Prevalence and predictive factors of sleep bruxism in children with and without cognitive impairment

Cristina Batista Miamoto[a]
Luciano José Pereira[b]
Maria Letícia Ramos-Jorge[c]
Leandro Silva Marques[a]

[a] Department of Clinical Dentistry, Vale do Rio Verde University - UNINCOR, Três Corações, MG, Brazil.

[b] Departament of Physiology and Pharmacology, Federal University of Lavras - UFLA, Lavras, MG, Brazil.

[c] Department of Pediatric Dentistry, Federal University of Jequitinhonha and Mucuri Valleys - UFVJM, Diamantina, MG, Brazil.

Abstract: Studies have found a higher prevalence of sleep bruxism (SB) in individuals with cognitive impairment. The aim of this study was to identify the prevalence and factors associated with the clinical manifestation of SB in children with and without cognitive impairment. The sample was made up of 180 individuals: Group 1 – without cognitive impairment; Group 2 – with Down syndrome; Group 3 – with cerebral palsy. Malocclusions were assessed based on the Dental Aesthetic Index (DAI); lip competence was assessed based on Ballard's description. The bio-psychosocial characteristics were assessed via a questionnaire and clinical exam. Statistical analysis involved the chi-square test ($p \leq 0.05$) and multivariate logistic regression. The prevalence of bruxism was 23%. There were no significant differences between the groups ($p = 0.970$). Individuals with sucking habits (OR [95% CI] = 4.44 [1.5 to 13.0]), posterior crossbite (OR [95% CI] = 3.04 [1.2 to 7.5]) and tooth wear facets (OR [95% CI] = 3.32 [1.2 to 8.7]) had a greater chance of exhibiting SB. Sucking habits, posterior crossbite and tooth wear facets were identified as being directly associated with the clinical manifestations of bruxism.

Descriptors: Down Syndrome; Cerebral Palsy; Malocclusion; Sleep Bruxism.

Introduction

Sleep bruxism (SB) is defined as a parafunctional behaviour of the mandible, characterized by clenching and/or grinding of the teeth.[1] The aetiology and characteristics of bruxism have not yet been well defined.[2] However, studies point to different associated risk indicators, such as local, psychological, genetic, neurological, systemic and social factors.[2-5] The prevalence of SB ranges from 3% to 90% in adults and from 7% to 88% in children.[6]

The consequence most commonly associated with SB is tooth wear, which may compromise the dentition in a localized or generalized manner. Headaches, temporomandibular disorders, joint pain and pain during mastication are also associated with the manifestation of SB. Severe bruxism for a prolonged period of time can result in muscle hypertrophy (massester and/or temporalis). The treatment of acute conditions involves physiotherapy, "biofeedback" electromyography and medication for the relief of anxiety and sleep improvements. Long-term approaches include forms of reducing stress, changes in lifestyle, the control of habits,

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Corresponding author:
Leandro Silva Marques
E-mail: *lsmarques.prof@gmail.com*

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myorelaxation plates and night-time dental guards to protect the teeth and mastication system.[7-9]

Only a few studies have investigated the diverse aspects related to SB in patients with physical and/or cognitive impairments.[10-13] Recent studies found a higher prevalence in individuals with Down syndrome when compared to individuals without cognitive impairment. Additionally, SB appeared to affect individuals with cerebral palsy twice as much as control individuals. Thus, understanding the biopsychosocial aspects associated with the manifestation of SB is a determining factor in planning strategies to aid healthcare professionals in dealing with these conditions.

The aims of the present study were to identify the prevalence and possible determinants of SB in individuals without cognitive impairment, with Down syndrome and with cerebral palsy.

Methodology

The participants in the present study were individuals (with Down syndrome or cerebral palsy) who were receiving medical-hospital care at four specialized institutions. A control group without cognitive or physical impairments was selected from among patients awaiting orthodontic treatment at a Dental School.

The following inclusion criteria were required for participation in the present study: not having been submitted to any type of orthodontic intervention and confirmed diagnosis of the condition. The project received approval from the Ethics Committee. The parents/guardians signed terms of informed consent.

The data were collected via a questionnaire and a clinical examination of the individuals. For the diagnosis of malocclusion, the criteria of the Dental Aesthetic Index (DAI) were used, which furnishes four outcome possibilities:

- normal occlusion or mild malocclusion, for which treatment is unnecessary (DAI < 25);
- defined malocclusion, for which treatment is elective (DAI = 26 to 30);
- severe malocclusion, for which treatment is highly desirable (DAI = 31 to 35); and
- very severe or debilitating malocclusion, for which treatment is necessary (DAI ≤ 36).[14]

This variable was characterized as: absent, moderate or severe.

Malocclusions were clinically classified as Class I, Class II and Class III, based on the Angle classification. Crossbite was also included and identified during the physical examination. Lip competence was assessed based on Ballard's criteria: mandible in physiological resting position and lips in juxtaposition (sealed), with no contraction of the orbicular muscles of the mouth or the mentalis.[15] Lip incompetence was recorded if the child needed to vigorously contract the orbicular muscles of the mouth and the mentalis in order to seal the lips. Drooling was assessed based on the classification drafted by Thomas-Stonell and Greenberg (1988).[16] Drooling was evaluated since research has demonstrated a significant relationship between sialorrhoea during sleep and bruxism.[5] Records from the institutions and reports from parents/guardians were also assessed.

The signs and symptoms of SB were recorded taking into account: sibling or parental reports of grinding sounds (at least three times a week), the presence of shiny and polished facets on the incisors and/or first permanent molars (primarily based on palatal surface and incisal edges and working cusps, respectively), taking into account the time of eruption. Wear facets on deciduous teeth were not considered. The presence of SB was confirmed by both parental reports and the presence of tooth wear, since the latter is a cumulative sign.[4]

Statistical analysis involved chi-square and Fisher's exact tests ($p \leq 0.05$) for evaluating the association between independent variables on the prevalence of SB in the overall sample and in each of the groups separately as well. Multivariate logistic regression was employed in order to find predictive factors associated with SB in the overall sample.

Results

The prevalence of SB in the overall sample was 23%. There was a 23% prevalence of bruxism among the individuals with Down syndrome; 25% prevalence among those with cerebral palsy; and

Table 1 - Distribution of independent variables in relation to SB.

	Sleep bruxism (SB)		p-value
	Absent n (%)	Present n (%)	
Groups			
Control	45 (75.5)	15 (25.0)	0.970
Cerebral palsy	46 (76.7)	14 (23.3)	
Down syndrome	46 (76.7)	14 (23.3)	
Gender			
Male	65 (77.4)	19 (22.6)	0.709
Female	72 (75.0)	24 (25.0)	
Age			
More than 10 years	69 (78.4)	19 (21.6)	0.479
3 to 10 years	68 (73.9)	24 (26.1)	
Behaviour			
Calm	116 (74.8)	39 (25.2)	0.319
Agitated	21 (84.0)	4 (16.0)	
Premature birth			
No	109 (76.2)	34 (23.8)	0.673
Yes	24 (72.7)	9 (27.3)	
Sucking habits			
No	127 (79.9)	32 (20.1)	0.001
Yes	10 (47.6)	11 (52.4)	
Worn facets			
No	116 (79.5)	30 (20.5)	0.029
Yes	21 (61.8)	13 (38.2)	
Facial type			
Long face	35 (70.0)	15 (30.0)	0.083
Average	86 (81.9)	19 (18.1)	
Short face	16 (64.0)	9 (36.0)	
Breathing			
Nasal	100 (78.1)	28 (21.9)	0.320
Mouth	37 (71.2)	15 (28.8)	
Drooling			
Absent	115 (77.7)	33 (22.3)	0.375
Moderate	10 (76.9)	3 (23.1)	
Severe	12 (63.2)	7 (36.8)	
Type of malocclusion			
Class I	52 (76.5)	16 (23.5)	0.361
Class II	51 (81.0)	12 (19.0)	
Class III	34 (69.4)	15 (30.6)	
Posterior crossbite			
Absent	103 (79.8)	26 (20.2)	0.062
Present	34 (66.7)	17 (33.3)	
Severity of malocclusion			
Absent or mild	54 (79.4)	14 (20.6)	0.258
Moderate	22 (84.6)	4 (15.4)	
Severe	61 (70.9)	25 (29.1)	
Caries			
Absent	54 (77.1)	16 (22.9)	0.772
1 to 2 teeth with cavities	43 (78.2)	12 (21.8)	
3 to 7 teeth with cavities	40 (72.7)	15 (27.3)	

23% prevalence among those with malocclusion (23%). Table 1 shows the frequency distribution of the independent variables in relation to the dependent variable (the presence or absence of SB).

Having cognitive impairment was not predictive of SB (p = 0.970). Sucking habits, limited mouth opening, tooth wear facets, facial type and posterior crossbite achieved p-values of ≤ 0.20 and were therefore included in the multivariate regression model. This model revealed that individuals with sucking habits (p = 0.007) had a fourfold greater chance of exhibiting SB. Moreover, the presence of tooth wear facets (p = 0.015) and posterior crossbite (p = 0.017) increased the risk of SB by approximately threefold (Table 2).

The influence of independent variables in each group showed that sucking habits were associated with SB in both cognitive impairment condition groups and that worn facets were only associated with SB in the Down syndrome group. For the controls, facial type and posterior crossbite were associated with SB (Table 3).

Discussion

The occurrence of sleep bruxism has been reported with varying frequencies in the general population.[6,9] In the present sample, the conditions of Down syndrome or cerebral palsy had no influence over the presence of bruxism when compared to a group of individuals without cognitive impairment

Table 2 - Multivariate logistic regression (forward stepwise procedure) for SB.

	Sleep bruxism			
	Non-adjusted OR (95% CI)	p-value	Adjusted OR (95% CI)	p-value
Sucking habit				
No	1.00		1.00	
Yes	4.36 (1.71-11.8)	0.002	4.44 (1.50-13.09)	0.007
Worn facets				
No	1.00		1.00	
Yes	2.39 (1.08-5.33)	0.032	3.32 (1.26-8.73)	0.015
Posterior crossbite				
Absent	1.00		1.00	
Present	1.98 (0.96-4.08)	0.064	3.04 (1.22-7.57)	0.017
Facial type				
Long face	1.00		1.00	
Average	0.52 (0.24-1.13)	0.097	0.44 (0.17-1.11)	0.084
Short face	1.31 (0.47-3.63)	0.600	1.64 (0.39-6.95)	0.501

(p = 0.970). Previous studies reported that individuals with cognitive impairment are more prone to this parafunction due to spasticity (an increase in muscle tone at the time of muscle contraction caused by an abnormal neurological condition – the spastic muscles are more resistant to contraction than normal muscles and also require more effort to relax and remain contracted for a period of time).[10-13] However, recent studies have found that the prevalence of SB is similar between such individuals and control individuals matched for age.[6,17] As a result of different study methods, clinical criteria and the populations studied, the reported prevalence of SB varies significantly. Another factor linked to this variability is related to the diagnostic criteria for bruxism through reports from the individual himself/herself, clinical exams and reports from parents and/or partners. Children and individuals with special needs have difficulty identifying and/or reporting teeth grinding habits coherently. Thus, SB in the present study was diagnosed from the reports of parents/guardians and clinical evaluations.[9,18]

Tooth wear facets were significantly predictive of SB in the present study, especially in Down syndrome patients, thereby corroborating the notion that one of the most common consequences of clenching and/or grinding the teeth is localized and generalized tooth wear.[6] The diagnosis of tooth wear facets should be considered with caution. Although it can be objectively measured, it may not directly reflect the degree of current SB, as individuals can exhibit the effects of past habits that are no longer present at the time of evaluation, which could lead to an overestimation of prevalence. Likewise, individuals who recently acquired the habit may not yet exhibit signs of tooth wear, which could lead to an underestimation of bruxism.[19,20]

In the present study, sucking habits and posterior crossbite increased the chance of an individual exhibiting SB by approximately threefold. The evaluation of associations between variables in separate groups indicated that the former variable was more important in cognitively impaired children, whereas the latter was more important in control subjects. The relationship between parafunctional habits and malocclusion is well established in the literature.[21] Sucking habits are related to an increase in overjet and a Class II molar pattern, which has been demonstrated to be related to SB.[21,22] However, the relationship between malocclusion and SB remains controversial. In a previous study involving 975 individuals between 7 and 19 years of age, no causal relationship was found between malocclusions and the presence of this parafunction.[23] On the other

Table 3 - Distribution of independent variables in relation to SB in each of the groups separately.

	Sleep bruxism					
	Control		Cerebral palsy		Down syndrome	
	Absent n (%)	Present n (%)	Absent n (%)	Present n (%)	Absent n (%)	Present n (%)
Gender	p = 0.755[F]		p = 0.744[C]		p = 0.818[C]	
Male	15 (78.9)	4 (21.1)	22 (78.6)	6 (21.4)	28 (75.7)	9 (24.3)
Female	30 (73.2)	11 (26.8)	24 (75)	8 (25)	18 (78.3)	5 (21.7)
Age	p = 0.136[C]		p = 0.136[C]		p = 0.064[F]	
> 10 years	25 (83.3)	5 (16.7)	22 (73.3)	8 (26.7)	28 (87.5)	4 (12.5)
≤ 10 years	20 (66.7)	10 (33.3)	24 (80)	6 (20)	18 (64.3)	10 (35.7)
Behaviour	*		p = 0.112[F]		p = 0.582[F]	
Calm	45 (75)	15 (25)	28 (70)	12 (30)	43 (78.2)	12 (21.8)
Agitated	–	–	18 (90)	2 (10)	3 (60)	2 (40)
Premature Birth	p = 0.987[F]		p = 0.738[F]		p = 0.291[F]	
No	42 (75)	14 (25)	32 (74.4)	11 (25.6)	35 (79.5)	9 (20.5)
Yes	2 (66.7)	1 (33.3)	13 (81.3)	3 (18.8)	9 (64.3)	5 (35.7)
Sucking habits	p = 0.205[F]		p = 0.036[F]		p = 0.036[F]	
No	37 (78.7)	10 (21.3)	45 (80.4)	11 (19.6)	45 (80.4)	11 (19.6)
Yes	8 (61.5)	5 (38.5)	1 (25)	3 (75)	1 (25)	3 (75)
Worn facets	p = 0.566[F]		p = 0.511[F]		p = 0.002[F]	
No	42 (73.7)	15 (26.3)	34 (79.1)	9 (20.9)	40 (87)	6 (13)
Yes	83 (100)	0 (0)	12 (70.6)	5 (29.4)	6 (42.9)	8 (57.1)
Facial type	p = 0.036[C]		p = 0.836[C]		p = 0.102[C]	
Long face	8 (57.1)	6 (42.9)	24 (77.4)	7 (22.6)	3 (60)	2 (40)
Average	37 (82.2)	8 (17.8)	21 (75)	7 (25)	28 (87.5)	4 (12.5)
Short face	0 (0)	1 (100)	1 (100)	0 (0)	15 (65.2)	8 (34.8)
Breathing	p = 0.258[F]		p = 0.775[C]		p = 0.168[F]	
Nasal	43 (76.8)	13 (23.2)	21 (75)	7 (25)	36 (81.8)	8 (18.2)
Mouth	2 (50)	2 (50)	25 (78.1)	7 (21.9)	10 (62.5)	6 (37.5)
Drooling	*		p = 0.070[CL]		p = 0.581[CL]	
Absent	45 (75)	15 (25)	25 (86.2)	4 (13.8)	45 (76.3)	14 (23.7)
Moderate	–	–	9 (75)	3 (25)	1 (100)	0 (0)
Severe	–	–	12 (63.2)	7 (36.8)	–	–
Type of malocclusion	p = 0.486[C]		p = 0.132[C]		p = 0.306[C]	
Class I	24 (77.4)	7 (22.6)	16 (66.7)	8 (33.3)	12 (92.3)	1 (7.7)
Class II	19 (76)	6 (24)	20 (90.9)	2 (9.1)	12 (75)	4 (25)
Class III	2 (50)	2 (50)	10 (71.4)	4 (28.6)	22 (71)	9 (29)
Posterior crossbite	p = 0.019[F]		p = 0.264[F]		p = 0.744[C]	
Absent	42 (80.8)	10 (19.2)	39 (79.6)	10 (20.4)	22 (78.6)	6 (21.4)
Present	3 (37.5)	5 (62.5)	7 (63.6)	4 (36.4)	24 (75)	8 (25)

Table 3 *(continued)*

Severity of malocclusion	p = 0.189[CL]		p = 0.852[CL]		p = 0.486[C]	
Absent or mild	37 (78.7)	10 (21.3)	15 (78.9)	4 (21.1)	24 (85.7)	4 (14.3)
Moderate	4 (66.7)	2 (33.3)	2 (66.7)	1 (33.3)	8 (88.9)	1 (11.1)
Severe	4 (57.1)	3 (42.9)	29 (76.3)	9 (23.7)	14 (60.9)	9 (39.1)
Caries	p = 0.127[CL]		p = 0.456[CL]		p = 0.819[CL]	
Absent	14 (77.8)	4 (22.2)	20 (74.1)	7 (25.9)	20 (80)	5 (20)
1 to 2 teeth with cavities	21 (87.5)	3 (12.5)	10 (71.4)	4 (28.6)	12 (70.6)	5 (29.4)
3 to 7 teeth with cavities	10 (55.6)	8 (44.4)	16 (84.2)	3 (15.8)	14 (77.8)	4 (22.2)

[C] chi square test, [F] Fisher's exact test, [CL] chi square test with linear trend, *chi square test not performed.

hand, an epidemiological study involving 2529 individuals found that occlusal alterations such as the loss of posterior teeth, the incisor relationships and posterior crossbite were significantly associated with the presence of bruxism.[24] Moreover, Sari and Sonmez found a positive association between an overjet greater than 6 mm, an overbite greater than 5 mm and posterior crossbite and SB in children with mixed dentition.[25]

Altered contact between the occlusal surfaces of the teeth during the movements of the mastication is believed to create a greater tendency towards mandibular deviation, thereby favouring SB.[7] However, occlusal factors have been contested and psychological aspects are considered to be the principal factors involved in the occurrence of bruxism.[2] Such factors should not be overlooked in individuals with Down syndrome or cerebral palsy, as these individuals have a high degree of dependence and frequent conditions of anxiety, which could trigger SB.[26] Behaviour was assessed in the present study, but no significant association with SB was found, which may explain the similarity in the prevalence of SB between the patients with special needs and the controls. It is important to stress that the individuals with cognitive impairment may not have had a greater prevalence of parafunctional habits due to the fact that they frequented specialized care centres. In such places, individuals have access to multidisciplinary treatment with physiotherapists, speech therapists, physicians, dentists, psychologists and occupational therapists, who considerably contribute towards the prevention, identification and control of behaviours or dysfunctions and provide information to parents and families.[27,28]

The importance of the present study resides in highlighting the fact that a significant portion of individuals with Down syndrome and cerebral palsy exhibit parafunctional habits. The maintenance of such habits from childhood into adulthood may compromise health, leading to problems linked to the temporomandibular joint, facial muscles, tooth wear, periodontal disease and even tooth loss stemming from trauma.[29]

Conclusions

The prevalence of bruxism in individuals with Down syndrome and cerebral palsy was similar to that found in individuals without cognitive impairment (approximately 24%). The predictive factors for SB in the population studied were the presence of sucking habits, posterior crossbite and tooth wear facets.

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