below).

An important study to the possible relation between stress and bruxism is by Pierce *et al.* (1995). These authors investigated in 100 bruxers the amount of self-reported stress in relation to electromyographically recorded bruxism during the night before the stress report (anticipatory stress) and the night following the report (current stress). A total of 15 nights was recorded. For both anticipatory stress and current stress, significant (positive) correlations with bruxism were found in eight individuals only. For the entire sample, no association between stress and bruxism could be demonstrated. Similarly, Goulet *et al.* (1993) found only a weak correlation between self-reported stress and bruxism.

Finally, in a controlled polysomnographical study to vigilance and reaction time, an increased level of anxiety was found in sleep bruxers (Major *et al.*, 1999). Anxiety was the only psychological outcome variable to reach statistical significance in this study.

In short, the role of psychological factors in the aetiology of bruxism is far from clear. It appears that this role differs between individuals and is probably smaller than previously assumed. Clearly, there is a need for more controlled studies to this subject, in which the susceptibility of an individual to psychological factors should be taken into account (Hartmann, 1994).

The bruxism generator model

From epidemiological studies, we know that rhythmic, sleep-related MMA occur in almost 60% of the adult population (Lobbezoo & Lavigne, 1997). The percentage of people who actually fulfil the cut-off criteria for a sleep bruxism diagnosis (Lavigne *et al.*, 1996) is considerably smaller. Hence, rhythmic MMAs may be considered normal sleep-related motor behaviour. Possibly, one or more of the factors described in the above overview contribute to an increase in frequency, duration and intensity of these muscle activities, thus enabling a diagnosis of sleep-related bruxism. This hypothesis was formulated by Lavigne and Montplaisir (1995) and is known as the bruxism generator model. This model fits nicely with the proposed multifactorial aetiology of bruxism (see Introduction).

Conclusion

Based on the evidence reviewed above, it seems that bruxism has a multifactorial aetiology indeed. There is strong evidence that the role of occlusal characteristics and other morphological factors is small, if at all present. There is convincing evidence, however, that (sleep-related) bruxism is part of an arousal response. Disturbances in the central dopaminergic system are implicated in the aetiology of bruxism as well. Further, there is a role for factors like smoking, alcohol, diseases, trauma and heredity, while the proposed role of stress and other psychological factors is probably smaller than hitherto assumed. In short, bruxism is mainly centrally mediated, not peripherally.

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References

Amir, I., Hermesh, H. & Gavish, A. (1997) Bruxism secondary to antipsychotic drug exposure: a positive response to propranolol. *Clinical Neuropharmacology*, **20,** 86.

Areso, M.P., Giralt, M.T., Sainz, B., Prieto, M., Garcia-Vallejo, P. & Gomez, F.M. (1999) Occlusal disharmonies modulate central catecholaminergic activity in the rat. *Journal of Dental Research*, **78,** 1204.

Ashcroft, G.W., Eccleston, D. & Waddell, J.L. (1965) Recognition of amphetamine addicts. *British Medical Journal*, **1,** 57.

Attanasio, R. (1997) An overview of bruxism and its management. *The Dental Clinics of North America*, **41,** 229.

Bader, G. & Lavigne, G.J. (2000) Sleep bruxism: an overview of an oromandibular sleep movement disorder. *Sleep Medicine Reviews*, **4,** 27.

Bailey, J.O. & Rugh, J.D. (1980) Effect of occlusal adjustment on bruxism as monitored by nocturnal EMG recordings. *Journal of Dental Research*, **59,** 317.

Clark, G.T. & Adler, R.C. (1985) A critical evaluation of occlusal therapy: occlusal adjustment procedures. *Journal of the American Dental Association*, **110,** 743.

Glaros, A. (1981) Incidence of diurnal and nocturnal bruxism. *Journal of Prosthetic Dentistry*, **45,** 545.

Gomez, F.M., Areso, M.P., Giralt, M.T., Sainz, B. & Garcia-Vallejo, P. (1998) Effects of dopaminergic drugs, occlusal disharmonies and chronic stress on non-functional masticatory activity in the rat assessed by incisal attrition. *Journal of Dental Research*, **77,** 1454.

Goulet, J.P., Lund, J.P., Montplaisir, J.Y. & Lavigne, G.J. (1993) Daily clenching, nocturnal bruxism, and stress and their association with TMD symptoms. *Journal of Orofacial Pain*, **7,** 120.

Greene, C.S. & Marbach, J.J. (1982) Epidemiologic studies of mandibular dysfunction: a critical review. *Journal of Prosthetic Dentistry*, **48,** 184.

Greene, C.S., Mohl, N.D., McNeill, C., Clark, G.T. & Truelove, E.L. (1998) Temporomandibular disorders and science: a response to the critics. *Journal of Prosthetic Dentistry*, **80,** 214.

Harness, D.M. & Peltier, B. (1992) Comparison of MMPI scores with self-report of sleep disturbance and bruxism in the facial pain population. *Journal of Craniomandibular Practice*, **10,** 70.

Hartmann, E. (1994) Bruxism. In: *Principles and Practice of Sleep Medicine* (eds M. H. Kryger, T. Roth & W. C. Dement), p. 598. W.B. Saunders Co., Philadelphia.

Hublin, C., Kaprio, J., Partinen, M. & Koskenvuo, M. (1998) Sleep bruxism based on self-report in a nationwide twin cohort. *Journal of Sleep Research*, **7,** 61.

Kampe, T., Edman, G., Bader, G., Tagdae, T. & Karlsson, S. (1997) Personality traits in a group of subjects with long-standing bruxing behaviour. *Journal of Oral Rehabilitation*, **24,** 588.

Kardachi, B.J.R., Bailey, J.O. & Ash, M.M. (1978) A comparison of biofeedback and occlusal adjustment on bruxism. *Journal of Periodontology*, **49,** 367.

Kuch, E.V., Till, M.J. & Messer, L.B. (1979) Bruxing and non-bruxing children: a comparison of their personality traits. *Pediatric Dentisrty*, **1,** 182.

Lavigne, G.J. & Manzini, C. (2000) Bruxism. In: *Principles and Practice of Sleep Medicine* (eds M. H. Kryger, T. Roth & W. C. Dement), p. 773. W.B. Saunders Co, Philadelphia.

Lavigne, G.J. & Montplaisir, J.Y. (1995) Bruxism: epidemiology, diagnosis, pathophysiology, and pharmacology. *Advances in Pain Research and Therapy*, **21,** 387.

Lavigne, G.J., Lobbezoo, F. & Montplaisir, J.Y. (1995) The genesis of rhythmic masticatory muscle activity and bruxism during sleep. In: *Brain and Oral Functions* (eds T. Morimoto, T. Matsuya & K. Takada), p. 249. Elsevier Science, Amsterdam.

Lavigne, G.J., Rompré, P.H. & Montplaisir, J.Y. (1996) Sleep bruxism: validity of clinical research diagnostic criteria in a controlled polysomnographic study. *Journal of Dental Research*, **75,** 546.

Lavigne, G.J., Lobbezoo, F., Rompré, P.H., Nielsen, T.A. & Montplaisir, J.Y. (1997) Cigarette smoking as a risk or exacerbating factor for restless legs syndrome and sleep bruxism. *Sleep*, **20,** 290.

Lavigne, G.J., Goulet, J.P., Zuconni, M., Morisson, F. & Lobbezoo. F. (1999) Sleep disorders and the dental patient. *Oral Surgery Oral Medicine Oral Pathology Oral Radiology and Endodontics*, **88,** 257.

Lavigne, G.J., Soucy, J.P., Lobbezoo, F., Manzini, C., Blanchet, P.J. & Montplaisir, J.Y. (2001) Double-Blind, crossover, placebo-controlled trial of bromocriptine in patients with sleep bruxism. *Clinical Neuropharmacology*, **24,** 145.

Lobbezoo, F. & Lavigne, G.J. (1997) Do bruxism and temporomandibular disorders have a cause-and-effect relationship? *Journal of Orofacial Pain*, **11,** 15.

Lobbezoo, F., Soucy, J.P., Montplaisir, J.Y. & Lavigne, G.J. (1996) Striatal D2 receptor binding in sleep bruxism: a controlled study with iodine-123-iodobenzamide and single photon emission computed tomography. *Journal of Dental Research*, **75,** 1804.

Lobbezoo, F., Lavigne, G.J., Tanguay, R. & Montplaisir, J.Y. (1997a) The effect of the catecholamine precursor L-dopa on sleep bruxism: a controlled clinical trial. *Movement Disorders*, **12,** 73.

Lobbezoo, F., Soucy, J.P., Hartman, N.G., Montplaisir, J.Y. & Lavigne, G.J. (1997b) Effects of the dopamine D2 receptor agonist bromocriptine on sleep bruxism: report of two single-patient clinical trials. *Journal of Dental Research*, **76,** 1611.

Lobbezoo, F., Rompré, P.H., Soucy, J.P., Iafrancesco, C., Turkewicz, J., Montplaisir, J.Y. & Lavigne, G.J. (2001a) Lack

of associations between occlusal and cephalometric measures, side imbalance in striatal D2 receptor binding, and sleep-related oromotor activities. *Journal of Orofacial Pain*, **15,** 64.

Lobbezoo, F., Van Denderen, R.J.A., Verheij, J.G.C. & Naeije, M. (2001b) Reports of SSRI-associated bruxism in the family physician's office. *Journal of Orofacial Pain*, (in press).

Macaluso, G.M., Guerra, P., Di Giovanni, G., Boselli, M., Parrino, L. & Terzano, M.G. (1998) Sleep bruxism is a disorder related to periodic arousals during sleep. *Journal of Dental Research*, **77,** 565.

Madrid, S., Vranesh, J.G. & Hicks, R.A. (1998) Cigarette smoking and bruxism. *Perceptual and Motor Skills*, **87,** 898.

Magee, K.R. (1970) Bruxism related to levodopa therapy. *Journal of the American Medical Association*, **214,** 147.

Major, M., Rompré, P.H., Guitard, F., Tenbokum, L., O'Connor, K., Nielsen, T. & Lavigne, G.J. (1999) A controlled daytime challenge of motor performance and vigilance in sleep bruxers. *Journal of Dental Research*, **78,** 1754.

Menapace, S.E., Rinchuse, D.J., Zullo, T., Pierce, C.J. & Shnorhokian, H. (1994) The dentofacial morphology of bruxers versus non-bruxers. *Angle Orthodontist*, **64,** 43.

Michalowicz, B.S., Pihlstrom, B.L., Hodges, J.S. & Bouchard, T.J. Jr (2000) No heritability of temporomandibular joint signs and symptoms. *Journal of Dental Research*, **79,** 1573.

Micheli, F., Pardal, M.F., Gatto, M., Asconapé, J., Giannaula, R. & Parera, I.C. (1993) Bruxism secondary to chronic antidopaminergic drug exposure. *Clinical Neuropharmacology*, **16,** 315.

Miller, V.J., Yoeli, Z., Barnea, E. & Zeltser, C. (1998) The effect of parafunction on condylar asymmetry in patients with temporomandibular disorders. *Journal of Oral Rehabilitation*, **25,** 721.

Milosevic, A., Agrawal, N., Redfearn, P.J. & Mair, L.H. (1999) The occurrence of toothwear in users of Ecstasy (3,4 MethyleneDioxyMethAmphetamine). *Community Dentistry Oral Epidemiology*, **27,** 283.

Olkinuora, M. (1972) Psychosocial aspects in a series of bruxists compared with a group of non-bruxists. *Proceedings of the Finnish Dental Society*, **68,** 200.

Pierce, C.J., Chrisman, K., Bennett, M.E. & Close, J.M. (1995) Stress, anticipatory stress, and psychologic measures related to sleep bruxism. *Journal of Orofacial Pain*, **9,** 51.

Ramfjord, S.P. (1961) Bruxism, a clinical and electromyographic study. *Journal of the American Dental Association*, **62,** 21.

Rugh, J.D. & Harlan, J. (1988) Nocturnal bruxism and temporomandibular disorders. *Advances in Neurology*, **49,** 329.

Rugh, J.D. & Robbins, J.W. (1982) Oral habit disorders. In: *Behavioral Aspects in Dentistry* (ed. B. D. Ingersoll), p. 179. Appleton-Century-Crofts, New York.

Rugh, J.D., Barghi, N. & Drago, C.J. (1984) Experimental occlusal discrepancies and nocturnal bruxism. *Journal of Prosthetic Dentistry*, **51,** 548.

Strange, P.G. (1993) Dopamine receptors in the basal ganglia: relevance to Parkinson's disease. *Movement Disorders*, **8,** 263.

Thorpy, M.J. (1990a) Glossary of terms used in sleep disorders medicine. In: *Handbook of Sleep Disorders* (ed. M. J. Thorpy), p. 779. Marcel Dekker, New York.

Thorpy, M.J. (1990b) Parasomnias. In: *International Classification of Sleep Disorders: Diagnostic and Coding Manual* (ed. M. J. Thorpy), p. 142. Allen Press, Rochester.

Waltimo, A., Nyström, M. & Könönen, M. (1994) Bite force and dentofacial morphology in men with severe dental attrition. *Scandinavian Journal of Dental Research*, **102,** 92.

Wrubble, M.K., Lumley, M.A. & McGlynn, F.D. (1989) Sleep-related bruxism and sleep variables: a critical review. *Journal of Craniomandibular Disorders: Facial and Oral Pain*, **3,** 152.

Young, D.V., Rinchuse, D.J., Pierce, C.J. & Zullo, T. (1999) The craniofacial morphology of bruxers versus nonbruxers. *Angle Orthodontist*, **69,** 14.

Yustin, D., Neff, P., Rieger, M.R. & Hurst, T. (1993) Characterization of 86 bruxing patients and long-term study of their management with occlusal devices and other forms of therapy. *Journal of Orofacial Pain*, **7,** 54.

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