Lack of Associations Between Occlusal and Cephalometric Measures, Side Imbalance in Striatal D2 Receptor Binding, and Sleep-Related Oromotor Activities

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Aims: *First, to evaluate possible orofacial morphologic differences between sleep bruxers and non-bruxers, and second, to determine possible correlations between morphologic factors and striatal D2 receptor expression in persons with sleep-related oromotor activities.* **Methods:** *Twenty subjects were included in this study; half of them had polysomnographically confirmed oromotor values above the cutoff points for sleep bruxism. For all participants, 26 standard occlusal measures were recorded clinically and from dental study casts. In addition, 25 standard angular and linear measures were taken from standardized cephalometric films, and variables were derived to evaluate dental and skeletal relationships. Fourteen of the 20 participants had also participated in a previous study that included iodine-123-iodobenzamide (I-123-IBZM) and single-photon emission-computed tomography (SPECT). For them, the side-to-side difference in striatal D2 receptor binding was determined as the neurochemical outcome measure.* **Results:** *Following the classical Bonferroni adjustment for multiple testing, no morphologic differences were found between the sleep bruxers and the non-bruxers. In addition, none of the morphologic variables were significantly associated with the neuroimaging data.* **Conclusion:** *Taking into account the low power of this retrospective, exploratory study, the results suggest that the orofacial morphology of sleep bruxers does not differ from that of non-bruxers. In addition, morphologic factors are probably not involved in the asymmetry in striatal D2 receptor distribution that was previously observed in association with sleep bruxism.*

J OROFAC PAIN 2001;15:64–71.

Key words: sleep bruxism, iodine-123-iodobenzamide, single-photon emission-computed tomography, dental occlusion, cephalometry

The etiology of sleep bruxism is still controversial. Many authors claim a multifactorial cause (for reviews, see Attanasio[1] and Lobbezoo and Lavigne[2]). In general, 3 groups of etiologic factors can be distinguished. First, pathophysiologic factors may be involved in the precipitation of bruxism. For example, it has been claimed that bruxism is part of an arousal response, thus linking sleep-related bruxism to the field of sleep

This paper was based in part on the thesis of Dr Iafrancesco, accepted by the Faculté de Médecine Dentaire, Université de Montréal, in fulfillment of the requirements for the master's degree.
A preliminary report of this study was presented at the meeting of the European Academy of Craniomandibular Disorders (EACD), October 21–24, 1999, Judean Hills, Israel.

disorders.[3–5] Altered brain chemistry (eg, an asymmetric nigrostriatal dopaminergic function) has been associated with bruxism as well.[6,7] Other pathophysiologic factors that have been implicated in bruxism are the effects of cigarette smoking,[8,9] alcohol, illicit drugs, trauma, disease, and medication (reviewed by Bader and Lavigne[10]). Second, psychologic factors, such as stress and personality, have been implicated in the pathophysiology of bruxism. So far, studies of these factors have yielded equivocal results.[11–14] Third, morphologic factors (eg, dental occlusion and anatomy of the orofacial skeleton) are thought to be involved in the etiology of bruxism. Occlusal discrepancies, eg, a slide between retruded contact position (RCP) and intercuspal position (ICP), were historically considered the most common cause of bruxism.[15] More recently, the role of occlusion has been debated and contested,[1,11,16] in part because it has been demonstrated that experimentally placed deflective occlusal contacts do not elicit bruxism.[17] From the above concise review, it is obvious that more studies are needed to elucidate the exact etiology of bruxism.

Using a rat model for bruxism, Gomez et al[18] and Areso et al[19] tested the relationship between pathophysiologic (alterations in central dopaminergic neurotransmission), psychologic (stress), and morphologic (occlusal disharmonies) etiologic factors. They found that placement of an acrylic cap on the mandibular central incisors caused an enhancement in apomorphine-induced non-functional masticatory activity. In addition, they observed the development of an imbalance between hemispheres in dopa accumulation after the acrylic cap was worn for a prolonged period of time. They carefully concluded that there might be a possible involvement of occlusal disharmonies in the putative role of central catecholaminergic neurotransmission in the etiology of parafunctional masticatory movements.

The authors performed a retrospective study to establish putative associations between various orofacial morphologic factors (occlusal and cephalometric measures), neurochemical factors (side-to-side differences in striatal D2 receptor binding), and sleep-related oromotor activities in a group of 20 subjects with or without a polysomnographically established diagnosis of sleep bruxism, 14 of whom also participated in a previous neuroimaging study.[6] Two null hypotheses were tested: *(1)* no morphologic differences are present between persons who fulfill the polysomnographic criteria for a diagnosis of sleep bruxism[20] and those who do not, and *(2)* no significant correlations are present between morphologic factors and the side imbalance in striatal D2 receptor expression.

Materials and Methods

Participants

From the group of individuals who participated in the previous neuroimaging study,[6] occlusal and cephalometric data were available for 14 persons. For 6 additional individuals, only morphologic data were present. All 20 persons demonstrated polysomnographically confirmed sleep-related oromotor activities; 10 of them were diagnosed with sleep bruxism according to the polysomnographic cutoff points that were established for this disorder by Lavigne et al.[20] Both the sleep bruxism group and the non-bruxism group consisted of 6 men and 4 women. The mean (± SD) ages were 33.2 (± 6.0) years for the sleep bruxism group and 31.2 (± 9.2) years for the non-bruxism group. The recruitment strategy and the inclusion and exclusion criteria were described previously.[6] All participants gave informed consent to procedures approved by the human subjects ethics committees of the Université de Montréal and the Notre-Dame and Sacré-Coeur hospitals in Montréal, Québec, Canada.

Polysomnography

A detailed description of the methods used for recording and scoring sleep and oromotor activities has been published previously.[6] Briefly, 2 consecutive polysomnographic recordings were obtained from all 20 participants. The first night was for habituation to sleeping in a laboratory environment and to rule out sleep-related disorders other than bruxism (eg, periodic limb movements during sleep or sleep apnea). The second night was for the collection of standard sleep variables (ie, total sleep time, sleep efficacy and latency, number of sleep stage shifts per hour of sleep, and sleep stage distribution) and oromotor variables. As oromotor outcome measures, the number of episodes with oromotor activities per hour of sleep (episodes/hour) and the number of oromotor bursts per episode (bursts/episode) were determined. These 2 variables have been used in previous studies to describe sleep-related oromotor activities and to establish possible sleep bruxism diagnoses.[7,8,20]

Orofacial Morphologic Measures

Occlusal Measures. In total, 26 standard occlusal measures were taken from each of the 20 participants, both clinically and from dental study casts. The metric study cast measures were recorded by means of a digital caliper (Absolute Digimatic). This caliper has a resolution of 0.01 mm and an error rate of 0.02 mm. The standard occlusal measures were:

- Number of teeth present in the mandible and maxilla, both per jaw and total
- Angle's classification of malocclusion, determined on the right and left sides, for both the first molars and the canines (Classes I to III)[21]
- Vertical (overbite) and horizontal (overjet) overlap of the maxillary and mandibular right central incisors (in mm)
- Functional occlusal scheme for mandibular contact movements to the right and left sides (canine rise or group function)[22]
- Slides from retruded contact position (RCP) to intercuspal occlusion (ICP) (in mm)
- Values (in mm) for maxillary and mandibular intercanine width (ie, the distance between the canine crown tips or the centers of the corresponding facets), intermolar width (ie, the distance between the centers of the occlusal surfaces of the first molars), and arch depth (ie, the distance between a line tangent to the labial surface of the central incisors and a line connecting the most distal points of the first molars). For both the width and the depth measures, maxillary/mandibular ratios were calculated. In addition, for both the maxilla and the mandible, ratios were made between intercanine and intermolar width and between intercanine width and arch depth.
- Anterior crowding in the mandible, scored on a 5-point scale (0 = no crowding; 1 = 1 to 3 mm of crowding; 2 = 3 to 5 mm of crowding; 3 = 5 to 7 mm of crowding; and 4 = more than 7 mm of crowding)

Cephalometric Measures. From each of the 20 participants, a lateral cephalometric film was taken in a standard fashion, including the use of a Picker GX 300 cephalostat. The settings were: KV major = 80 ± 10; KV minor = 84 ± 2; mA = 200 L; time = ⅒ second. The films were taken with the left side of the participant's head placed closest to the film while the participant looked straight ahead in the natural head posture[23] and kept the teeth in ICP with the orofacial musculature relaxed. Following landmark identification, 25 standard angular and linear measures were taken. The intraclass correlation coefficients[24] for the intraobserver and interobserver reliability of this tracing procedure, as determined on a sample of 20 randomly chosen films that were not included in the present study, were high and ranged from 0.866 to 0.995.

The points and planes that were used to determine the cephalometric measures in the present study are illustrated in Fig 1. Definitions and normative values are given by Rakosi,[25] McNamara,[26] and Owen.[27] The standard cephalometric measures are described below.

The following measures quantified the dental relationships:

- Maxillary incisor position, measured as the angle between the maxillary incisor axis through incision superius (Is) and the sella-nasion (SN) line (Is-SN); and mandibular incisor position, measured as the angle between the mandibular incisor axis, through incision inferius (Ii), and the mandibular plane (MP) (Ii-MP)
- Bimaxillary interincisal angle, measured posteriorly between the maxillary and mandibular central incisor axes (Is-Ii)

The following cephalometric measures were used to quantify the anteroposterior skeletal relationships:

- Maxillary position relative to the head, measured as the angle from S to N to point A (SNA angle); and mandibular position relative to the head, measured as the angle from S to N to point B (SNB angle)
- Bimaxillary skeletal angle, measured from A to N to B (ANB angle)
- Maxillary, mandibular, and chin positions relative to the head, measured parallel to Frankfort horizontal (FH) as the linear distance between a line running from N perpendicular to FH (ie, nasion-perpendicular) and A (maxillary position; N-A); between N-perpendicular and B (mandibular position; N-B); and between N-perpendicular and pogonion (Pg) (chin position; N-Pg)
- Mandibular position relative to the maxilla, measured parallel to FH as the linear distance between a line running from A perpendicular to FH and B (A-B)

The following cephalometric measures quantified the vertical skeletal relationships:

- Vertical aspect of the ramus, measured as the linear distance between craniofaciale (Cf) and gonion (Go) (Cf-Go)

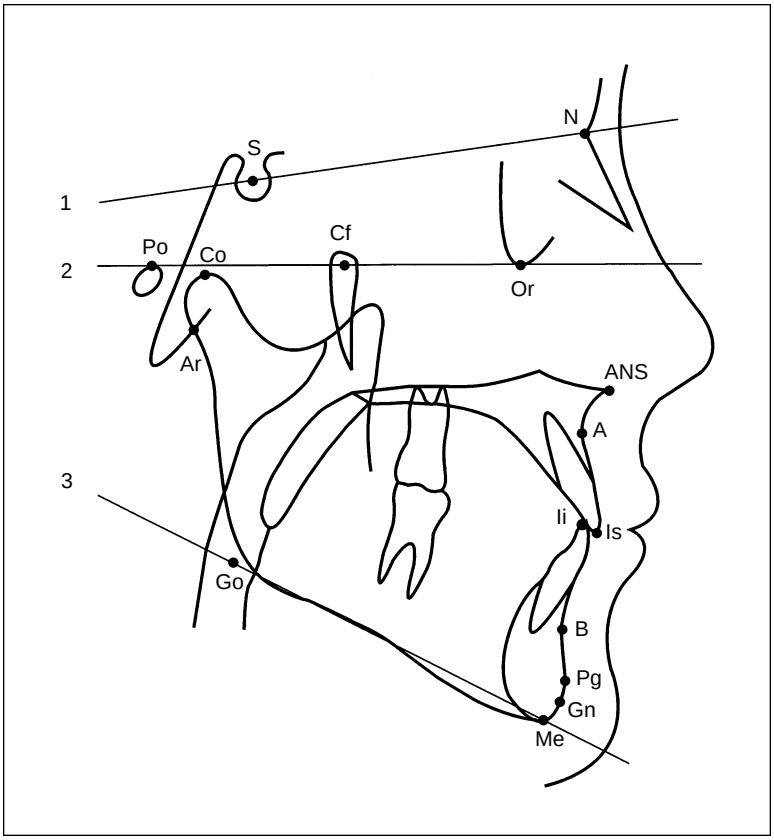

Fig 1 Points and planes that were used to determine the cephalometric outcome measures. For full definitions, see Rakosi,[25] McNamara,[26] and Owen.[27] A = point A (subspinale); ANS = anterior nasal spine; Ar = articulare; B = point B (supramentale); Cf = craniofaciale; Co = condylion; Gn = gnathion; Go = gonion; Ii = incision inferius; Is = incision superius; Me = menton; N = nasion; Or = orbitale; Pg = pogonion; Po = porion; S = sella. Planes: 1 = sella-nasion (SN) line; 2 = Frankfort horizontal (FH); 3 = mandibular plane (MP).

- Ratio between posterior and anterior facial height, measured from S to Go (PFH) and from N to Me (AFH), respectively (PFH/AFH)[28]
- Relative contribution of the upper facial height (UFH; linear distance between N and anterior nasal spine [ANS] perpendicular to FH) and the lower facial height (LFH; linear distance between ANS and menton [Me] perpendicular to FH) to the total facial height, calculated as: UFH % = UFH/(UFH + LFH) × 100 and LFH % = LFH/(UFH + LFH) ×100, respectively
- Effective mandibular length, measured as the linear distance between condylion (Co) and gnathion (Gn) (Co-Gn), minus the effective midfacial length (measured as the linear distance between Co and A [Co-A]) to provide a value for maxillomandibular differential (MD)
- Ratio of the LFH to the vertical aspect of the ramus (LFH/Cf-Go)
- Gonial angle (an expression of mandibular form), measured from Ar to Go to Me (Ar-Go-Me)
- Indication of vertical excess or deficiency, measured as the angle between FH and MP (FMA angle)
- Relative convexity or concavity of the profile, measured from N to A to Pg (N-A-Pg)

Neuroimaging

For 14 of the 20 participants (7 sleep bruxers, 7 non-bruxers; 8 men, 6 women; mean age = 28.9 ± 8.1 years), single-photon emission-computed tomography (SPECT) images were obtained 90 minutes after injection of 185 MBq of the specific, high-affinity D2 receptor antagonist ^{123}I-(S)-(-)-2-hydroxy-6-methoxy-N-((1-ethyl-2-pyrrolidyl) methyl)benzamide (iodine-123-iodobenzamide). Imaging took place as soon as the second polysomnographic recording was analyzed. Preparation of the radioligand and image acquisition, processing, and analysis were performed as described previously.[6] For SPECT outcome measure, the side-to-side difference between unilateral values of the striatal D2 binding potential was calculated.

Statistics

To test the first null hypothesis (no morphologic differences between sleep bruxers and non-bruxers), 2 independent-samples *t* tests were applied for the quantitative morphologic variables that were normally distributed (as tested with Kolmogorov-

Table 1a Quantitative Occlusal Variables (Mean ± SD) of 10 Non-Bruxers and 10 Sleep Bruxers*

	Maxillary measures		Mandibular measures		Bimaxillary measures	
Variable	Non-Bruxers	Bruxers	Non-Bruxers	Bruxers	Non-Bruxers	Bruxers
No. of teeth	14.2 ± 1.2	13.7 ± 1.5	14.2 ± 1.1	13.4 ± 1.4	28.4 ± 2.1	27.1 ± 2.5
Overbite (mm)					2.0 ± 1.7	1.4 ± 1.3
Overjet (mm)					2.1 ± 1.1	1.9 ± 0.9
RCP-ICP slide (mm)					0.1 ± 0.3	0.4 ± 0.3
Intercanine width (mm)	33.4 ± 1.6	32.1 ± 2.4	25.4 ± 1.3	26.7 ± 3.5		
Maxillary/mandibular					1.3 ± 0.1	1.2 ± 0.1
Intermolar width (mm)	44.8 ± 3.9	46.4 ± 2.5	40.1 ± 2.7	41.9 ± 3.0		
Maxillary/mandibular					1.1 ± 0.1	1.1 ± 0.1
Arch depth (mm)	36.6 ± 3.7	36.1 ± 2.4	32.1 ± 2.9	31.1 ± 2.0		
Maxillary/mandibular					1.1 ± 0.1	1.2 ± 0.1
Intercanine/intermolar width	0.8 ± 0.1	0.7 ± 0.04	0.6 ± 0.1	0.6 ± 0.1		
Intercanine width/arch depth	0.9 ± 0.1	0.9 ± 0.1	0.8 ± 0.1	0.9 ± 0.1		

*Taken both clinically and from study casts. No significant differences were found between non-bruxers and sleep bruxers ($P > 0.05$).

Table 1b Qualitative Occlusal Variables of 10 Non-Bruxers and 10 Sleep Bruxers*

Subject group	Angle's classification				Occlusal scheme		Anterior crowding (mandible only)†
	R. canine	L. canine	R. first molar	L. first molar	Right	Left	
Non-Bruxers	9 Class I, 1 Class II	9 Class I, 1 Class III	6 Class I, 2 Class II, 2 Class III	5 Class I, 2 Class II, 3 Class III	6 canine rise, 4 group function	6 canine rise, 4 group function	0: n = 2, 1: n = 5, 2: n = 2, 3: n = 1
Bruxers	9 Class I, 1 missing	4 Class I 5 Class II	5 Class I, 1 Class II, 1 Class III, 3 missing	3 Class I, 2 Class II, 5 missing	1 canine rise, 9 group function	1 canine rise, 9 group function	0: n = 5, 1: n = 4, 2: n =1

*Taken both clinically and from study casts. No significant differences were found between non-bruxers and sleep bruxers ($P > 0.05$).
†0 = no crowding; 1 = 1 to 3 mm of crowding; 2 = 3 to 5 mm; 3 = 5 to 7 mm; 4 = more than 7 mm.

Smirnov tests). Similarly, such comparisons were made for the standard sleep and oromotor variables. For the qualitative variables (ie, Angle's classifications, functional occlusal schemes, and anterior crowding), differences between both groups were analyzed with either the Pearson Chi-square test or, for 2 × 2 tables, the Yates' corrected Chi-square test.

The second null hypothesis (no significant correlations between neurochemical and morphologic factors) was tested by calculating Pearson's correlation coefficients between the side-to-side differences in striatal D2 receptor binding and the quantitative morphologic outcome measures. For the qualitative morphologic variables, Pearson's correlation coefficients were estimated with analyses of variance.

All statistical analyses were performed with significance set at the 0.05 probability level. To correct for multiple testing, the classical Bonferroni adjustment was applied.

Results

None of the standard sleep variables collected during the second polysomnographic recording differed significantly between the sleep bruxers and the non-bruxers (data not presented; values published previously[6,20]). The oromotor variables, however, had higher values (mean ± SD) among the sleep bruxers than among the non-bruxers: the sleep bruxers had 6.2 ± 3.5 episodes with oromotor activities per hour of sleep, as opposed to 1.1 ± 0.8 episodes/hour for the non-bruxers (*t* test; $P < 0.01$). The numbers for oromotor bursts/episode were 5.7 ± 3.1 for the sleep bruxers and 1.3 ± 1.1 for the non-bruxers (*t* test; $P < 0.01$).

In Tables 1 and 2, the means and standard deviations (or, for qualitative variables, the frequencies) are presented for the occlusal and cephalometric variables, respectively. None of the morphologic variables differed significantly between the 10 sleep bruxers and the 10 non-bruxers. The data of 14 of the 20 participants were fur-

Table 2 Cephalometric Variables (Mean ± SD) of 10 Non-Bruxers and 10 Sleep Bruxers

Variable	Non-Bruxers	Bruxers
Dental relationships		
Maxillary incisor position (Is-SN) (deg)	103.4 ± 7.9	95.3 ± 12.9
Mandibular incisor position (Ii-MP) (deg)	90.0 ± 6.6	91.2 ± 7.2
Bimaxillary interincisal angle (Is-Ii) (deg)	133.5 ± 10.9	135.0 ± 13.9
Anteroposterior skeletal relationships		
SNA angle (deg)	80.9 ± 2.4	79.7 ± 3.5
SNB angle (deg)	78.9 ± 2.7	77.3 ± 3.6
ANB angle (deg)	2.0 ± 2.6	2.4 ± 2.4
Maxillary position (N-A) (mm)	–0.1 ± 3.4	–0.5 ± 2.4
Mandibular position (N-B) (mm)	–3.9 ± 8.4	–5.2 ± 5.1
Chin position (N-Pg) (mm)	–1.9 ± 9.1	–3.2 ± 4.2
Mandible-maxilla (A-B) (mm)	3.8 ± 6.1	4.7 ± 4.6
Vertical skeletal relationships		
Vertical aspect of ramus (Cf-Go) (mm)	69.1 ± 7.0	71.4 ± 5.1
Posterior facial height (mm)	83.7 ± 8.6	84.3 ± 6.2
Anterior facial height (mm)	128.3 ± 9.3	128.9 ± 8.5
PFH/AFH (× 100%)	65.2 ± 4.0	65.6 ± 6.2
Upper facial height (mm)	57.2 ± 3.9	55.9 ± 4.5
Lower facial height (mm)	70.9 ± 7.6	72.1 ± 5.7
Upper facial height %	44.7 ± 2.8	43.6 ± 2.3
Lower facial height %	55.3 ± 2.8	56.4 ± 2.3
Mandibular length (Co-Gn) (mm)	124.7 ± 9.0	122.0 ± 6.3
Midfacial length (Co-A) (mm)	91.0 ± 6.1	89.8 ± 5.5
Maxillomandibular differential (mm)	33.7 ± 5.4	32.2 ± 4.8
LFH/Cf-Go (mm)	1.0 ± 0.1	1.0 ± 0.1
Gonial angle (Ar-Go-Me) (deg)	128.0 ± 5.1	125.1 ± 7.0
FMA angle (deg)	22.6 ± 10.5	24.3 ± 5.9
Profile convexity/concavity (N-A-Pg) (deg)	1.6 ± 5.2	2.0 ± 5.1

No significant differences were found between non-bruxers and sleep bruxers ($P > 0.05$).

ther analyzed, but no significant Pearson's correlation coefficients were found between the SPECT outcome measure and any of the occlusal and cephalometric variables ($0.010 < r < 0.575$; $P > 0.05$).

Discussion

The results of this retrospective, combined polysomnographic, morphometric, and neuroimaging study indicate that standard occlusal and cephalometric variables do not differ between sleep bruxers and non-bruxers. Moreover, the results suggest that these variables are probably not involved in the asymmetry in striatal D2 receptor distribution that was previously found in association with sleep-related bruxism.[6]

The presence of sleep-related oromotor activities was established by means of the current most recommended technique for the evaluation of such activities—namely, polysomnography in combination with audio and video recordings.[20] In the neuroimaging part of the present study, we aimed to include a broad range of oromotor activities. Therefore, the data of all 14 participants in our previous study[6] were included in the calculation of Pearson's correlation coefficients. Thus, the occlusal and cephalometric variables were correlated not only with the striatal D2 binding variables of the sleep bruxers, but also with those of the persons with non-bruxism sleep-related oromotor activities. In this way we included a broader range of oromotor activities and increased the low power of the "bruxists-only" sample.

Nevertheless, the statistical power of the neuroimaging part of the present study remained low. Cohen[29] considered $r = 0.50$ a large effect size. With 14 participants and without Bonferroni correction, there is only a 47% chance of finding such an effect size to be significant when α is set at 0.05. The statistical power of the morphologic comparisons between the sleep bruxers and the non-bruxers was low as well: sample size calculations

showed that at least 75 persons per group (and for some variables, even several thousand persons per group) would be required to reach statistical significance.

The values of the morphologic factors of the participants who received a sleep bruxism diagnosis did not differ significantly from those of the non-bruxers. In addition, the mean values of the cephalometric measures that were included in the present study fell within or only slightly outside the "mean ± 2 times SD" range (ie, the range that encompasses roughly 95% of the observations) of the normative values that were derived from the literature.[25–27] The absence of significant correlations between the morphologic variables and the SPECT outcome measure might thus be a result of the absence of important morphologic deformations. Gomez et al[18] found that placement of an acrylic cap on the mandibular central incisors caused an enhancement in apomorphine-induced non-functional masticatory activities in the rat, leading to a bilateral difference in dopa accumulation in the striatum when the cap was worn for a prolonged period.[19] An occlusal cap is a robust disharmony in comparison with, for example, the cutting of a mandibular incisor, which did not cause the above-mentioned dopaminergic effects in the striatum of the rat. More studies are needed to investigate the occurrence of striatal imbalances in dopamine distribution in humans when more robust occlusal disturbances are present and/or added. Because of its retrospective nature, our study did not include large discrepancies and deformations: persons who were missing more than 2 natural posterior teeth (not including the third molars) and persons with distinct orthodontic abnormalities (eg, deformities that would require surgery) were excluded from participation in our earlier study.[6] However, given the scarcity of extreme morphologic deformations in the general population, one might dispute the clinical relevance of a putative finding of positive correlations between a bilateral difference in striatal D2 receptor binding and such large occlusal discrepancies.

The fact that we could not demonstrate the findings of Gomez et al[18] and Areso et al[19] in the human situation might also reflect the axiom that animal studies cannot be compared directly with human studies, despite the obvious similarities in the mechanisms involved in the genesis of the oromotor activities (eg, involvement of the central dopaminergic system). Similarly, artificial occlusal disturbances, such as those built in by Gomez et al[18] and Areso et al,[19] cannot be compared directly with natural ones, since the latter are probably more likely caused by bruxism, rather than being the cause of bruxism.

Another explanation for the absence of significant correlations between the morphologic variables and the SPECT outcome measure, as well as for the absence of morphologic differences between the sleep bruxers and the non-bruxers, might be the use in the present study of standard sets of occlusal and cephalometric variables. Perhaps the use of additional measures to quantify morphology might have yielded some significant differences and/or correlations. For example, we could not verify the findings of Miller et al,[30] who found that condylar height differed between bruxers and non-bruxers, and those of Young et al,[31] who found differences in bizygomatic and cranial widths between both groups, because these measures were not included in the present study. In neither of these studies, however, was the presence or absence of bruxism, as assessed by self-report and a clinical examination, confirmed polysomnographically. This hampers the interpretation of their results. The absence of a polysomnographic confirmation of the bruxism status also hampers the interpretation of the study by Menapace et al,[32] although as in the present findings, these authors found no differences in dentofacial morphology between bruxers and non-bruxers. Waltimo et al focused primarily on tooth wear in relation to the morphology of the craniofacial structures.[33] In that study, a more rectangular form of the maxillary dental arch was found in patients with severe dental attrition than in control subjects. In addition, the authors found that patients with severe attrition had a more rectangular facial morphology than controls, together with an anteriorly rotated mandible, a small anterior facial height, and a large bimaxillary interincisal angle. These findings could not be confirmed in the present study, possibly because Waltimo et al[33] did not use polysomnography to classify their patients, and because the sleep bruxers in the present study were not selected on the basis of severe attrition. Instead, the participants in the present study were selected without any morphologic discrimination, thus eliminating selection bias as a confounding variable. As a consequence, however, and also because of the retrospective nature of the present study, attrition data were not available to allow for a comparison with the study of Waltimo et al.[33]

Given the low power of the present retrospective study, the results suggest that there are no morphologic differences between sleep bruxers and non-bruxers. Moreover, the results indicate that

morphologic factors are probably not involved in the asymmetry in striatal D2 receptor distribution that was previously found in association with sleep-related bruxism. Future studies with larger samples and an experimental and prospective character may yield more definitive conclusions.

Acknowledgments

We thank Dr J. G. C. Verheij, Academic Centre for Dentistry Amsterdam (ACTA), for his statistical advice, and Dr M. Naeije, ACTA, for his comments on the manuscript. This study was supported by the Canadian Medical Research Council (MRC), the Fonds de la recherche en santé du Québec (FRSQ), and the Netherlands Institute for Dental Sciences (IOT).

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