Community Dent Oral Epidemiol 2004; 32: 307–11

COMMUNITY DENTISTRY AND ORAL EPIDEMIOLOGY

Reported bruxism and biopsychosocial symptoms: a longitudinal study

J. Ahlberg[1,2], A. Savolainen[1,2], M. Rantala[1,3], H. Lindholm[4] and M. Könönen[1,3]

[1]Helsinki University Central Hospital, Helsinki, [2]Finnish Broadcasting Company, Helsinki, [3]Institute of Dentistry, University of Helsinki, Helsinki, [4]Finnish Institute of Occupational Health, Helsinki, Finland

Ahlberg J, Savolainen A, Rantala M, Lindholm H, Könönen M. Reported bruxism and biopsychosocial symptoms: a longitudinal study. Community Dent Oral Epidemiol 2004; 32: 307–11.

Abstract – *Objectives and Methods:* In this follow-up study of 30–50-year-old employees ($n = 211$) of the Finnish Broadcasting Company (YLE), respondents completed questionnaires in both 1999 and 2000 containing items on demographic data, tobacco use, levels of perceived bruxism, affective disturbance, sleep disturbance, somatic symptoms, pain symptoms and temporomandibular disorder (TMD) symptoms. *Results:* Bruxism was significantly more prevalent among smokers ($P = 0.005$). Age, marital status, and gender were not associated with bruxism. Subjects in the frequent bruxism group ($n = 74$) reported the TMD-related painless symptoms, affective disturbance and early insomnia significantly more often than average. In the multivariate analyses, clustered pain symptoms ($P = 0.001$), TMD-related painless symptoms ($P = 0.004$) and smoking ($P = 0.012$) were significantly positively associated with frequent bruxism, when the independent effects of age and gender were controlled for. *Conclusions:* It was concluded that successful management of TMD necessitates smoking cessation, as tobacco use may both amplify the patient's pain response and provoke bruxism. Psychosocial factors and perceived stress should not be ignored, however.

Key words: biopsychosocial; bruxism; smoking

Jari Ahlberg, Chief Dental Officer, Box 80, Fin-00024 Finnish Broadcasting Company, Helsinki, Finland
Tel: +358-40-743-7140
e-mail: jari.ahlberg@yle.fi

Submitted 3 July 2003;
accepted 15 January 2004

Many theories to explain the controversial character of bruxism have emerged over the years (1–6). Two groups of proposed etiological factors can be distinguished: peripheral (morphological) and central (pathophysiological and psychological). At present, peripheral morphological factors, e.g. occlusal discrepancies, are considered to play a minor role, if any, whilst central factors, such as disorders in the dopaminergic system and stress, have been suggested as more important in this disorder. Smoking and alcohol consumption have also been linked to bruxism, and studies suggest that age, gender and genetic factors may influence its prevalence (7–8).

Stress is known to be an initiating, predisposing and perpetuating factor for physical impairment, psychological symptoms and sleep disorders (9–11), whereas bruxism has been considered to be closely associated with temporomandibular disorders (TMD) (12, 13). However, in a recent study among 1339 non-patients, a significant association between reported bruxism and stress experience was found (14). There is also evidence that bruxism may be a sign of a sleep disturbance which appears concomitantly with the transient arousal response (15–17). If stress can affect sleep, it would be fair to assume that stress may provoke bruxism which, in turn, may increase the probability of TMD (18–20).

In our recent cross-sectional survey among multiprofessional media personnel, we reported biopsychosocial factors describing affective disturbance, sleep disturbance, somatic symptoms, pain symptoms and TMD symptoms (21). The present longitudinal study comprises a randomly drawn sample of the same study population and covers bruxism and perceived symptoms over a 24-month period. The aim was to analyze the associations

between reported bruxism and the previously identified biopsychosocial symptom variables, while controlling for the effects of age, gender and tobacco use.

Materials and methods

A total of 1339 employees of the Finnish Broadcasting Company (age: 30–55 years, mean 46, SD 6 in both genders) completed a questionnaire in 1999, with a response rate of 75%. Work duties varied according to gender ($P < 0.001$): males (altogether 51%) were more likely to work in production (65%), service (63%) and management (60%), while females were more likely to be journalists (59%) or work in administration (87%). The study population is described in more detail elsewhere (21).

One-fifth of the 1339 respondents to the baseline questionnaire were randomly selected for clinical examinations and follow-up. In 2000, 205 employees (87%) of the follow-up group completed another questionnaire. Both questionnaires included items on:

- demographic data;
- tobacco use;
- frequency of affective disturbance, sleep disturbance, somatic symptoms, pain symptoms and TMD symptoms (21);
- bruxism: self-assessed frequency of tooth grinding (11).

Each question covering frequency of symptoms over the past 12 months had a Likert-type scale of 1 to 5, where 1 = never; 2 = seldom; 3 = sometimes; 4 = often and 5 = continually. Thus the symptom variables covered perceptions over a 24-month period.

Variables for the present study

Bruxism was calculated as the sum (range 2–10) of self-reported bruxism in 1999 and 2000. Subjects with sum bruxism scores of higher than the overall mean of 3.4 were termed as frequent bruxers ($n = 74$), while those with scores below/equal to the mean value were labeled as non-frequent bruxers ($n = 131$).

Altogether, 23 symptom variables which loaded significantly in the previously identified biopsychosocial factors describing affective disturbance, sleep disturbance, somatic symptoms, pain symptoms, and TMD symptoms were included in the present study (21). For each symptom variable, a sum variable (range 2–10) of the two observations was calculated and the sum response score was dichotomized at its arithmetic mean, i.e. each variable describes a perception which is present more often or as/less often than on average in the studied population.

All tobacco use (including cigarettes, cigars and pipe) was considered as smoking. Former smokers who had stopped tobacco use less than a year ago were included in the smoker group.

Statistical methods

The chi-square test was used to compare the severity of studied physical symptoms in the high and low bruxism groups, and also to compare self-reported bruxism between age, marital status, gender and smoking groups. The logistic regression model was used to analyze the independent effects of the background variables on the probability of frequent bruxism. Independent variables included in the multivariate model were: symptom variables (see Tables 2 and 3) that associated significantly with frequent bruxism in the cross tabulations as total sum variables (cut-off point the arithmetic mean of the total sum), gender (male = 0, female = 1), smoking (no = 0, yes = 1) and age in years as a continuous variable. The forced entry method was used, i.e. all selected independent variables were entered in a single step in the regression model.

Results

Bruxism was significantly more prevalent among smokers ($P = 0.005$). Age, marital status, and gender were not associated with bruxism (Table 1).

Subjects in the frequent bruxism group were significantly more likely to report all studied TMD-related painless symptoms than what was reported on average (Table 2). Similarly, affective disturbance and early insomnia were significantly more prevalent in frequent bruxers, as were some of the somatic and pain symptoms (Table 2). Affective disturbance and pain symptoms were overall more prevalent in the study population than other studied symptoms (Table 2).

According to the logistic regression, pain symptoms ($P = 0.001$) and TMD-related painless symptoms ($P = 0.004$) were significantly positively associated with frequent bruxism (Table 3). Smokers were also 1.2–4.9 times more likely to report frequent bruxism than non-smokers ($P = 0.012$) (Table 3).

Table 1. Percentages for low and high bruxism by age group, marital status, gender and smoking

	n	Low bruxism ($n = 131$) *n* (%)	High bruxism ($n = 74$) (%)	*P*-value
Age (years)				0.349
30–39	44	19.8	24.3	
40–49	84	38.9	44.6	
50–65	77	41.2	31.1	
Marital status				0.935
Married or cohabiting	147	71.0	73.0	
Single	33	16.8	14.9	
Divorced or widowed	25	12.2	12.2	
Gender				0.557
Female	108	48.9	44.6	
Male	97	51.1	52.7	
Smoking				0.005
Yes	64	24.4	43.2	
No	141	75.6	56.8	

Statistical evaluation by chi-square test.

Discussion

The biopsychosocial symptom variables used in the present study have been tested and analyzed in a larger sample of employees of the same company, and selection of the present variables was based on these comprehensive analyses (21). The symptom variables covered perceptions over a 24-month period and our findings strengthen the notion that TMD symptoms relate to on-going bruxism.

For decades it has been claimed that bruxism is provoked mainly by occlusal disturbances (22, 23). Yet, based on extensive experimental and epidemiologic data, there is little evidence of the capability of premature contacts or other minor occlusal disturbances to produce bruxism, or that eliminating such very common interferences could reduce the parafunction (24). Nevertheless, a recent literature review shows a clear transition from a mechanistic perspective toward psychologic and biopsychosocial concepts regarding the etiology, pathogenesis and therapy of TMD (5).

It is also noteworthy that, more frequently, bruxism was significantly associated with pain symptoms and affective disturbance, which are often regarded as consequences of prolonged stress. A coherent relationship between total stress experience and reported bruxism was recently shown among the present employees (11). As stress-related symptoms are reportedly comorbid (25, 26), bruxism in the present study may in fact be a sign of stress. However, reporting of bruxism and physical symptoms may be influenced by negative affectivity, and individuals with subjective distress may be more likely to perceive, overreact to and complain about their sensations (27).

In clinical studies, the relationship of bruxism and subjective stress has remained unclear: electromyographically measured bruxism has been only weakly associated with stress (13), whereas on the other hand, increased levels of anxiety have been reported in sleep bruxers in a controlled polysomnographical study (28). Also, using questionnaires, as in the present study, may cause difficulties in defining the actual prevalence of bruxism: it may be even more common among populations than surveys indicate, but, because of its potential subconscious nature, not registered as a behavior by individuals. Although data on bruxism gathered by questionnaires may be difficult to operationalize, numerous surveys have been performed to evaluate possible interactions between bruxism and psychological factors.

Smokers complain of discomforting or disabling musculoskeletal pain more often than non-smokers (29): A recent survey among nearly 13 000 Britons found that current smokers had about a 50% higher incidence of reporting 'pain in the past year preventing activity' compared with those who never smoked. Also, pain at many sites (lower back, shoulders, elbows, hands, neck and knees) was more intensive in smokers. This association held even among respondents with white-collar or other jobs that did not require heavy lifting or moving. Possible explanations were that nicotine may centrally affect the smoker's pain response, or that tobacco use may reduce the blood supply to tissues.

Apart from musculoskeletal symptoms (including TMD), the incidences of sleep disorders, anxiety, depression, mood disorders, perceived stress, chronic pain, gastrointestinal symptoms, and various psychosomatic complaints are also reportedly higher in tobacco users than in lifelong non-smokers (30). It has been suggested that those who choose to take up smoking may be psychologically predisposed to feel and report pain at lower thresholds. Tobacco use has been addressed as a cause of bruxism (6, 7). In the present study, smoking was independently associated with bruxism in the multivariate model, as the pain and TMD symptoms. These items may have some interaction. However, our results underscore the

Table 2. Biopsychosocial symptoms in low and high bruxism groups

	Low bruxism [n = 131 (%)]	High bruxism [n = 74 (%)]	P-value	Mean sum (SD)
Affective disturbance				
Lethargy	41.2	58.1	0.020	5.4 (1.4)
Tiredness after normal sleep	39.7	53.4	0.059	5.3 (1.8)
Irritability	51.9	60.3	0.250	5.5 (1.3)
Ttiredness/weakness	37.2	52.7	0.032	5.3 (1.6)
Anxiety	42.2	57.5	0.036	5.4 (1.5)
Worry about own health	50.4	67.6	0.017	4.9 (1.6)
Sex dysfunction	45.7	63.9	0.013	3.9 (1.8)
Sleep disturbance				
Middle insomnia	49.6	59.5	0.175	5.7 (1.9)
Early insomnia	46.6	62.5	0.030	4.9 (1.7)
Nightmares	32.0	38.4	0.364	4.1 (1.4)
Somatic symptoms				
Breathing difficulty	41.1	47.3	0.390	2.9 (1.4)
Palpitations	41.9	60.3	0.012	3.7 (1.7)
Nausea	45.4	54.8	0.198	2.8 (1.1)
Hand tremor	32.8	31.9	0.902	3.1 (1.4)
Sweatiness	35.4	38.9	0.621	4.2 (1.7)
Stomach ache	30.0	41.1	0.109	4.3 (1.6)
Loose bowels	44.2	47.9	0.606	4.6 (1.6)
Irritable bowel	31.5	45.2	0.047	4.1 (1.8)
Pain symptoms				
Neck pain	52.7	64.9	0.090	6.0 (2.1)
Head pain	36.2	44.6	0.235	5.0 (1.6)
Back pain	55.0	64.4	0.191	5.0 (1.8)
Pain affecting work	41.9	54.1	0.094	3.8 (1.8)
Eye pain	35.4	58.9	0.001	4.2 (1.6)
Jaw pain	11.8	53.4	0.000	3.1 (1.5)
Toothache	26.7	47.9	0.002	3.1 (1.2)
Tender teeth	49.6	75.3	0.000	3.9 (1.5)
Earache	43.8	60.3	0.025	2.9 (1.2)
TMD-related painless symptoms				
Biting difficulty	20.6	64.4	0.000	2.8 (1.4)
Teeth do not fit	27.1	63.0	0.000	2.9 (1.5)
Difficulty moving jaw	14.5	47.2	0.000	2.6 (1.3)
Difficulty opening jaw	13.7	39.7	0.000	2.5 (1.1)
Jaw locking	12.2	26.0	0.012	2.4 (1.0)
TMJ clicking	21.4	44.6	0.000	3.4 (2.0)
Muscle tiredness in jaws	23.7	71.2	0.000	2.9 (1.4)
Blocked ears	41.5	63.5	0.003	3.8 (1.7)
Hearing difficulty	29.8	43.8	0.043	3.2 (1.7)

Mean values of the sum variables and percentages for a symptom perceived as more frequent than on average. Chi-square test.

Table 3. Variables entered in the multivariate model

	Items	Mean sum (SD)	β	Odds ratio	95% CI	P-value
Affective disturbance	4	4.8 (1.2)	−0.23	0.8	0.4–1.4	0.566
Sleep disturbance	1	4.9 (1.7)	0.52	1.7	0.8–3.4	0.142
Somatic symptoms	2	3.9 (1.4)	0.13	0.7	0.5–2.4	0.736
Pain symptoms	5	2.8 (0.8)	1.21	3.4	1.6–7.1	0.001
TMD-related painless symptoms	9	2.9 (1.0)	1.11	3.1	1.4–6.5	0.004
Gender (female)			−0.39	0.7	0.3–1.3	0.263
Smoking			0.89	2.4	1.2–4.9	0.012
Age (in years)			−0.02	n.a.	n.a.	0.585

Mean sum scores for the selected symptom variables, and effects of the studied independent variables on the probability of frequent bruxism.

need for tobacco use to be controlled in studies on TMD and bruxism.

This longitudinal study revealed a relationship between bruxism, pain symptoms, TMD symptoms and smoking. We may conclude that successful management of TMD necessitates smoking cessation, as tobacco use may both amplify the patient's pain response and provoke bruxism. Nevertheless, psychosocial factors and perceived stress should not be ignored.

References

1. Reding GR, Zepelin H, Robinson JE Jr, Zimmerman SO, Smith VH. Nocturnal teeth-grinding: all night psychophysiological studies. J Dent Res 1968;47: 786–97.
2. Faulkner K. Bruxism: a review of the literature. Part I. Aust Dent J 1990;35:266–76.
3. Faulkner K. Bruxism: a review of the literature. Part II. Aust Dent J 1990; 35: 355–61.
4. Lavigne GJ, Montplaisir J. Bruxism: epidemiology, diagnosis, pathophysiology, and pharmacology. In: Fricton JR, Dubner RB, editors. Orofacial pain and temporomandibular disorders. New York: Raven Press; 1995. p. 387–404.
5. Molin C. From bite to mind: TMD – a personal and literature review. Int J Prosthod 1999;12:279–88.
6. Lobbezoo F, Naeije M. Bruxism is mainly regulated centrally, not peripherally. J Oral Rehab 2001; 28:1085–91.
7. Madrid G, Madrid S, Vranesh JG, Hicks RA. Cigarette smoking and bruxism. Percept Mot Skills 1998;87:898.
8. Hublin C, Kaprio J, Partinen M, Koskenvuo M. Sleep bruxism based on self-report in a nationwide twin cohort. J Sleep Res 1998;7:61–7.
9. McEwen B. Protective and damaging effects of stress mediators. New Engl J Med 1998;338:171–9.
10. Von Onciul J. Stress at work. BMJ 1996;313:745–8.
11. Ahlberg J, Könönen M, Nissinen M, Rantala M, Sarna S, Lindholm H et al. Self reported stress among multiprofessional media personnel. Occup Med 2004;54: (in press).
12. Pingitore G, Chrobak V, Petrie J. The social and psychlogic factors of bruxism. J Prosthet Dent 1991; 65:443–6.
13. Pierce CJ, Christman K, Bennett ME, Close JM. Stress, anticipatory stress, and psychologic measures related to sleep bruxism. J Orofac Pain 1995;9:51–6.
14. Ahlberg J, Rantala M, Savolainen A, Suvinen T, Nissinen M, Sarna S et al. Reported bruxism and stress experience. Community Dent Oral Epidemiol 2002;30:405–8.
15. Lavigne GJ, Montplaisir JY. Restless legs syndrome and sleep bruxism: prevalence and associations among Canadians. Sleep 1994;17:739–43.
16. Kato T, Montplaisir JY, Guitard F, Sessle BJ, Lund JP, Lavigne GJ. Evidence that experimentally induced sleep bruxism is a consequence of transient arousal. J Dent Res 2003;82:284–8.
17. Macaluso GM, Guerra P, Di Giovanni G, Boselli M, Parrino L, Terzano M. Sleep bruxism is a disorder related to to periodic arousals during sleep. J Dent Res 1998;77:565–73.
18. LeResche L. Epidemiology of temporomandibular disorders: implications for the investigation of etiologic factors. Crit Rev Oral Biol Med 1997;8: 291–305.
19. Lavigne GJ, Goulet JP, Zuconni M, Morrison F, Lobbezoo F. Sleep disorders and the dental patient: an overview. Oral Surg Oral Med Oral Pathol Oral Radiol Endodont 1999;88:257–72.
20. Rantala MAJ, Ahlberg J, Suvinen TI, Nissinen M, Lindholm H, Savolainen A, et al. Temporomandibular joint related painless symptoms, orofacial pain, neck pain, headache and psychosocial factors among non-patients. Acta Odontol Scand 2003;61:217–22.
21. Ahlberg J, Suvinen T, Rantala M, Lindholm H, Nikkilä H, Nissinen M et al. Distinct biopsychosocial profiles emerge among nonpatients. J Psychosom Res 2002;53:1077–81.
22. Dawson P. Temporomandibular join pain-dysfunction problems can be solved. J Prosthet Dent 1973;29:100–112.
23. Shore N. Temporomandibular joint dysfunction and occlusal equilibration. Philadelphia, PA: JB Lippincott; 1976.
24. Wilding RJC. The association between chewing efficiency and occlusal contact area in man. Arch Oral Biol 1993;38:589–96.
25. Clark G, Adler R. A critical evaluation of occlusal therapy: occlusal adjustment procedures. J Am Dent Assoc 1985;110:743–50.
26. Aaron LA, Burke MM, Buchwald D. Overlapping conditions among patients with chronic fatique syndrome, fibromyalgia, and temporomandibular disorder. Arch Intern Med 2000;160:2398–401.
27. Korzun A. Facial pain, depression and stress – connections and directions. J Oral Pathol Med 2002;31:615–9.
28. Turner JA, Dworkin SF, Mancl L, Huggins KH, Truelove EL. The roles and beliefs, catastrophizing, and coping in the functioning of patients with temporomandibular disorder. Pain 2001;92:41–51.
29. Palmer KT, Syddall H, Cooper C, Coggon D. Smoking and musculoskeletal disorders: findings from a British national survey. Ann Rheum Dis 2003;62:33–6.
30. US Department of Health and Human Service. The health consequences of smoking. nicotine addiction: a report of the Surgeon General. Washington, DC: US Department of Health and Human Services, Public Health Service, Centers of Disease Control, Office on Smoking and Health, DHHS publication; 1998.