SLEEP BRUXISM IN A CHILD WITH SLEEP DISTURBANCES AND PSYCHOSOCIAL FACTORS: A MULTIDISCIPLINARY CASE REPORT

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ABSTRACT

Introduction: Sleep bruxism (SB) is a parafunctional activity of the masticatory muscles characterized by clenching or grinding of the teeth during sleep. In children, SB may lead to severe tooth attrition, restoration failure, and impaired sleep quality, often influenced by psychosocial factors. Early and multidisciplinary intervention is crucial to prevent further oral and functional complications. **Methods:** This case report describes the comprehensive management of a child diagnosed with severe sleep bruxism. Clinical and radiographic examinations were conducted, and the Children's Sleep Habits Questionnaire (CSHQ – Abbreviated) was administered to assess sleep quality and behavioral patterns. Dental management included restorative treatment and occlusal protection, combined with sleep hygiene education and family counseling. **Case Report**: A 5-year-old girl presented with a damaged and dislodged bioflex crown, accompanied by severe attrition of the maxillary and mandibular anterior teeth. **Conclusion**: Severe sleep bruxism in children requires a multidisciplinary approach addressing dental, behavioral, and psychosocial factors. SSC restoration, acrylic occlusal splint, and sleep hygiene education proved effective in protecting dentition and improving the patient's sleep and well-being.

Keyword : Sleep bruxism; child; stainless steel crown; occlusal splint; sleep hygiene; psychosocial factors

INTRODUCTION

Sleep bruxism (SB) is defined as a repetitive jaw-muscle activity characterized by teeth clenching or grinding and/or mandibular bracing or thrusting during sleep. SB is currently recognized as a sleep-related movement disorder with a multifactorial etiology involving complex neurophysiological, psychological, and environmental interactions, rather than being attributed solely to occlusal discrepancies. Current evidence suggests that environmental exposures and genetic predisposition, together with central neurotransmitter imbalances regulating masticatory muscle activity, contribute to its development. In children, SB often co-occurs with alterations in sleep architecture and has been associated with anxiety, excessive screen use, high sugar intake, and sleep-disordered breathing.[1]

In pediatric populations, sleep bruxism warrants particular attention because it overlaps with critical periods of dental and craniofacial growth. Recurrent parafunctional activity during these years can accelerate tooth wear, precipitate restoration fractures, contribute to temporomandibular disorders, and impair oral function. Reported prevalence varies widely (≈3.5–46%), largely reflecting differences in case definitions and assessment methods. For example, a Brazilian cohort of preschoolers estimated a prevalence of about 14%, whereas an Indonesian survey of adolescents aged 12–15 years reported 4.96%, underscoring regional heterogeneity and the diagnostic limitations that hinder cross-study comparisons.[2,3]

The etiology of pediatric SB is multifactorial. Neurobiological contributions include dysregulation of dopaminergic and serotonergic signaling that governs rhythmic masticatory muscle activity and is temporally coupled with cortical micro-arousals and autonomic surges during non-REM sleep. Beyond biological mechanisms, psychological and emotional stressors such as anxiety, sibling rivalry, and diminished parental attention play a significant role in the persistence of SB. Environmental and behavioral factors, including poor sleep hygiene, irregular sleep schedules, and exposure to noise or light during sleep, have also been shown to exacerbate the condition.[4-6]

The present case illustrates these multifactorial dimensions in a 5-year-old female who presented with severe anterior attrition and fractured restorations associated with persistent bruxism. The patient also exhibited delayed sleep onset, nocturnal restlessness, and daytime fatigue, as indicated by findings from the Children's Sleep Habits Questionnaire (CSHQ). Psychosocial stressors, including reduced maternal attention following the birth of a sibling and limited peer interactions, appeared to further aggravate her bruxism. This case underscores the importance of a multidisciplinary approach that integrates restorative care, occlusal protection, and psychosocial support in order to preserve dental structures and improve overall quality of life in pediatric patients with SB.[7-9]

CASE REPORT

The case report was written in accordance with the CARE case report guidelines.

2.1 Patient

In February 2025, a 5-year-old female patient was brought to the pediatric dental clinic with the primary complaint of a damaged Bioflex crown on the mandibula left. The child's mother reported that the crown had detached and fractured, likely due to the patient's persistent tooth grinding habit during sleep. The mother's main concern was to restore the damaged teeth and to prevent further wear. The patient had no known systemic medical conditions and no significant history of trauma. Family history revealed that the patient is the second of four siblings. Since the age of two, shortly after the birth of her younger sibling, the mother's attention to the patient had decreased due to caregiving demands for the newborn. The patient often expressed feelings of loneliness, as her older siblings due to a considerable age gap were unwilling to play with her, and she frequently cried when teased by them. Psycho-socially, the child was reported to be very active during nighttime, staying awake past midnight while other family members slept. During daytime, she was prone to falling asleep during activities. The mother also reported that the patient had difficulty initiating sleep and often woke feeling restless. Previously, the patient had received Bioflex crowns on the mandibula left primary to manage severe attrition caused by bruxism. However, due to ongoing parafunctional grinding during sleep, the crowns were damaged and failed within a relatively short period. No prior occlusal splint therapy or behavioral management for bruxism had been attempted before this visit.

2.2 Patient Examination

At the initial adaptation visit, extraoral assessment showed symmetrical facial contours with no asymmetry. Maximum unassisted interincisal opening was within age-appropriate norms; a mild rightward terminal deviation was noted on full opening, which corrected on closure. No joint sounds (clicking/crepitus) were detected on bilateral TMJ palpation or auscultation. Palpation of the masseter and temporalis muscles elicited mild, non-radiating tenderness consistent with parafunctional overuse, with no pain at rest. Intraorally, there was severe attrition of the maxillary primary incisors RA and RB with marked loss of clinical crown height. Early occlusal wear facets were present on the maxillary posterior teeth. The Bioflex crown on RB was fractured and partially dislodged. The remaining dentition was free of caries (ICDAS code 0), and the gingival tissues appeared healthy.

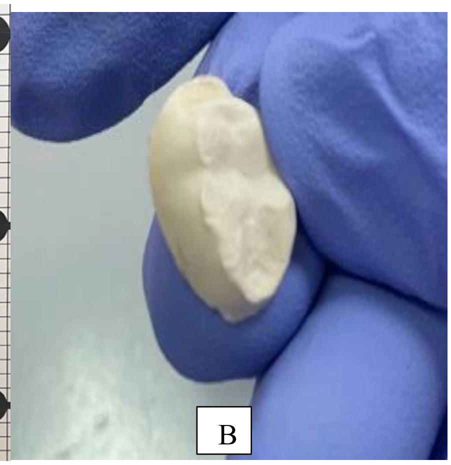

Figure A. Patient Profile Figure
Figure B. Bioflex crown is damaged

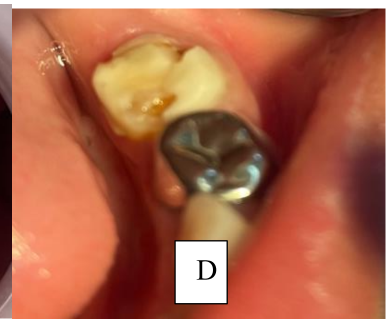

Figure C. severe dental attrition of the primary incisors RA and RB
Figure D. Intraoral photo of 75 teeth

2.3 Diagnostic Assessment

The diagnosis of probable pediatric sleep bruxism was established based on a combination of parental observations, clinical findings, and validated sleep behavior assessment. The patient's mother reported frequent nocturnal tooth grinding, often audible during the night, accompanied by restless sleep and delayed sleep onset, with the child typically falling asleep after midnight. Clinical examination revealed severe attrition of the maxillary primary incisors (RA and RB) with substantial crown height loss, fractured and partially dislodged Bioflex crowns, early attrition of posterior teeth, consistent with chronic nocturnal masticatory activity. Sleep behavior was assessed using the Children's Sleep Habits Questionnaire (CSHQ – abbreviated) (Table 1), which demonstrated elevated scores in bedtime resistance (43), sleep behavior disturbances (33), and morning wakefulness difficulties (14), indicating significant impairment in sleep initiation, quality, and daytime functioning, while waking during the night was scored at 2, suggesting relatively infrequent nocturnal awakenings. These findings are consistent with prior studies linking poor sleep quality and behavioural sleep disturbances to increased rhythmic masticatory muscle activity during sleep   Although polysomnography (PSG), the gold standard for confirming sleep bruxism, was not available, the clinical and behavioral findings fulfilled the International

Consensus criteria for probable sleep bruxism. Differential diagnoses, including awake bruxism, other sleep-related movement disorders, and parafunctional habits, were considered but excluded based on history and examination, reinforcing the diagnosis of clinically significant pediatric sleep bruxism.

Table 1. CSHQ subscale scores for the patient

	Score	**Interpretation**
Bedtime Resistance	43	Markedly elevated – difficulty initiating sleep
Sleep Behaviour Disturbance	33	Elevated – restless or disrupted sleep patterns
Waking During the Night	2	Low – infrequent nocturnal awakenings
Morning Wakefulness	14	Elevated – difficulty waking and morning lethargy

The CSHQ total score is obtained by summing the responses to all scored items, yielding a possible range from 33 to 99. Each item is rated according to the frequency of the behavior, with responses assigned 1, 2, or 3 points. A score of 1 indicates that the behavior occurs rarely, whereas a score of 3 reflects frequent occurrence. Higher scores correspond to greater severity or frequency of negative sleep-related behaviors. A total score exceeding 41 has been reported to identify approximately 80% of children with clinically diagnosed sleep disorders, making it a useful screening threshold in pediatric populations.

2.4 Therapeutic Intervention

The therapeutic intervention was delivered over four clinical sessions. At the first visit (18 February 2025), the fractured Bioflex crowns on the mandibular primary teeth were removed, followed by tooth preparation and placement of stainless-steel crowns (SSC) to provide durable resistance against the excessive occlusal forces associated with bruxism. Caregivers were given individualized oral hygiene instructions to promote long-term restorative success. During the second visit (12 April 2025), an alginate impression (Hydrogum 5, Zermack, Italy) and wax bite registration were obtained for the fabrication of a maxillary acrylic occlusal splint with full occlusal coverage, prescribed to protect the dentition and reduce mechanical loading on anterior restorations. At the third appointment (14 May 2025), the splint was tried in and adjusted to ensure balanced bilateral contacts. The caregiver received instructions regarding consistent nighttime use, appliance hygiene, and safe storage.During the follow-up appointment (13 June 2025), the splint was found to be intact, with no additional fractures or significant wear observed. Minor occlusal adjustments were performed to maintain comfort and optimal fit, and the patient demonstrated improved protection of the anterior teeth. This staged approach combined restorative treatment with protective appliance therapy to address both the structural and functional consequences of pediatric sleep bruxism.

Therapeutic intervention also addressed psychosocial factors and sleep habits, given the strong association between emotional stress, disrupted sleep, and pediatric bruxism. Caregiver counselling was initiated to improve parent–child interaction, with emphasis on providing consistent attention and emotional support, especially in light of the patient's feelings of social isolation following changes in family dynamics. Structured daily routines were recommended to reduce evening hyperactivity and improve emotional stability. Sleep hygiene education was provided to the caregiver, including establishing a consistent bedtime before 09:00 PM, creating a quiet and dimly lit sleep environment, limiting stimulating activities such as screen time before bed, and avoiding caffeine-containing foods or beverages. Bedtime relaxation strategies, such as reading aloud or calming music, were encouraged to reduce pre-sleep arousal. In addition, the caregiver was advised to monitor for signs of persistent sleep disturbance, emotional distress, or social withdrawal, with a plan for referral to a pediatric psychologist if symptoms persisted. This multidisciplinary approach aimed to optimize sleep quality, reduce nocturnal parafunctional activity, and address the underlying psychosocial contributors to the patient's bruxism.

Tabel 2. Timeline Historical

Date	Event
18 Feb 2025	First visit: Examination and replacement of damaged Bioflex crowns with stainless steel crowns (SSC) on RA and RB
12 Apr 2025	Second visit: Alginate impression for fabrication of acrylic occlusal splint
14 May 2025	Third visit: Insertion and adjustment of acrylic occlusal splint
13 Jun 2025	Fourth visit: One-month follow-up and evaluation of splint adaptation

Figure 5. Occlusal Splint

2.5 Follow-Up Outcomes

At the one-month follow-up, the stainless-steel crowns remained stable, with no additional loss of anterior tooth structure. The maxillary occlusal splint was well tolerated, and caregiver compliance with nightly use was satisfactory. Although parental reports indicated that bruxism episodes persisted, progression of tooth wear was effectively arrested, and the child reported fewer complaints of morning jaw discomfort. Sleep behavior showed partial improvement, with an earlier bedtime achieved on most nights and fewer nighttime awakenings, though occasional bedtime resistance persisted. Follow-up of the occlusal splint was tailored to the patient's young age and ongoing craniofacial growth. An initial review within two weeks evaluated comfort, retention, and occlusal balance, followed by monthly visits to monitor eruption patterns and identify potential interferences with jaw development. Minor occlusal refinements were performed to preserve balanced coverage and prevent over-eruption of uncovered teeth. Given the dynamic changes during the mixed dentition stage, the splint was prescribed for short-term use, with an anticipated duration of three months before reassessment for possible remaking or discontinuation. Parents were instructed to maintain consistent nightly use, ensure proper hygiene of the appliance, and promptly report any discomfort, fracture, or bite alteration. Close clinical monitoring, combined with ongoing counseling on sleep hygiene and behavioral strategies, aimed to protect the dentition from further attrition while minimizing adverse effects on growth and occlusion.

DISCUSSION

Bruxism presents a multifactorial etiology, involving an interplay of biological, psychological, hereditary, and extrinsic factors rather than a single isolated cause. Biological mechanisms in the etiology of bruxism involve disturbances in central neurotransmitter systems, particularly dopaminergic and serotonergic pathways, which are known to modulate the motor activity of the masticatory muscles. Disruptions in these systems have been implicated as potential contributors to the pathophysiology of bruxism, as altered dopamine and serotonin signaling can influence arousal thresholds, motor excitability, and rhythmic masticatory muscle activity during sleep. Recent neurobiological evidence reinforces this view, highlighting that dysfunction in dopaminergic and serotonergic circuits may act as a central trigger for sleep bruxism, rather than peripheral occlusal factors alone. In our patient, the clinical picture of late sleep onset, restless nights, and morning tiredness reported in the CSHQ aligns with the literature linking poor sleep quality and arousal instability with SB episodes.[10-11]

Chronic stress and psycho-emotional disturbances have been highlighted as the most significant contributors, acting through the limbic system and autonomic pathways to increase muscle tone and parafunctional masticatory activity. In children, the immaturity of neuroregulatory systems amplifies vulnerability, with stressors such as family dynamics, reduced parental attention, and limited coping capacity predisposing them to sleep bruxism. Our patient demonstrated typical psychosocial triggers, including reduced parental attention after the birth of a sibling, which align with literature identifying psycho-dependent bruxism as one of the most destructive forms, often associated with sleep disturbances, headaches, and dentoalveolar wear.[12]

Sleep disturbances also played a central role. The Children's Sleep Habits Questionnaire (CSHQ) revealed patterns of late sleep onset, restless nights, and morning tiredness. These findings align with the literature showing that arousal instability and poor sleep quality are closely associated with rhythmic masticatory muscle activity (RMMA) and the occurrence of sleep bruxism episodes. Costa et al. (2021) emphasize that the etiology of sleep bruxism in preschoolers is multifactorial, involving biological, psychological, hereditary, and extrinsic contributors such as poor sleep quality and mouth breathing brux. In our 5-year-old patient, the persistent bruxism episodes coincided with restless sleep and parental reports of poor nighttime behavior on the CSHQ, supporting the role of disturbed sleep architecture as a key trigger. Thus, in our patient, sleep fragmentation likely perpetuated bruxism behavior and contributed to severe dental attrition.[13]

Recent literature emphasizes that the diagnosis of bruxism should not rely solely on caregiver reports and clinical observation, but ideally be complemented by objective assessments such as electromyography (EMG) to monitor masticatory muscle activity and polysomnography (PSG) to document nocturnal behaviors in a sleep laboratory setting. However, the implementation of EMG or PSG in pediatric populations remains limited, as these tests are expensive, require specialized facilities, and involve the child being monitored outside the home environment, which can be challenging for both families and clinicians.[14]

Determining the clinical management strategy for a patient with pediatric sleep bruxism requires careful integration of dental, behavioral, and psychosocial considerations. In this case, the presence of severe anterior attrition and fractured restorations necessitated a restorative approach to preserve tooth structure, for which stainless steel crowns were selected due to their superior durability under heavy occlusal forces compared with composite or bioflex crowns.To further protect the dentition and redistribute occlusal load, a maxillary acrylic occlusal splint was fabricated and delivered, with regular follow-up adjustments to accommodate growth, given the patient's psychosocial background, including reduced parental attention and sleep onset difficulties, counselling and sleep hygiene recommendations were also incorporated, reflecting evidence that psychosocial instability and sleep disturbances act as significant risk factors for pediatric bruxism. A multidisciplinary management strategy, therefore, was adopted combining restorative dentistry, occlusal protection, and behavioral guidance to stabilize the stomatognathic system, mitigate further tooth wear, and address underlying etiological factors in line with current consensus recommendations.[15]

Most clinicians approach the treatment of bruxism empirically. It is believed that in children between the ages of three and five years, the chewing surfaces of the teeth undergo physiological wear to allow for the growth and development of the jaws. The incidence of bruxism has also been shown to decrease from approximately age 9 to 10 years, supporting the belief that most children with bruxism will not exhibit symptoms of the SB during adolescence and adulthood. Researchers have observed that the symptoms of SB in children with Down syndrome also decrease with age (approximately 12 years) .[16]

Recent evidence shows that lifestyle factors, particularly excessive screen-time and high sugar consumption, are significantly associated with increased frequency of paediatric sleep bruxism (SB) (Restrepo et al., 2021). In their cross-sectional study of 440 children aged 4–8 years, Restrepo and colleagues demonstrated that children consuming sugar daily and engaging in more than two hours of screen exposure per day had more than double the odds of exhibiting SB compared with their peers. This finding is clinically relevant to our patient, who presented with severe attrition of the anterior dentition and poor sleep quality confirmed by the CSHQ. Although parental reporting in this case did not specifically indicate excessive sugar intake or prolonged screen use, the child's psychosocial background and irregular sleep patterns could have acted in a similar way to disturb arousal regulation and increase susceptibility to SB episodes. These associations underscore the importance of evaluating and counselling families about dietary habits and digital media exposure as part of a comprehensive management plan for paediatric bruxism.[17]

A systematic review and meta-analysis by de Souza Melo et al. demonstrated that sleep bruxism (SB) is significantly associated with an increased risk of ceramic restoration failure, particularly for anterior restorations such as laminate veneers. The analysis indicated a hazard ratio as high as 7.74 in bruxers, suggesting that patients with SB were almost eight times more likely to experience restoration failure compared with non-bruxers. Nevertheless, the overall meta-analysis revealed no consistent association with all types of ceramic restorations due to high heterogeneity among studies and the low certainty of available evidence. In the present case, the paediatric patient exhibited severe SB that resulted in fractured Bioflex crowns and pronounced attrition of the primary maxillary incisors. excessive occlusal loading from parafunctional activity predisposes restorative materials to premature failure. In children, where enamel and restorative materials are more vulnerable, bruxism acts as a potent risk factor for accelerated breakdown of restorations. From a clinical perspective, these findings underscore the necessity of selecting restorative materials with higher resistance to occlusal load in paediatric bruxism patients. In this case, stainless steel crowns (SSC) were favoured over Bioflex crowns to provide superior durability under parafunctional forces.[18]

Based on the systematic review by Hardy & Bonsor et al, occlusal splints are widely prescribed to protect teeth and reduce muscle hyperactivity in bruxism. The review found that while splints may reduce masseter muscle activity and tooth wear in some patients. The use of occlusal splints in paediatric bruxism remains controversial, as systematic reviews highlight limited and heterogeneous evidence regarding their ability to reduce sleep bruxism episodes or muscle activity. However, their clinical relevance in children lies not solely in suppressing parafunctional activity but in providing protection to vulnerable dentition during growth. Although the splint may not eliminate nocturnal bruxism entirely, it provides functional protection during critical stages of dental and craniofacial development. Long-term monitoring is essential, particularly since occlusal splints must be periodically adjusted or remade to accommodate growth in children. In our patient, the acrylic occlusal splint successfully prevented further crown fractures and reduced functional load on newly placed stainless steel crowns, stabilizing the occlusion. While behavioural and psychosocial interventions remain important adjuncts, pharmacological options are generally unsuitable in young children, making the splint a pragmatic first-line therapy.[19]

The limitation of this study is limited by the absence of objective diagnostic tools such as polysomnography (PSG) or electromyography (EMG), which are considered the gold standard for confirming sleep bruxism. The diagnosis was based primarily on parental reports, clinical signs, and questionnaire-based assessments. Additionally, the short follow-up period restricts the ability to evaluate the long-term effectiveness of stainless steel crowns and occlusal splint therapy in preventing further tooth wear. Psychosocial factors were identified through history-taking rather than standardized psychological evaluation, which limits the depth of understanding regarding the behavioural and emotional contributors to bruxism in this child. Therefore, while the interventions used in this case were successful in stabilizing the condition, broader conclusions about their efficacy should be made with caution.

CONCLUSIONS

This case highlights the importance of a multidisciplinary approach in the management of pediatric sleep bruxism. Severe dental attrition and fractured restorations in a 5-year-old child were successfully stabilized through the placement of stainless-steel crowns and the delivery of an acrylic occlusal splint, which provided effective protection of the developing dentition. The use of validated sleep assessment tools, such as the CSHQ, further revealed significant sleep disturbances, emphasizing the contribution of psychosocial and behavioral factors to the persistence of bruxism. By combining restorative intervention, occlusal protection, and parental guidance on sleep hygiene, the treatment strategy not only preserved dental structures but also addressed the multifactorial etiology of bruxism. Importantly, because occlusal splints require periodic adjustment in children to accommodate craniofacial and dental arch growth, regular follow-up visits are essential to ensure optimal fit, function, and prevention of potential adverse effects. Continued monitoring and tailored interventions remain critical to achieving long-term oral health and functional stability in pediatric patients with sleep bruxism.

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Informed Consent Statement: Informed consent was obtained from all subjects involved in the study. Written informed consent has been obtained from the patients' legal guardians to publish this paper.

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REFERENCES

1. Restrepo-Serna C, Winocur E. Sleep bruxism in children, from evidence to the clinic: a systematic review. *Front Oral Health*. 2023;4:1166091. DOI: 10.3389/froh.2023.1166091
2. Machado, E., Dal-Fabbro, C., Cunali, P. A., & Kaizer, O. B. (2014). Prevalence of sleep bruxism in children: A systematic review. *Dental Press Journal of Orthodontics, 19*(6), 54–61. https://doi.org/10.1590/2176-9451.19.6.054-061.oar

3. Nurwindasari D, et al. Prevalensi Bruxism pada Anak Usia 12-15 tahun di Masa Pandemi COVID-19 *Eur J Mol Clin Med*. 2021;8(3):1717-1724.
4. Kuang B, Li D, Lobbezoo F, de Vries R, Hilgevoord A, de Vries N, Huynh N, Lavigne G, Aarab G. Associations between sleep bruxism and other sleep-related disorders in adults: a systematic review. *Sleep Med Rev*. 2022;62:101592. 10.1016/j.sleep.2021.11.008
5. Marceliano CRV, Gavião MBD. Possible sleep bruxism and biological rhythm in school children. *Clin Oral Investig*. 2023;27(6):2979-2992. https://doi.org/10.1007/s00784-023-04900-y
6. Soares JP, Giacomin A, Cardoso M, Serra-Negra JM, Bolan M. Association of gender, oral habits, and poor sleep quality with possible sleep bruxism in schoolchildren. *Braz Oral Res*. 2020;34:e019. DOI: 10.1590/1807-3107bor-2020.vol34.0019
7. Manfredini D, Winocur E, Guarda-Nardini L, Paesani D, Lobbezoo F. Epidemiology of bruxism in adults: a systematic review of the literature. *J Orofac Pain*. 2015;29(1):26-41. DOI: 10.11607/jop.921
8. Owens JA, Spirito A, McGuinn M. The Children's Sleep Habits Questionnaire (CSHQ): psychometric properties of a survey instrument for school-aged children. *Sleep*. 2000;23(8):1043-1051. https://doi.org/10.1093/sleep/23.8.1d
9. Serra-Negra JM, Paiva SM, Fulgêncio LB, et al. Environmental factors, sleep bruxism and sleep characteristics in children. *Cranio*. 2020;38(6):405-410. DOI: 10.1016/j.sleep.2013.08.797
10. Lobbezoo F, Naeije M. Bruxism is mainly regulated centrally, not peripherally. J Oral Rehabil. (2001) 28:1085–91. DOI: 10.1046/j.1365-2842.2001.00839.x
11. Uchima Koecklin KH, Aliaga-Del Castillo A, Li P. The neural substrates of bruxism: current knowledge and clinical implications. *Front Neurol.* 2024;15:1451183. https://doi.org/10.3389/fneur.2024.1451183
12. Wieckiewicz M, Paradowska-Stolarz A, Wieckiewicz W. *Psychosocial aspects of bruxism: the most paramount factor influencing teeth grinding*. BioMed Res Int. 2014;2014:469187. https://doi.org/10.1155/2014/469187
13. Costa SV, de Souza BK, Cruvinel T, Oliveira TM, Neto NL, Machado MAAM. Factors associated with preschool children's sleep bruxism. *Cranio*. 2021;39(6):518–525. 10.1080/08869634.2021.1903663
14. Lobbezoo F, Ahlberg J, Raphael KG, Wetselaar P, Glaros AG, Kato T, et al. International consensus on the assessment of bruxism: Report of a work in progress. *J Oral Rehabil.* 2018 Nov;45(11):837–844. https://doi.org/10.1111/joor.12663
15. Jokubauskas L, Baltrušaitytė A. Efficacy of biofeedback therapy on sleep bruxism: A systematic review and meta-analysis. *J Oral Rehabil*. 2018;45(6):485–95. DOI: 10.1111/joor.12628
16. Firmani M, Reyes M, Becerra N, Flores G, Weitzman M, Espinosa P. Sleep bruxism in children and adolescents. *Rev ChilPediatr*. 2015;86(5):373–379. https://doi.org/10.1016/j.rchipe.2015.05.001

17. Restrepo C, Santamaría A, Manrique R. *Sleep bruxism in children: relationship with screen-time and sugar consumption.*Sleep Med X. 2021;3:100035. https://doi.org/10.1016/j.sleepx.2021.100035
18. de Souza Melo G, Batistella EA, Bertazzo-Silveira E, Gonçalves TMSV, de Souza BDM, Porporatti AL, Flores-Mir C, De Luca Canto G. Association of sleep bruxism with ceramic restoration failure: A systematic review and meta-analysis. *JProsthet Dent*. 2018;119(3):354–362. https://doi.org/10.1016/j.prosdent.2017.07.005
19. Hardy RS, Bonsor SJ. *The efficacy of occlusal splints in the treatment of bruxism: A systematic review*. *J Dent*. 2021;108:103621. https://doi.org/10.1016/j.jdent.2021.103621