Oral Motor Parafunctions Among Heavy Drug Addicts and Their Effects on Signs and Symptoms of Temporomandibular Disorders

Ephraim Winocur, DMD
Senior Teacher

Anat Gavish, DMD
Senior Teacher

Guy Volfin
DMD Candidate

Michele Halachmi, DMD
Instructor

Esther Gazit, DMD
Clinical Associate Professor

Department of Occlusion and Behavioral Sciences
The Maurice and Gabriela Goldschleger School of Dental Medicine
Tel Aviv University
Tel Aviv, Israel

Correspondence to:
Prof Esther Gazit
Department of Occlusion and Behavioral Sciences
The Maurice and Gabriela Goldschleger School of Dental Medicine
Tel Aviv University
Tel Aviv, Israel
Fax: +972-3-6409250

This paper was written in partial fulfillment of the DMD thesis requirement for Guy Volfin, School of Dental Medicine, Tel Aviv University.

Aims: *To investigate the prevalence of temporomandibular disorders (TMD), bruxism, and other oral habits among drug addicts compared to a normal, non-addicted, matched control population, and to assess the detrimental effect of long-term drug abuse on the parameters studied.* **Methods:** *Subjects included 55 drug-addicted patients (51 males and 4 females) randomly selected from long-term addicts using "hard" narcotics and attending a methadone maintenance center and a control group of 52 normal non-addicted individuals (48 males and 4 females) matched to the addicts for age, gender, and socioeconomic status. A clinical examination and a questionnaire were used. One examiner determined that all questions were correctly understood and answered, and a second examiner performed the clinical examinations and was unaware of the results of the questionnaire.* **Results:** *The addicted group had a high prevalence of orofacial motor behavior (bruxing, clenching) as well as signs and symptoms of TMD (morning headache, joint noises, joint and masticatory muscle tenderness to palpation, and tooth wear) compared to the controls. Active (voluntary) jaw opening was significantly smaller, although within an acceptable range when compared to the controls.* **Conclusion:** *Long-term drug abuse detrimentally affects the stomatognathic system, as expressed in a high prevalence of oral motor behavior and signs and symptoms of TMD.*

J OROFAC PAIN 2001;15:56–63.

Key words: substance-abuse disorders, bruxism, oral motor parafunctions, temporomandibular disorders

The literature is controversial concerning iatrogenic drug-induced bruxism and the efficacy of medications in the treatment of bruxism.[1] Most of the existing data are purely anecdotal observations based on case reports or on animal studies, and therefore their findings should be considered cautiously. It is well established that certain substances suppress or exacerbate bruxing activity in animals and in humans,[1–3] and substances may be related to the dopaminergic, serotonergic, or adrenergic systems. In several animal studies, the facilitatory effect of dopaminergic drugs on rhythmic jaw movement has been described.[4,5] It has also been suggested that the central dopaminergic system might be involved in the modulation of sleep bruxism in rats.[6] In contrast, the capability of the catecholamine precursor L-dopa to exacerbate bruxism in humans has been challenged.[7] Long-term exposure to anti-dopaminergic drugs is associated with daytime bruxism, which ceases during sleep in humans.[8]

Table 1 Study Population Characteristics

	Addiction period			Rehabilitation period				
Group	Duration*	Major drugs	Additional drugs	Duration*	Major drugs†	Additional drugs	Total duration of drug abuse*	Alcohol use
Addicts	12.6 ± 6.0 (3 to 25)	Cocaine, heroin	Ecstasy, marijuana, tranquilizers, crack, LSD	2.5 ± 2.1 (0.5 to 9)	Cocaine, heroin	Ecstasy, marijuana, tranquilizers, crack, LSD	15.1 ± 6.5 (4 to 30)	Occasional
Controls	—	None	None	—	None	None	—	Occasional

*In years, mean ± SD and range.
†Most of the subjects admitted to an occasional use of heroin and cocaine during the rehabilitation period.

Fitzgerald and Healy observed diurnal bruxism secondary to selective serotonin reuptake inhibitor (SSRI) medication in 5 of 6 patients,[9] which persisted in 2 of these patients after the drug was discontinued. Amir et al found acute bruxism and akathisia occurring as early side effects of anti-psychotic drug treatment in 2 patients.[10] The addition of propranolol (a beta-adrenergic blocker) relieved both of these complications. Muscle relaxants, eg, diazepam (Valium)[11] and methocarbamol (Robaxin),[12] partially suppress orofacial activity. Ware and Rugh proposed tricyclic antidepressants for the treatment of "destructive bruxism" occurring in rapid eye movement (REM) sleep.[13] In contrast, in a double-blind randomized study, Mohamed et al[14] found no association between the intake of a tricyclic antidepressant (amitriptyline) and bruxism. Hartman observed a 60% increase in bruxing activity following subjects' use of alcoholic drinks.[15]

Stress has been implicated as a factor that exacerbates bruxism. In rats, the evidence supports the existence of anatomic connections between limbic structures (amygdala) and the trigeminal motor nucleus.[16] Since the amygdala is implicated in the mediation of stress, it could participate in the stress-provoked accentuation of tardive dyskinesia and spontaneous orofacial dyskinesia.[17] In rabbits, bruxism-like activity has been reported after amygdala stimulation.[18]

To the best of the authors' knowledge, the association between drug abuse and oral parafunctions has not yet been investigated. These substances act on specific neurotransmitter pathways and cause neurochemical changes similar to those of the natural rewarding stimuli.[19] Chronic cocaine intake increases brain reward thresholds and elicits dysregulation of the brain reward system, resulting in compulsive drug use.[20,21] The effect of drug abuse on the dopamine system in the mediation of rewarding and addicting properties is well established.[22,23] Some experiments suggest that serotonin receptors and serotonin transmission in the nucleus accumbens may mediate some of the rewarding properties of drug abuse.[24]

The purpose of this study was to evaluate the prevalence of oral motor parafunctions and temporomandibular disorder (TMD) signs and symptoms in a group of drug addicts compared to an age- and gender-matched non-addicted control group.

Subjects and Methods

A total of 55 subjects (51 males and 4 females), ranging in age from 24 to 46 years (mean 34.4 ± 6.1), were randomly selected from addicts using "hard" narcotics who attended the Methadone Maintenance Center in Hadera, Israel. All were heavily addicted to heroin and cocaine but reported consuming other drugs as well (eg, ecstasy, marijuana, tranquilizers, "crack," LSD, etc). Most had been in prison for prolonged periods, where they continuously consumed combinations of "hard" drugs. Some received pharmacologic treatment with methadone during their time in prison, concomitant with their use of drugs of abuse. The total time of use of drugs ranged from 4 to 30 years (mean 15.1 ± 6.5) (Table 1). No alcohol addiction was noted in the subjects' files from the Methadone Maintenance Center. The Department for the Treatment of Addictions in the Israel Ministry of Health approved the present study. Inclusion criteria for the addicted group were: *(1)* age between 18 and 50 years, *(2)* current administration of methadone (hard drug addicts), *(3)* presence of a natural dentition or a fixed dental rehabilitation, *(4)* no history of facial or cervical injury, and *(5)* no history of general neurologic

disturbances, hormonal diseases, neoplasm, or psychiatric diseases.

The control group consisted of 52 patients (48 males and 4 females) who ranged in age from 18 to 50 years (mean 35.3 ± 10.4) and who were hospitalized in the Orthopedic Department of Western Galilee Hospital, Naharya, Israel, following treatment for a fracture. Inclusion criteria for the controls were identical to the addicted group, except for the use of drugs. Control subjects had no history of alcohol or illicit drug addiction; no pharmacologic treatment known to influence sleep, motor activity, or dopaminergic transmission (eg, benzodiazepines, L-dopa, neuroleptics, tricyclic antidepressants); no history of trauma in the head, neck, or back region; and no pain for at least 1 week following fracture treatment and prior to examination.

A questionnaire and a clinical examination were used in the study. One experienced examiner determined first that all questions were correctly understood and answered, and a second examiner performed the clinical examinations and was unaware of the results of the questionnaire.

Information regarding symptoms of TMD and oral habits was obtained from the questionnaire. Symptoms included temporomandibular joint (TMJ) noises (feeling of clicking, popping, or grating during jaw movements); joint catching (sudden, momentary sticking of the jaw that prevents full opening but is self-releasing); joint locking (limited opening and feeling that the jaw is caught without the ability to release); joint pain (pain in the joint area); joint tension (feeling of increased tension within the joint, forcing a tension-releasing movement); morning headache upon awakening; and morning stiffness (difficulty in opening and yawning upon awakening). Oral habits included daily frequency of gum chewing; present nail biting; daily frequency of biting hard foreign objects; daily clenching (awareness of daytime bruxing, clenching, or grinding); bruxing during sleep (knowledge of nighttime bruxing, clenching, or grinding); and "jaw play" (habit of performing small non-functional mandibular movements without tooth contact).

Clinical examination included measurements of the interincisal distance in active (voluntary) maximal mouth opening and passive (assisted) maximal opening by the examiner applying finger pressure to extend the opening to its maximal capacity. The presence of joint clicking/crepitation in opening and closing upon palpation over the TMJ area and joint sensitivity to palpation (force of approximately 1 lb) of the lateral aspect of the joint were assessed according to Dworkin and LeResche.[25] The degree of wear on one mandibular canine only was recorded on a scale modified from Johansson et al[26] (Grade 0 = no visible wear facets, Grade 1 = enamel only, Grade 2 = enamel and dentin, extensive cusp abrasion).

Muscle sensitivity to palpation (tenderness on manual palpation of approximately 2 lb[25]) was carried out on the superficial and deep masseter and the anterior and middle portion of the temporalis (bilaterally for all muscles), and a scale of 0 to 3 was used (0 = no pain, 1 = mild pain, 2 = moderate pain, 3 = severe pain). The following means were computed for each subject: mean muscle sensitivity to palpation for the 2 masseter muscles (right and left), for the 2 temporalis muscles (right and left), and for the 4 muscles together. Subjects who presented with at least 2 muscle sites of moderate (degree 2) or severe (degree 3) sensitivity were determined to be suffering from increased muscle sensitivity.

Statistical Analysis

BMDP statistical software[27] was used to analyze the data. Student *t* tests for continuous variables (age, mean muscle sensitivity, active and passive maximal mouth opening, and the difference between active and passive maximal mouth opening) and Chi-square tests for qualitative variables (self-reported symptoms, clinical signs, and self-reported oral habits) were used to evaluate differences between groups (addicted versus control). Relative risk ratios and 95% confidence intervals were computed. A Bonferroni correction was performed to reduce the possibility of type I error.

Results

The prevalence of self-reported symptoms and clinical signs exhibited by the 2 groups are shown in Figs 1 and 2. To reduce the possibility of type I error, all *P* values were multiplied by 16 (Bonferroni correction). The most prevalent symptom in both groups was joint noise: 54.5% in the addicts and 34.6% in the controls (Fig 1). In all self-reported symptoms studied, a higher prevalence was found in the addicted group compared to the controls, except for joint tension. Statistically significant differences were found for the symptom of morning headache ($P = 0.018$). Since the prevalence of this symptom in the control group was 0, it was not possible to calculate the relative risk ratio.

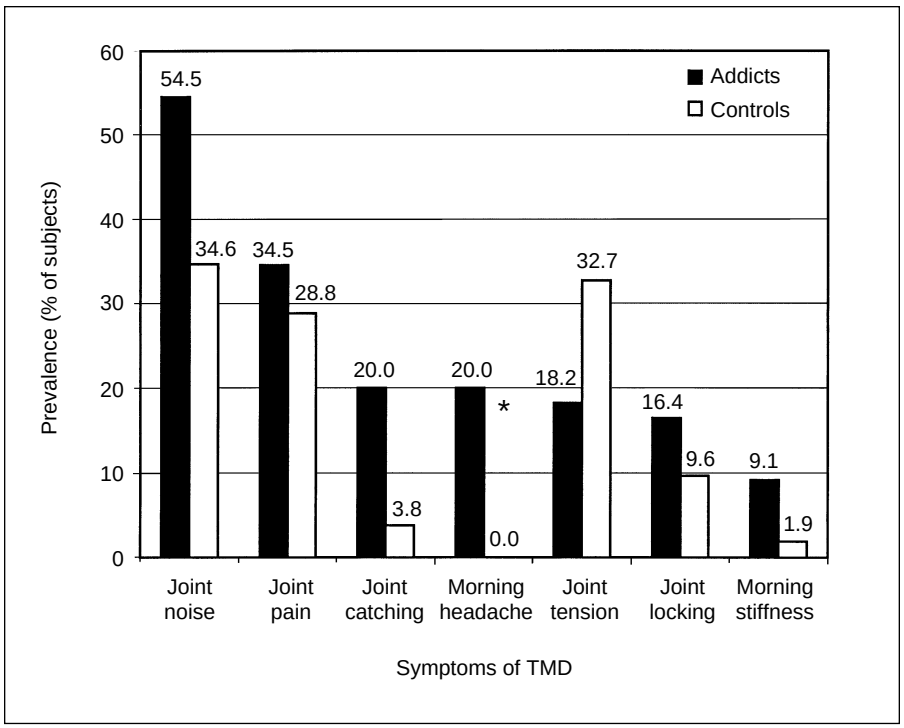

Fig 1 Prevalence of self-reported symptoms of TMD. *$P < 0.05$ (Chi-square test).

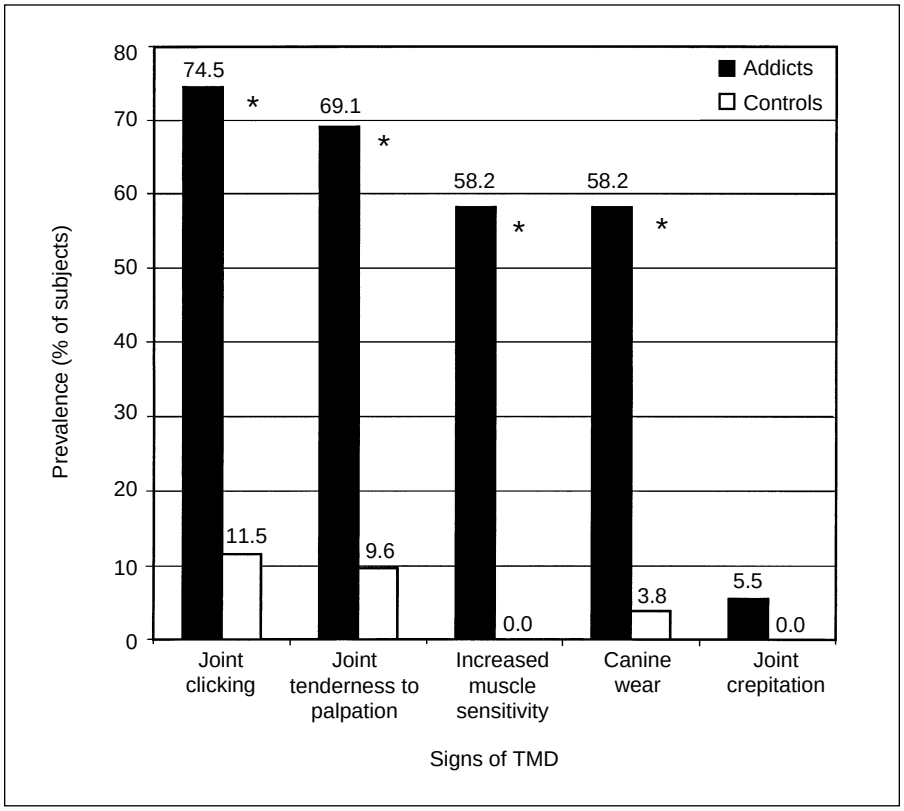

Fig 2 Prevalence of clinical signs of TMD. *$P < 0.001$ (Chi-square test).

All clinical signs examined showed a statistically significantly higher prevalence in the addicted group ($P < 0.001$) compared to the control group, except for joint crepitation, which showed the lowest prevalence (Fig 2). The highest relative risk ratio was found for canine wear (15.13; 95% confidence interval [CI] 3.82 to 59.97), followed by joint tenderness to palpation (7.19; 95% CI 3.07 to 16.84) and joint clicking (6.46; 95% CI 3.00 to 13.93). It was not possible to calculate the relative risk ratio for increased muscle sensitivity, since the prevalence of this sign in the control group was 0. The data concerning muscle sensitivity to palpation are presented in Tables 2 and 3. The mean sensitivity of the masseter muscle and the temporalis muscle was significantly greater in the addicted subjects compared to the control subjects (Table 2). In 21.8% of the addicts, all muscle sites showed moderate to severe sensitivity, and in 16.3% of the addicts 6 of 8 sites were sensitive at Grade 2 or more (Table 3). In 40% of the addicts, no site of Grade 2 or more was found. None of the control subjects had sensitive muscle sites (Grade 2 or more).

Data concerning the range of mouth opening are presented in Table 4. The maximal active and passive mouth opening values were significantly decreased in the addicted group compared to the control group ($P < 0.001$), although they were still within normal range.

The degree of canine wear seen in the study population is presented in Table 5.

The prevalence of self-reported oral habits (Fig 3) revealed more frequent performance of daytime clenching, sleep bruxism, and jaw play among the addicted subjects. The habits of gum chewing, biting hard objects, and nail biting showed opposite results. To reduce the possibility of type I error, all *P* values were multiplied by 6 (Bonferroni correction). Significant differences between groups were found for daytime clenching ($P = 0.00186$, RR = 2.21 [95% CI, 1.38 to 3.53]) and sleep bruxism ($P = 0.0075$, RR = 2.46 [95% CI, 1.32 to 4.58]), both of which were more prevalent in the addicted subjects, while gum chewing ($P = 0.00258$, RR = 0.34 [95% CI, 0.18 to 0.66]) was significantly more prevalent in the control group. There was a trend for more jaw play in the addicted subjects ($P = 0.0687$, RR = 2.17 [95% CI, 1.15 to 4.12]). A statistically significant association (Student *t* test) was found between duration of addiction and the habits of jaw play ($P = 0.0001$), gum chewing ($P = 0.004$), and daytime clenching ($P = 0.047$).

Table 2 Muscle Sensitivity to Palpation

	Sensitivity (mean ± SD)		
Muscle	Addicts	Controls	*P* value*
Masseter	1.6 ± 0.9	0.1 ± 0.2	< 0.001
Temporalis	1.2 ± 0.8	0.1 ± 0.2	< 0.001
Masseter and temporalis	1.4 ± 0.9	0.08 ± 0.2	< 0.001

*According to *t* test for independent samples.
Mean muscle sensitivity to palpation on a scale of 0 to 3. Represents both right and left muscles (masster, temporalis, and both, respectively), as explained in Methods.

Table 3 No. of Moderate and Severely Tender Muscle Sites Among the Addicted Patients

No. of tender sites	Percentage of population
0	40.0
1	1.8
2	9.2
3	3.6
4	7.3
5	0.0
6	16.3
7	0.0
8	21.8

Numbers represent the percentage of subjects presenting with tender muscle sites as specified in the first column. Definition of tender muscle sites as specified in Methods.

Table 4 Range of Mouth Opening

	Mouth opening (mm, mean ± SD)		
Parameter	Addicts	Controls	*P* value*
Active opening	41.2 ± 7.1	47.9 ± 3.8	< 0.001
Passive opening	45.9 ± 5.4	49.7 ± 3.6	< 0.001
Difference	4.64 ± 2.65	1.84 ± 1.78	< 0.001

*According to *t* test for independent samples.
Definition of mouth opening range given in Methods.

Table 5 Degree of Canine Wear

	Percent of subjects with wear	
Degree of wear	Addicts	Controls
No wear (0)	3.6	78.8
Enamel only (1)	38.2	17.3
Dentin and enamel (2)	58.2	3.9

Numbers represent the percentage of subjects in each group presenting with degree of canine wear as specified in the first column. Degree of wear was assessed as described in Methods.

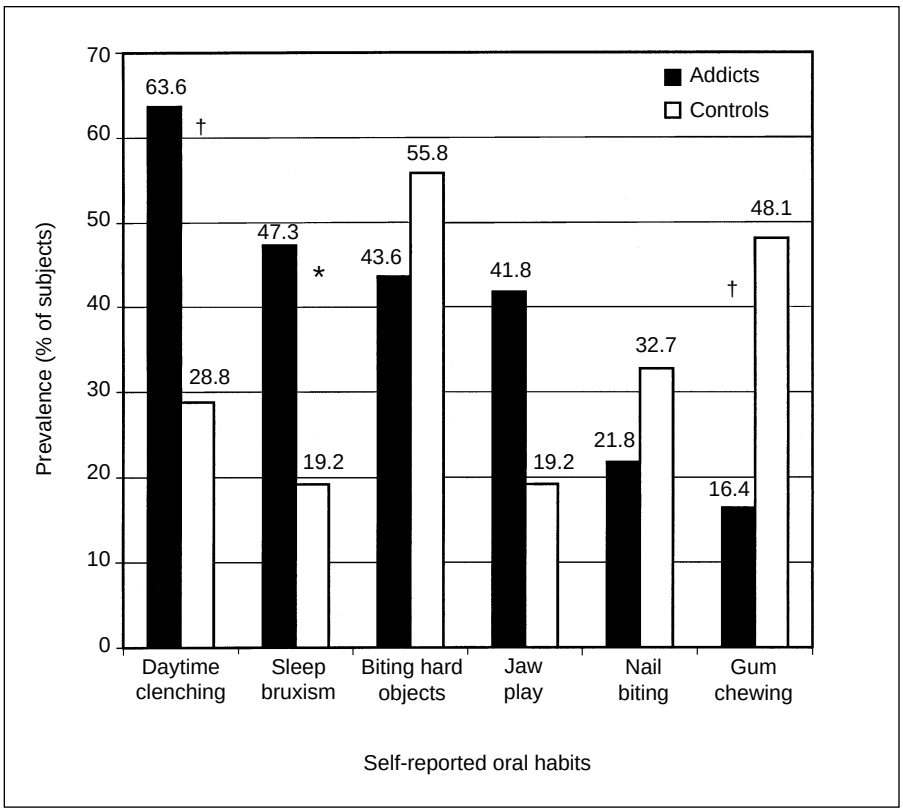

Fig 3 Prevalence of oral habits. *$P < 0.01$, †$P < 0.005$ (Chi-square test).

Discussion

To the best of our knowledge, this is the first controlled study conducted on a heavily drug-addicted population regarding oral motor parafunctions and TMD. Previous clinical reports showed severe bruxism, myofascial pain, chewing-like movements, and tongue rubbing among drug users.[28–30] In contradiction to this, Takahama[31] reported that morphine addiction did not influence the prevalence of bruxism. Since the observations were not statistically assessed, it was impossible to compare those claims to the findings of this study. However, the present results correspond to the observation that drug abuse exacerbates oral motor parafunction.

The most significant findings in this study population were the high prevalence of signs and symptoms of TMD and of nocturnal bruxing and diurnal clenching. Daytime clenching (64%) and self-reported sleep bruxism (47%) in the addicted population were higher than those of the control group (29% and 19%, respectively) and other adult population groups studied (20 to 25%).[32,33] Detection of sleep bruxism was established indirectly in this study on the basis of wear facets and self-reports of morning headache and morning stiffness. These 3 parameters were more prevalent among the addicted group, but only sleep bruxism and morning headache were statistically significantly more prevalent. Direct detection of bruxing activity by polysomnography could make an unequivocal diagnosis possible, but it was not applicable to this study group.

It could be hypothesized that heavy and prolonged use of certain drugs was directly related to these findings. Another contributing factor could be the severe prolonged effect of emotional distress on these subjects, which could lead to enhanced bruxing activities.[34] The prevalence of the habit of jaw play among the addicted group was similar to that of a previous study[35] conducted on adolescent females. In the present control group, the prevalence was insignificantly lower.

The high prevalence of oral activities (clenching, bruxing, and jaw play) could be an oral motor

behavior related to the long-term use of drugs and interpreted as a form of oral tardive dyskinesia rather than oral habits.[1] A syndrome that resembles tardive dyskinesia and results from long-term treatment with methadone has been reported.[36] Cocaine and methadone hydrochloride can cause movement disorders (eg, tremor, choreic movements, dyskinesia).[36–38] In the addicted subjects, no such side effects were observed. It is possible that subjects suffering from these side effects, as well as from hallucinations or dizziness, were not examined.

The high prevalence of signs and symptoms in the addicted group must be interpreted with caution. It is our clinical impression that this particular study group was more susceptible or apprehensive of any clinical examination and more prone to hypochondriasis. The examiner who performed the clinical examination was unaware of the result of the questionnaire but was not blind to the group (ie, addicted versus control). This bias, if present, may have affected the higher prevalence of the sensitivity to palpation of muscles and joints to some extent. On the other hand, the muscle hypersensitivity could be related to the mentally or physically compromised health of the addicted group.

In addition to the well-known detrimental effects of long-term drug abuse, this study has underlined the potential damage to the stomatognathic system as expressed in the high prevalence of oral motor behaviors and of signs and symptoms of TMD.

Acknowledgments

The authors thank Dr Heled Ludmila, Director of the Methadone Maintenance Center, Hadera, and Dr Gershon Volpin, Director of the Orthopedic Department, Western Galilee Hospital, Naharya, Israel, for their assistance and cooperation. The authors also thank Ms Rita Lazar for editorial assistance, Ms Ilana Gelernter for statistical consultation, and especially Professor Ilana Eli for her invaluable assistance and advice.

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