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Interrelation between Sleep Bruxism and Obstructive Sleep Apnea: Literature Review

Nabiel Alghazali^{1*}, Mohammed I. Malki², Mesfer Ibrahim A. Alshehri³, Wafa H. Alfaifi⁴, Afnan J. Khardali⁴, Hessah M. M. Majrashi⁴, Shaima M. A. Al-hussein⁴ and Hala M. Maghrabi⁵

¹School of Dentistry, Prosthodontics department, Aleepo University, Syria, Saudi Arabia. ²College of Medicine, QU Health, Qatar University, P. O.Box 2713, Doha, Qatar. ³Ministry of Health, Abha, Asseir, SA, Saudi Arabia. ⁴College of Dentistry, Jazan University, Jazan, SA, Saudi Arabia. ⁵Private Practice, Jeddah, SA, Saudi Arabia.

Authors' contributions

This work was carried out in collaboration among all authors. All authors read and approved the final manuscript.

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Review Article

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ABSTRACT

Obstructive sleep apnea (OSA) is a clinical risk factor for sleep bruxism (SB). Although no clear causative link has been defined, both conditions are inter related to sleep-related arousal reactions [1]. A literature review was conducted on PubMed and ScienceDirect databases for 2000–2021 period. The majority of studies revealed an association between OSA and SB events.

Keywords: Bruxism; sleep apnea.

1. BACKGROUND

In 2012, bruxism was defined internationally as "a repetitive jaw muscle activity characterized by clenching or grinding of the teeth and/or by bracing or thrusting of the mandible" [2]. This condition was classified as awake or sleep bruxism (SB) [1,2].According to the International

*Corresponding author: E-mail: nabeelgza6@gmail.com;

Classification of Sleep Disorders clinical criteria, SB includes the presence of regular or repeated tooth-grinding sounds happening during sleep and one or more of the following clinical aspects: [1] abnormal tooth wear resulting from tooth grinding during sleep and [2] transient morning jaw muscle pain or fatigue, temporal headache, and/or jaw locking upon awakening and reported as a cause of tooth grinding during sleep [2].

Malocclusion and occlusal interferences are initially considered as the primary etiological factor for bruxism; thus, dental practitioners tend to perform occlusal adjustments or occlusal splints to overcome such problem [3,4]. However, recent data revealed that SB is no longer attributed to occlusal problems or is not any more related solely to stress, anxiety, or depression [5-7]. The most recent hypotheses on the primary etiology of SB are in favor to central and autonomic nervous systems in the genesis of masticatory muscle activity during sleep [8].

Smoking, usage of some certain medications, and breath related problems can also be risk factors for SB [9]. Obstructive sleep apnea (OSA) syndrome has always been considered as one of the primary risk factors for SB [10,11]. This, paper aims to review literature to determine the relation between OSA and SB.

2. REVIEW RESULTS

2.1 Definiens and Mechanisms of OSA and SB

A new survey revealed that SB occurs repeatedly in 6%–8% and occasionally in 7%–13% of Canadian population. This condition is a clinical risk factor for tooth wear, tooth damage, and fractures, mastication's muscle spasm, fatigue and pain, headaches, and temporomandibular joint disorders [12]. Feu et al. conducted a systematic review and revealed that SB occurrence can be associated with esophageal acidification, smoking, and disturbances in the central dopaminergic system [13].

SB mechanism was illustrated [6–8] mostly rhythmic masticatory muscle activity (RMMA) episodes in relation to sleep micro-arousals associated with brain and cardiac activity [14-16]. Some studies showed that certain episodes of bruxism-related motor activity are not necessarily pathological [16-18]. OSA is defined as repetitive episodes of upper air way obstruction during sleep that generally result in a decreased blood oxygen saturation. This sleep disruption will lead to a body response characterized by brain arousal and sympathetic system activation [19,20].

OSA is generally caused by anatomical alterations that frequently manifest as then arrowing or blockage of pharyngeal upper airways while sleeping [20]. This condition is now considered as a serious public health problem because of its frequency and consequences, including excessive daytime sleepiness, cardiopulmonary problems, and type 2 diabetes [20-22].

2.2 Prevenances and Sources of OSA

The prevalence of OSA was 74.7% in males and52.1% in females aged 40 years or older [23]. However, a substantial number of people affected by OSA remain unrecognized [24]. Un diagnosed OSA has a great economic influence on healthcare systems as mentioned by public health records [25,26].

Several studies have assessed the relation between SB and OSAs and found that patients with sleep-disordered breathing (SDB) have an increased number of risk factors for SB [27-29]. A recent work concluded that tooth wear may be a diagnostic tool to identify patients at risk of having OSA [30].

Other works have assured the relevance of respiratory medicine in dental practice [31,32]. A recent review paper in 2014 revealed that the interrelation between SB and SDB can neither be confirmed nor discredited because of the lack of scientific proof and further studies on this field [33,34]. Since then, research on this topic has really improved in the last few years, so we tried to combine the old with recent review in this topic, and investigated the association between OSA and SB.

2.3 Previous Reviews

In 1986, Philipis et al. revealed the association between sleep apnea disorders and tooth clenching episodes [35]. Recent studies showed that 30% of patients with OSA suffer from tooth clenching as a secondary problem, 35% of people who complain of SB also experience snoring, and 17.6% suffer from OSA [36]. A pilot study in 2016 [37] revealed that SB is common in the majority of patient suffering from OSA with a prevalence rate of 77.8 %, which is higher than the 47.6% reported by Sjeholm et al. [38] and 47.8% by Hosoya et al. [39].

A recent study in 2019 showed that tooth wear is associated with several conditions, such as sleep disorders, orofacial pain, xerostomia, and SB, all of which are interlinked with one another. This finding assures the association among OSA, dental bruxism, and SB [40].

Hypertension, high body mass index BMI, and low mean oxygen saturation SpO2 are independent risk factors that lead to frequent SB episodes. Low values of SpO2 are commonly associated with OBA cases [41].

SB is also associated with multiple factors including insomnia, periodic limb movements while sleep, sleep-disordered breathing problems (apnea/hypopnea, A/H), gastroesophageal reflux disease, and some neurological disorders (e.g., sleep epilepsy and rapid eye movement behavior disorder) [42].

Another study reported SB occurrence in 21.0% of Japanese children and its independent relationships with age, snoring, and movements during sleep. In addition, Sb was found to be related to sleep-disorders in children [43].

Tan revealed that one-third of adults with OSA have SB and show relatively high respiratory-related arousals and oxygen desaturations [44].

Four important risk factors cause SB, such as emotional stress, consumption of tobacco, alcohol, or coffee, anxiety and depression disorders, and sleep apnea disorder [45].

2.4 Recent Concluding Remarks

This part involves recent publications about OSA. The studies and reviews summarize the updated diagnostic instruments and treatment modalities for OSA in adults, young individuals, and children.

Michalek–Zrabkowska et al. defined simple snoring as the production of sound in the upper aerodigestive tract during sleep that is not accompanied by other pathologies. In addition, SB involves repetitive phasic, tonic, or mixed masticatory muscle actions throughout sleeping. Their study examined the association between simple snoring and SB in 129 patients without OSA by using polysomnography. The results showed that bruxism episode index was positively correlated with maximum snore strength, and phasic bruxism was positively correlated with snore intensity in all sleep locations. Bruxers had significantly decreased average and minimum heart rate compared with non-bruxers. Supine and non-supine sleep positions seemed to have a significant effect on snore intensity and SB. In addition, a negative correlation was found between phasic bruxism and lowest heart rate, and this finding can be attributed to the effect of sympatho-vagal homeostasis [46].

In 2021, Smardz et al. measured and evaluated the effect of gender and age on snoring and SB in non-apneic snoring patients by using a singlenight video polysomnography. A total of 137 snoring non-apneic participants were included. Gender group results presented significantly higher total snore index and snore train were in males than in females. In addition, males showed severe bruxism with many recurrent episodes and high bruxism episode index scores. Association investigation revealed the existence of significant linear relationships between age and snore index in the supine sleep position and between snore train and snore index in non-rapid eye movement at the second sleep stage. Thirdage quartile analysis showed that the average, maximum, and minimum audio volumes in the non-supine sleep position were significantly high for the older group. Group analysis with median age as criterion showed that the bruxism episode index and bruxism phasic episodes were significantly higher in the younger group than in the older group. Therefore, age and gender influence snoring and SB [47].

Farahvash and Micieli stated that OSA is an indication of obstructed airways throughout sleep that substantially influence the condition of life and raise the risk of several systemic diseases. OSA has been investigated as a risk component for several neuro-ophthalmic conditions and has a strong relationship with non-arthritic anterior ischemic optic neuropathy (NAION). The results showed that the incidences of glaucoma and stroke were significantly associated with OSA and thus should be considered by neuroophthalmologists. Patients with NAION have a significantly higher incidence of OSA, and OSA diagnosis significantly increases the risk for NAION development. Non-agreement with incessant positive airway pressure in patients with OSA was also found to be a risk factor for fellow-eve involvement. However, evidence is insufficient recommend routine to eve OSA examinations in patients with for papilledema and conduct a sleep study for a patient newly diagnosed with idiopathic intracranial hypertension [48].

Smardz et al. investigated In 202. the coexistence of sleep-related breathing disorders (SRBDs) and SB among patients from the Clinic of Prosthetic Dentistry operating at Wroclaw Medical University. One niaht video polysomnography (PSG) was employed, and SB was evaluated using bruxism episode index (BEI) and the types of electromyographical pathways (phasic, tonic, and mixed). Apnea/hypopnea index (AHI) and oxygen desaturation index (ODI) were applied to assess respiratory events. Patients were divided into two groups according to SB occurrence: studied (bruxers, $BEI \ge 2$) and control (non-bruxers, BEI < 2) aroups. Quantitative outcomes presented the lack of a statistically significant relationship between BEI and AHI in both groups. Moreover, gualitative analysis revealed a statistically significantly correlation between the increase in AHI and ODI and the increase in the quantity of tonic categories for electro-myo- graphical pathways. On the basis of this positive correlation, the incidence of tonic episodes may be the key to understanding the fundamental connection between SB and SRBD and thus require further precise examinations [49].

In 2020, da Costa Lopes et al., 2020 published a systematic review to clarify recent knowledge on the possible association and connection between SB and OSA examined using full-night polysomnography. Among the collected 270 articles,7 was included. Furthermore, only four of these studies supported the association between SB and OSA. Their main findings were as follows: (a) a subtype of patients with OSA may have SB as a defensive response to respiratory events, (b) maximum episodes of bruxism occur shortly after the end of apnea/hypopnea (AH) events, (c) bruxism incidents arise secondary to arousals from AH events, and (d) a relationship occurs between the frequency of SB and AH events. The remaining three studies did not support the association, and their main findings are as follows: AH episodes are related to nonspecific SB oromotor actions, SB episodes are not directly associated with the end of AH events, and patients with OSA do not experience more SB events than control group [50].

Patient SDB includes, mouth breathing, snoring, upper airway resistance syndrome, and OSA. OSA is defined as five or more episodes of complete (apnea) or partial (hypopnea) upper airway obstruction per hour of sleep. This condition is predicted to involve 24% of middleaged men and 9% of middle-aged women. Approximately 2%-3% of children have OSA, and this value increases to 30%-40% in obese children [51] Bhattacharee et al. reported that the affected patients suffer from daytime drowsiness, cognitive impairment, and increased risk of heart attacks, strokes, uncontrolled hypertension, and diabetes. Untreated OSA can meaningfully harm a patient's quality of life and increase morbidity due to medical complications or transport- or work-related accidents [52].

Prosthodontists are important in preserving patients' general health by reestablishing and maintaining physiological oral function and aesthetic appearance. SB and orofacial pain are situations that require the dentist to have diagnostic and management skills. With their additional training and expertise in oral anatomy, occlusion, and temporomandibular joint function, prosthodontists should recognize the signs and symptoms of OSA, refer to the sleep physician for diagnosis, and collaborate with the health team surrounding the patient to provide care that will improve the patient's oral and general health [52].

OSA in children is regularly due to inflamed tonsils and adenoids peaking at 5–6 years of age. Craniofacial morphological features often exist in children with airway problems, such as narrow maxilla, anterior open-bite, mouth breathing, and dolichocephalic profile [53-54].

In children, SA is diagnosed when a minimum of one apnea event occurs for each hour of sleep as observed during a diagnostic sleep study. For adults, more than five events per hour indicate SA. Sleep apnea in a child arises when the muscles of the upper airway sufficiently relax to temporarily reduce or obstruct airflow. This event can happen repeatedly over the course of a night and may cause in disordered sleep because the brain tries to awaken the body and return to normal breathing. Both events can be associated with a drop in blood oxygen levels. Surprising signs of sleep apnea in children include mouth breathing, bedwetting, sleepwalking, restlessness, and sweating during sleep. Several potential consequences for a child's mental and physical health are also associated with SA. The incidence of SA in preschool-aged children is approximately 1% to 3%, with the highest cases reported between ages 2 and 6 years. During normal growth, a child's tonsils and adenoids tend to be proportionately larger than their airway, and these enlargements render the airway to be easily obstructed. The risk of sleep apnea is also high in severely overweight or obese adolescents. Children of any age who have asthma or allergies also are highly prone to developing SA. The long-term effects of SA in children include cognitive, behavioral, and psychosocial difficulties, growing interruptions, and impacts on cardiovascular condition [55].

Martinot et al. conducted an observational study identify stereotypical mandibular to jaw movements (MJMs) in patients with SB and automatically detect RMMAs by using an artificial intelligence (AI)-based approach. Simultaneous MJM recordings from polysomnography with masseter electromvography were obtained for 67 patients with suspected OSA. The recording system consisted of a small hardware device attached on the chin that communicates to a cloud-based infrastructure. An extreme gradient boosting multiclass classifier was trained on 79,650 10-second epochs of MJM data from 39 patients with a history of SBx targeting three episodes (n=1072), microlabels: RMMA arousals (n=1311), and MJM occurring at the breathing frequency (n=77,267). The results were validated on unseen data from 28 patients, and the model showed a good epoch-by-epoch agreement in the Kappa test. A balanced accuracy of 86.6% was also found for the MJM events as per the RMMA standards. Good agreement was observed between the MJM analytic model and manual EMG signal scoring of RMMA. Therefore, SB can be reliably identified, guantified, and characterized using MJM for automated analysis supported by AI technology [56].

3. CONCLUSION

Sleep apnea is considered an important risk factor that can lead to SB. Dental and medical practitioners should be informed about the close relationship between SB and OSA to help discover and treat such serious medical problems. However, no scientific evidence has supported the conclusive relationship between SB and OSA. A well-designed, randomized research with control groups is needed to explore possible mechanisms common to SB and OSA and whether OSA treatment could recover SB

negative oral health results in subjects with SB and in combination of OSA.

CLINICAL SIGNIFICANCE

This work is important, especially for dental practitioners, to assess every patient with SB and detect any underdiagnosed sleep disorders by carefully reviewing medical and dental history and referring the patient to sleep medicine clinics in case of suspected presence of any sleep disorder.

CONSENT

It is not applicable.

ETHICAL APPROVAL

It is not applicable.

COMPETING INTERESTS

Authors have declared that no competing interests exist.

REFERENCES

- 1. Jokubauskas L and Baltrusaytate A: Relation between obstructive sleep apnea syndrome and SB: a systematic review. J of Oral Rehabilitation. 2017;44:144-153.
- American Academy of Sleep Medicine. International Classification of Sleep Disorders, 3rd ed; American Academy of Sleep Medicine: Darien, IL, USA. 2014; 232.
- Kardachi BJ, Bailey JO, Ash MM: A comparison of biofeedback and occlusal adjustment on bruxism. J Periodontol. 1978;49:367–372.
- Huynh NT, Rompré PH, Montplaisir JY, Manzini C, et al. Comparison of various treatments for SB using determinants of number needed to treat and effect size. IntJ Prosthodont. 2006;19:435–441.
- Manfredini D, Visscher CM, Guarda-Nardini L. Occlusal factors are not related to self-reported bruxism. J Orofac Pain. 2012;26:163–167.
- Lobbezoo F, Rompré PH, Soucy JP, lafrancesco C, et al. Lack of associations between occlusal and cephalometric measures, side imbalance in striatal D2 receptor binding, and sleep-related

promotor activities. J Orofac Pain. 2001:15:64–71.

- Ommerborn MA, Giraki M, Schneider C, Fuck LM, et al. Effects of SB on functional and occlusal parameters: A prospective controlled investigation. Int J Oral Sci. 2012;4:141–145.
- 8. Klasser GD, Rei N, Lavigne GJ. SB etiology: the evolution of a changing paradigm. J. Can. Dent. Assoc. 2015;81:f2.
- Lavigne GJ, Khoury S, Abe S, Yamaguchi T, Raphael K. Bruxism physiology and pathology: An overview for clinicians. J. Oral Rehabil. 2008;5:476–494.
- 10. Hosoya H, Kitaura H, Hashimoto T, Kinbara M, et al. Relationship between SB and sleep respiratory events in patients with obstructive sleep apnea syndrome. Sleep Breath. 2014;18:837–844.
- Saito M, Yamaguchi T, Mikami S, Watanabe K, et al. Weak association between SB and obstructive sleep apnea. A sleep laboratory study. Sleep Breath. 2016;20:703–709.
- 12. Carra M, Huynh N, Morton P, Rompr'e P, et al. Prevalence and risk factors of SB and wake-time tooth clenching in a 7- to 17-yr-old population. Eur J Oral Sci.0 2011;119:386–394.
- Feu D, Catharino F, Quint~ao C, Almeida M. A systematic review of etiological and risk factors associated with brux- ism. J Orthod. 2013;40:163–171.
- 14. Lavigne G, Huynh N, Kato T, Okura K, et al. Genesis of SB: Motor and autonomiccardiac interactions. Arch Oral Biol. 2007; 52:381–384.
- Lavigne G, Khoury S, Abe S, Yamaguchi T, Raphael K. Bruxism physiology and pathology: an overview for clinicians. J Oral Rehabil. 2008;35:476–494.
- 16. Klasser GD, Rei N, Lavigne GJ. SB etiology: the evolution of a changing paradigm. J Can Dent Assoc. 2015;81:f2.
- 17. Manfredini D. Guarda-Nardini L, Marchese-Ragona R, Lobbezoo F. Theories on possible temporal relationships between SB and obstructive sleep apnea events. An expert opinion. Sleep Breath. 2015;19:1459–1465.
- 18. 18-Raphael K, Santiago V, Lobbezoo F. Is bruxism a disorder or a behavior? Rethinking the international consensus on defining and grading of bruxism. J Oral Rehabil. 2016;43:791–798.

- 19-American Academy of Sleep Medicine. International classification of sleep disorders revised: Diagnostic and coding manual. Chicago (IL): American Academy of Sleep Medicine; 2001.
- 20. Park J, Ramar K, Olson E. Updates on definition, consequences, and management of obstructive sleep apnea. Mayo Clin Proc. 2011;86:549–555.
- 21. Zimmerman M, Aloia M. Sleep-disordered breathing, and cognition in older adults. Curr Neurol Neurosci Rep. 2012;12:537– 546.
- 22. Golbin J, Somers V, Caples S. Obstructive sleep apnea, cardiovascular disease, and pulmonary hypertension. Proc Am Thorac Soc. 2008;5:200–206.
- 23. Tahrani A, Ali A, Stevens M. Obstructive sleep apnoea and diabetes. CurrOpinPulm Med. 2013;19:631–638.
- 24. Heinzer R, Marti-Soler H, Haba-Rubio J. Prevalence of sleep apnea syndrome in the middle to old age general population. Lancet Respir Med. 2016;4:e5–e6.
- 25. Punjabi N. The epidemiology of adult obstructive sleep apnea. Proc Am Thorac Soc. 2008;5:136–143.
- 26. Tarasiuk A, Reuveni H. The economic impact of obstructive sleep apnea. Curr Opin Pulm Med. 2013;19:639–644.
- 27. Leger D, Bayon V, Laaban J, Philip P. Impact of sleep apnea on economics. Sleep Med Rev. 2012;16:455–462.
- 28. Ohayon MM, Kasey LK, Guilleminault C. Risk factors for SB in the general population. Chest. 2001;119:53–61.
- 29. Sjoholm TT, Lowe AA, Miyamoto K, Fleetham JA. SB in patients with sleepdisordered breathing. Arch Oral Biol. 2000;45:889–896.
- Lam MHB, Zhang J, Li AM, Wing YK. A community study of SB in Hong Kong children: association with comorbid sleep disorders and neurobehavioral consequences. Sleep Med. 2011;12:641–645.
- Dur'an-Cantolla J, Alkhraisat M, Mart'inez-Null C, Aguirre J. Frequency of obstructive sleep apnea syndrome in dental patients with tooth wear. J Clin Sleep Med. 2015; 11:445–450.
- Kostrzewa-Janicka J, Jurkowski P, Zycinska K. Sleep-related breathing disorders and bruxism. Adv Exp Med Biol. 2015;873:9–14.

- Mayer P, Heinzer R, Lavigne G. SB in respiratory medicine practice. Chest. 2016; 149:262–271.
- De Luca Canto G, Singh V, Gozal D. SB and sleep-disordered breathing: a systematic review. J Oral Facial Pain Headache. 2014;28:299–305.
- Phillips BA, Okeson J, Paesani D. Effect of sleep position on sleep apnea and parafunctional activity. Chest. 1986;90: 424-9.
- 36. 36-Lavigne GJ, Khoury S, Abe SK. Bruxism physiology and pathology: an overview for clinicians. J Oral Rehabil. 2008;35:476-94.
- Winck M, Drummond M, Viana P, et al. SB associated with obstructive sleep apnoeasyndrome - A pilot study using a new portable device. Rev Port Pneumol. 2017;23:22-6.
- Sjöholm TT, Lowe AA, Miyamoto K, Fleetham JA, Ryan CF. SB in patients with sleep-disordered breathing. Arch Oral Biol. 2000;45:889-96.
- 39. Hosoya H, Kitaura H, Hashimoto T, Ito M. Relationship between sleep bruxism and sleep respiratory events in patients with obstructive sleep apnea syndrome. Sleep Breath. 2014;18:837-44.
- 40. Wetselaar P, Manfredini D, Ahlberg J, Johansson N. Associations between tooth wear and dental sleep disorders: A narrative overview. J Oral Rehabil. 2019; 46:765–775.
- Martynowicz H, Marzena PD, Kazubowska K, Skomro R, et al. Evaluation of Intensity of SB in Arterial Hypertension. J. Clin. Med. 2018;7:327.
- 42. Mayer P, Heinzer R, Lavigne G. SB in Respiratory Medicine Practice. Chest. 2016;149(1):262-71.
- 43. Tachibana M, Kato T, Kato-Nishimura K, Matsuzawa S. Associations of SB with age, sleep apnea, and daytime problematic behaviors in children. Oral Dis. 2016;22 (6):557-65.
- 44. Tan MWY, Yap AU, Chua AP, Wong JCM.Prevalence of SB and Its Association with Obstructive Sleep Apnea in Adult Patients: A Retrospective Polysomnographic Investigation. J Oral Facial Pain Headache. Summer. 2009;33(3):269–277.
- 45. Kuhn M, Türp JC. Risk factors for bruxism. Swiss Dent J. 2018;128(2):118-124.
- 46. Michalek-Zrabkowska M, Wieckiewicz M, Macek, P, Gac P, et al. The Relationship

between Simple Snoring and SB: A Polysomnographic Study. Int J Environ Res Public Health. 2020;17(23):8960. DOI: 10.3390/ijerph17238960

- Smardz J, Wieckiewicz M, Gac P, Poreba R, et al. Influence of age and gender on SB and snoring in non-apneic snoring patients: A polysomnographic study. J Sleep Res. 2021;30(3):e13178. DOI: 10.1111/jsr.13178
- 48. Farahvash Α. Micieli JA. Neuro-Ophthalmological Manifestations of Obstructive Sleep Apnea: Current Perspectives. Eye Brain 2020;12:61-71. DOI: 10.2147/EB.S247121. eCollection 2020.
- Smardz J, Martynowicz H, Wojakowska A, Michalek-Zrabkowska M, et al. The meaning of the masticatory muscle tonictype electromyographic pathway correlated with SB and sleep-related breathing disorders - A polysomnographic study. Sleep Med. 2020;68:131-137. DOI: 10.1016/j.sleep.2019.08.025. Epub 2019 Oct 2.
- 50. da costa lopes AJ, Cunha TC, Monteiro MC, Serra-Negra JM., et al. Is there an association between SB and obstructive sleep apnea syndrome? A systematic review. Sleep Breath. 2020; 24(3):913-921.

DOI: 10.1007/s11325-019-01919-y.

- Bhattacharee R, Kim J, Kheirandish-Gozal L: Obesity and obstructive sleep apnea syndrome in children: a tale of inflammatory cascades. Pediatr Pulmonol. 2001;46:313-323.
- 52. Wu JC, Dubois NM, Linkous JG. The Sleep-disordered Breathing. American College of Prosthodontists. Position Statements. 2020;1-4.
- 53. Gozal D, Pope DW Jr: Snoring during early childhood and academic performance at ages thirteen to fourteen years. Pediatrics. 2001;107:1394-1399.
- 54. Flores-Mir C, Korayem M, Heo G, et al: Craniofacial morphological characteristics in children with obstructive sleep apnea syndrome: a systemic review and metaanalysis. J Am Dent Assoc. 2013;144:269-277.
- Peters P. Symptoms and Consequences of Sleep Apnea in Children. Updated on April 17, 2020 and Medically reviewed by Jassey JB.

56. Martinot J-P, Le-Dong N-N, Cuthbert V, Denison S, et al. Artificial Intelligence Analysis of Mandibular Movements Enables Accurate Detection of Phasic SB in OSA Patients: A Pilot Study. Nat Sci Sleep. 2021;13:1449-1459. DOI: 10.2147/NSS.S320664. e Collection 2021.

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