Episode 158 – Sleep – Part Three: Obstructive Sleep Apnea and Oral Health

June 12, 2026

Overview

Sleep-related breathing disorders include snoring, upper airway resistance syndrome, hypopnea, and sleep apnea. Sleep apnea refers to pauses in breathing that occur during sleep. These breathing pauses, called apneas or apnea events, can last for 10 seconds or longer. Individuals with sleep apnea can stop breathing hundreds of times each night, resulting in disrupted sleep and low levels of oxygen.[1] [1] [2]

Sleep apnea can range from mild to severe, based on the average number of apneas and hypopneas per hour of sleep, which is referred to as the apnea-hypopnea index (AHI). Hypopneas are partial obstructions of the upper airway resulting in a 20%-50% decrease in airflow and 3%-4% oxygen desaturation. AHI is expressed as the number of events or episodes per hour. The more events that occur, the more severe the sleep apnea and the greater the likelihood of adverse effects. [3] [4]

AHI in adults is graded as:

- Mild: 5 to 14 events per hour
- Moderate:15 to 29 events per hour
- Severe: 30 or more events per hour [5] [6]

AHI thresholds for children differ compared to adults, as they are still developing and breathe at a faster rate than adults. A normal score for children is less than one event per hour. [6] [7]

The pediatric AHI range is:

- Mild: 1 to 4 events per hour
- Moderate: 5 to 9 events per hour
- Severe: 10 or more events per hour [7] [8]

The pediatric range is typically used for children 2 months post-term to 18 years. In some cases, the adult scale is used for teenagers. [7] [8]

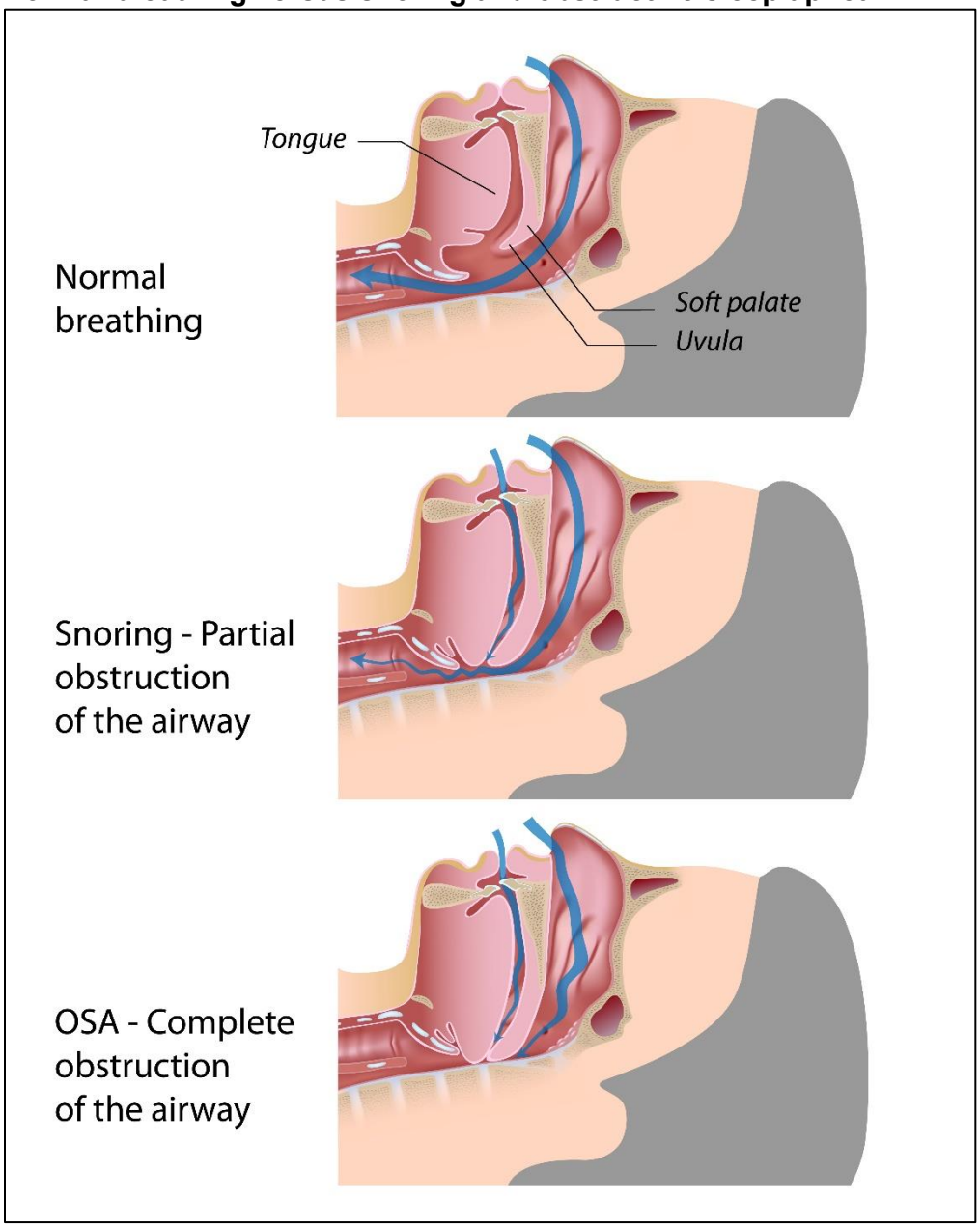
Main types of sleep apnea include central sleep apnea and obstructive sleep apnea. Central sleep apnea (CSA) occurs when the brain fails to transmit signals to the muscles responsible for respiration. CSA is a less common form of sleep apnea. [1]

[1] Listen to Episodes 156 and 157 for discussion on sleep disorders and sleep-disordered breathing.

Obstructive sleep apnea

Obstructive sleep apnea (OSA) is the most common type of sleep apnea and occurs when the upper airway is partially or completely obstructed during sleep, inhibiting airflow. The obstruction often occurs when the soft tissue in the back of the throat relaxes and collapses during sleep. Relaxed throat muscles, a narrow airway, a large tongue, or extra adipose tissue in the throat can also block the airway. OSA interrupts normal sleep patterns and nighttime breathing and includes episodes of intermittent hypoxia (lack of oxygen) and frequent sleep arousal. [1] [9] [10]

Normal breathing versus snoring and obstructive sleep apnea



Sleep apnea can affect anyone at any age. For example:

- 1 in 5 adults (20%) has at least a mild form of sleep apnea.
- 1 in 15 adults (6.6%) has at least moderate sleep apnea.
- Over 1 in 4 adults (26%) have a high risk of having or developing OSA.
- 1%-5% of children are estimated to have OSA. OSA can occur at any age in childhood. However, it is most common in children ages 2 to 7. In prepubertal children, OSA occurs equally among males and females. In adolescents, the prevalence tends to be higher in males. [11] [12]

Untreated OSA can lead to daytime sleepiness and trouble thinking clearly, and increases the risk of vehicle and workplace accidents, depression, diabetes,[2] cardiovascular and cerebrovascular diseases,[3] and early death. [1] [9] [13] [14] [15]

Untreated OSA places strain on the heart and vascular system. Episodes of apnea can trigger intermittent hypoxia. The drop in oxygen levels activates the sympathetic nervous system, increasing heart rate and blood pressure. Repeated episodes over time can lead to cardiac remodelling and dysfunction (e.g., left ventricular hypertrophy and diastolic dysfunction). Over time, these structural and functional changes can progress to more severe forms of cardiac dysfunction (e.g., heart failure). [10] [15]

Chronic intermittent hypoxia and recurrent surges in blood pressure can lead to endothelial dysfunction and the development of atherosclerotic plaques in the cerebral vasculature, altering cerebral blood flow. These changes can increase the risk of stroke and other cerebrovascular events and contribute to cognitive impairment and vascular dementia.[4] [10] [15]

Children with OSA may also experience poor weight gain, growth delays, and failure to thrive. Cognitive, learning, and behaviour issues are associated with untreated OSA. The lack of oxygen resulting from apnea can significantly affect neurocognitive development, attention, memory, and academic performance. Attention-deficit hyperactivity disorder (ADHD) is associated with OSA in children. These complications may inhibit a child's ability to learn and function well at school, leading to behavioural difficulties, irritability, and social issues. [10] [16] [17]

OSA in children may also affect orofacial development, leading to differences in dental arch dimensions compared with children without OSA. These include a narrower maxilla, deeper palatal height, a receding mandible (deficient chin), crossbite, overjet, and tooth malposition. These alterations may result from chronic postural adaptations of the head, jaw, and tongue aimed at maintaining an open airway during sleep. These irregularities can affect breathing patterns, occlusion, and facial structure. [10]

[2] Listen to Episodes91, 93, and 94 for discussion on diabetes.
[3] Refer to Episodes 37, 79, 80, and 81 for information on cardiovascular and cerebrovascular diseases.
[4] Listen to Episode 29 for discussion on dementia and oral health.

Symptoms
Signs and symptoms of OSA in adults may include:
- Excessive daytime sleepiness
- Loud snoring with pauses in breathing
- Gasping or choking during sleep
- Morning headache
- Waking with a dry mouth
- Irritability or mood changes
- Poor concentration or memory loss
- Hypertension
- Diabetes
- Heart, liver, and kidney disease
- Depression
- Lowered sex drive; impotence
- Gastroesophageal reflux disease (GERD)
- Dental erosion attributed to GERD
- Sleep bruxism [1] [5] [9] [18] [19] [20]

Gastroesophageal reflux disease (GERD) is often associated with OSA. Several mechanisms may explain this relationship. Intermittent hypoxia and negative pressure within the chest during apnea episodes may worsen reflux by impairing lower esophageal sphincter function, increasing intra-abdominal pressure, and promoting backflow of gastric acid. OSA and GERD also share common risk factors, including obesity,[5] male sex, alcohol use, and smoking. [19] [21] [22]

Sleep bruxism may be associated with OSA as clenching teeth during sleep may expand the airway and possibly reduce obstruction. Signs of bruxism include worn tooth enamel, fractured teeth, tooth pain and sensitivity, and masseter muscle soreness. Treatment of OSA may reduce bruxism during sleep. [1] [5]

Signs and symptoms of OSA in children may include:
- Snoring reported by parents. Snoring may not be reported even when OSA is severe. Infants and young children with OSA do not always snore.
- Observed apnea.
- Snorting, gasping, coughing, or choking during sleep.
- Restless sleep.
- Unusual sleep positions (e.g., sitting up or with the neck hyperextended to open the airway).
- Frequent awakenings.
- Nighttime sweating.
- Bedwetting (nocturnal enuresis) that starts after a long period of dry overnights.
- Morning headache.
- Attention problems.
- Doing poorly at school.

[5] Refer to Episodes 120, 121, and 122 for information on obesity and oral health.

- Developmental delay.
- Hyperactivity as a manifestation of sleepiness.
- Acting impulsive or aggressive.
- Restlessness.
- Saying they feel sleepy or falling asleep at school or during short car or bus rides.
- Poor weight gain.
- Dark undereye circles and an overall look of fatigue.
- Nasal obstruction.
- Mouth breathing.
- Bruxism and tooth wear (e.g., attrition, erosion). [10] [12] [23] [24] [25] [26] [27]

Risk factors in adults

General risk factors for OSA in adults may include:

- Obesity or excess weight. OSA is 7 times more common among individuals with obesity (BMI of ≥30). However, not all adults with OSA have obesity.
- Male sex.
- Post-menopausal status.
- Older age.
- Pregnancy, especially during the third trimester when weight gain is greatest. Developing OSA during pregnancy can result in adverse maternal-fetal outcomes, including preeclampsia, intrauterine growth, and low birth weight.[6]
- Family history of OSA.
- Tobacco use (nicotine relaxes the muscles that keep the airways open).[7]
- Alcohol consumption.
- Sedative use. [1] [2] [3] [4] [28] [29] [30] [31]

Extraoral and intraoral risk factors associated with OSA in adults may include:

- Large neck circumference: >43 cm (>17 in) in males; >41cm (>16 in) in females. Excess adipose tissue in the neck area increases the risk of upper airway soft tissue obstruction.
- Chronic nasal congestion.
- Decreased nasal passage size.
- Small and/or recessed mandible (microretrognathia).
- Steep mandibular plane.
- Enlarged tonsils or adenoids.
- Increased anterior face height (i.e., long looking face).
- Class II malocclusion.
- Large tongue (macroglossia).
- Thick, broad, and scalloped tongue.
- Reduced intermaxillary space.
- Narrow high arched palate, which can reduce nasal space and increase nasal resistance. A narrow high palate may not accommodate the tongue, resulting in a low, flat tongue posture and crowding of the upper airway.

[6] Refer to Episodes 108, 109, and 110 for information on pregnancy and oral health.
[7] Listen to Episode 101 and 131 for discussion on tobacco use and nicotine pouches, respectively.

- Elongated soft palate and uvula.
- Edentulism. [1] [3] [4] [28] [29] [30] [32] [33]

Edentulism can alter upper airway anatomy during sleep by affecting the resting position of the mandible, tongue, and surrounding muscles. The loss of occlusal vertical dimension after tooth loss reduces lower facial height and causes mandibular rotation, which may decrease airway space. Along with progressive alveolar bone resorption, these changes can lead to mandibular overclosure and an unnatural tongue position, increasing the risk of airway obstruction. [4] [33] [34]

Pediatric risk factors

Pediatric risk factors for OSA may include:

- Enlarged tonsils and/or adenoids, which can narrow the upper airway, especially during sleep and obstruct airflow.
- Nasal obstructions (e.g., deviated septum, allergic rhinitis).
- Decreased nasal passage size.
- Exposure to allergens and second-hand smoke, which can contribute to nasal inflammation and congestion.
- Obesity.
- Small mandible (micrognathia).
- Recessed mandible (retrognathia)
- High, narrow, vaulted palate and maxillary constriction.
- Low lying tongue position.
- Increased soft palate volume.
- Malocclusion, posterior crossbite, and anterior open bite.
- Increased anterior facial height.
- Disorders (e.g., cerebral palsy, myotonic dystrophy, Down syndrome).
- Family history of sleep apnea. [4] [10] [12]

Nasal obstruction and enlarged adenoids and tonsils can result in mouth breathing and dry mouth. Dry mouth increases the risk of dental caries and periodontal diseases. The typical facial structure associated with mouth breathing, referred to as “adenoid face” or “long face” syndrome, is characterized by an inability to maintain a lip seal, a narrow maxillary arch, increased anterior face height, a steep mandibular plane angle, and a retrognathic mandible.

There are similarities between the orofacial characteristics of chronic mouth breathing and orofacial risk factors for individuals with OSA. Common orofacial features of chronic mouth breathing include an open lip posture, Class II malocclusion, crossbite, increased anterior facial height, enlarged soft palate, a narrow, high arched palate, a low, flat tongue, and a steep mandibular occlusal plane. These orofacial features suggest a posterior inclination of the mandible and pharyngeal tissues, which may be associated with an increased risk of OSA.

While chronic mouth breathing is not a direct cause of OSA, the orofacial architecture associated with mouth breathing may indicate an underlying risk of OSA. [4] [35]

Additional impact on oral health

Oral health problems are more common among individuals with OSA, with increasing evidence demonstrating an association between OSA and alterations in the oral microbiome, taste dysfunction, periodontitis, dental caries, tooth wear, orofacial pain, temporomandibular disorders, and mandibular tori.

Oral microbiome

Sleep disorders may impact the composition and structure of the oral microbiome,[8] potentially influencing oral health. Several studies have shown OSA can alter the oral bacterial flora in adult and pediatric populations. Research found significant alterations in the composition of the oral microbiome in children with OSA compared with children without OSA. These alterations were particularly evident in the relative abundances of Firmicutes, Proteobacteria, Bacteroidetes, Fusobacteria, and Actinobacteria. These studies suggest alteration of oxygen concentrations due to OSA may reshape the oral bacterial community. Moreover, the decreased airflow during sleep apnea can lead to a dry mouth, creating an environment that favours the growth of certain bacteria. [36] [37] [38] [39] [40]

Taste dysfunction

Taste dysfunction can result from various factors, such as infections, adverse effects of medications, nerve damage, nutritional deficiencies, or underlying health conditions. [36]

Huang et al. (2023) investigated whether OSA was associated with the risk of taste dysfunction. They conducted a nationwide cohort study involving 15,730 participants. The results revealed those with OSA had a significantly increased risk of incident taste dysfunction over a 15-year follow-up period, particularly in females with OSA. This study suggests a positive association between OSA and taste dysfunction.

Taste dysfunction associated with sleep disorders may be related to alterations in oral flora. Dry mouth associated with OSA may also impair taste. Future research should further investigate this relationship to help determine causation. [36] [41]

Periodontitis

Several studies have reported an association between OSA and an increased risk of periodontitis. Potential explanations for this relationship include oxidative stress, systemic inflammation, mouth breathing, and shared risk factors, which may increase susceptibility to both conditions. During sleep, apnea and hypopnea episodes in OSA promote oxidative stress, and the resulting reactive oxygen species contribute to inflammatory pathways. Individuals with OSA tend to exhibit elevated levels of interleukin-6 (IL-6), high-sensitivity C-reactive protein (hs-CRP), and interleukin-1β in gingival crevicular fluid. Both IL-6 and IL-1β are cytokines involved in periodontal tissue destruction.[9]

[8] Refer to Episode 32 for research on the oral microbiome.
[9] Refer to Episodes 5, 6, 37, and 47 for discussion on periodontal disease.

OSA may contribute to both physical and psychological stress due to disrupted and nonrestorative sleep, which may further increase the risk of periodontitis. Individuals with OSA often mouth breathe during sleep to compensate for partial or complete upper airway obstruction. Mouth breathing can lead to dry mouth. The reduced salivary cleansing increases plaque accumulation and subsequent inflammation, thereby increasing the risk of periodontal disease. OSA and periodontitis also share several risk factors, including obesity, diabetes, alcohol consumption, and tobacco use, which may contribute to the onset and progression of both disorders. [42] [43] [44] [45]

Several studies suggest OSA can affect the oral bacterial flora. A systematic review by Bianchi et al. (2023) reported OSA and related conditions, such as mouth breathing, are associated with alterations in oral microbiota composition, which may help explain the observed association between OSA and periodontitis. Similarly, Chen et al. (2021) found changes in the salivary microbial community structure among individuals with OSA, including a significant increase in the periodontal pathogen *Prevotella*. [36] [38] [39] [46] [47]

Téllez-Corral et al. (2022) found a higher prevalence of stage III periodontitis in participants with severe OSA. Periodontal pathogens, such as *Prevotella melaninogenica* and the yeast *Candida albicans*, altered the oral microbiota of participants with periodontitis and OSA in terms of diversity, possibly increasing the severity of periodontal disease. The association between yeasts and periopathogenic bacteria could help explain why people with severe OSA have a higher risk of stage III periodontitis. [48]

Li et al. (2025) reported OSA was associated with distinct shifts in salivary microbiota, including reduced microbial richness and altered functional profiles, which may contribute to early periodontal dysbiosis. [49]

Dental caries

OSA may increase the risk of dental caries in adult and pediatric populations. People with OSA commonly report mouth breathing and dry mouth. Dry mouth can lead to several oral complications, including disturbances in salivary function, reduced salivary clearance, and increased plaque formation and bacterial colonization. These factors may predispose individuals with OSA to a higher prevalence of dental caries.[10] [35] [50]

Tooth wear

Tooth wear is the irreversible loss of tooth structure and is cumulative, resulting in greater wear among older individuals.[11] Tooth wear is associated with hypersensitivity, pain, and aesthetic concerns. Tooth substance loss may occur through chemical processes (erosion), mechanical processes (attrition or abrasion), or a combination of both. Several factors can influence the pattern of tooth wear, including enamel thickness and hardness, oral pH, age, malocclusion, occlusal trauma, and bruxism. Bruxism can

[10] Listen to Episodes 86 and 87 for additional information on dental caries.
[11] Refer Episode 82 for discussion on tooth wear.

contribute to tooth surface loss (attrition), particularly when combined with acid erosion. Sleep bruxism is associated with OSA and GERD. [51]

Tooth wear is a common finding in individuals with sleep disorders. Durán-Cantolla et al. (2015) evaluated the frequency of OSA among participants with tooth wear who were being treated with an occlusal splint. They found participants with tooth wear had a high frequency of OSA and a statistically significant association between OSA severity and tooth wear severity. The authors suggest tooth wear assessment can be a tool to identify clients with possible OSA.

All participants in this study had varying degrees of tooth wear treated with an occlusal splint. Clients with tooth wear often receive occlusal splints and/or oral rehabilitation. However, oral health clinicians may not refer such clients to a sleep clinic to rule out OSA. Consequently, these clients may not have the opportunity for early diagnosis and treatment of OSA, potentially increasing the risk of medical complications associated with OSA. It is also worth noting evidence exists indicating the use of an occlusal splint may worsen OSA. [10] [51] [52]

Anitua et al. (2017) also found the severity of tooth wear was significantly correlated with OSA severity and concluded tooth wear severity may indicate the presence of OSA. These findings underscore the important role of oral health professionals in identifying individuals potentially at risk for OSA. [53]

Orofacial pain

Chronic orofacial pain and sleep disorders are prevalent conditions that significantly impact quality of life. Increasing evidence suggests a bidirectional relationship between these disorders, whereby sleep disturbances may exacerbate orofacial pain and vice versa. Orofacial pain refers to pain localized to the face and oral cavity. This pain can originate from nearby structures, result from diseases or nervous system dysfunction, or be referred from distant sources. Orofacial pain includes various types of pain, such as dental, mucosal, myofascial, musculoskeletal, neurovascular, and neuropathic pain. [54] [55] [56]

A systematic review by Colonques-Sanmartín et al. (2025) indicated a strong association between chronic orofacial pain and sleep disorders, particularly OSA and insomnia. The authors concluded addressing sleep-related issues could be an effective complementary approach in managing orofacial pain. However, more research is necessary to clarify the underlying mechanisms, determine potential moderating factors, and develop interdisciplinary interventions for individuals with sleep disorders and orofacial pain. [54]

OSA and orofacial pain, including temporomandibular disorders (TMDs), are common conditions that often occur together but are typically treated separately. Although these disorders have distinct mechanisms, evidence suggests significant interaction, especially shared anatomical and neurophysiological pathways. This interconnection has prompted exploration of the bidirectional relationship between sleep apnea and orofacial pain, where one condition may worsen or contribute to the development of the

other. For instance, the mechanical forces associated with TMD can influence airway dynamics, while sleep disruptions caused by OSA can intensify orofacial pain symptoms. This relationship between sleep apnea and orofacial pain presents a complex clinical challenge that requires integrated management. [57]

A review by Patil et al. (2025) examined the association between sleep apnea and orofacial pain. The findings showed the co-occurrence of sleep apnea and orofacial pain, particularly TMD and bruxism, demonstrates how sleep-disordered breathing leads to compensatory neuromuscular responses. These responses manifest as increased activity in the masticatory muscles and sleep bruxism, which can trigger or worsen TMD dysfunction and associated orofacial pain syndromes. Furthermore, chronic orofacial pain can negatively impact sleep quality, potentially worsening sleep-disordered breathing by disrupting jaw mechanics and altering muscle tone. The neurological interaction between these conditions involves central nervous system modulation of pain signals, leading to greater pain sensitivity. [57]

Addressing sleep apnea and orofacial pain together is essential for improving client outcomes, as treatment of one condition can directly affect the other. Oral health professionals should understand this interrelationship and adopt a comprehensive, multidisciplinary approach to diagnosis and treatment. [57]

Temporomandibular disorders

Temporomandibular disorders (TMDs) are a group of disorders affecting the temporomandibular joint (TMJ), masticatory muscles, and associated structures. TMDs can manifest as persistent TMJ and muscular pain, joint sounds, restricted mouth opening, abnormal mandibular function, and functional limitations, which can adversely affect daily activities and overall well-being. TMDs may also be associated with headache[12] secondary to TMD. [58] [59] [60] [61]

The pathophysiology of TMDs is multifactorial. Key mechanisms include overloading of the masticatory system, which can lead to inflammation and pain in the joint capsule or the muscles involved in chewing. These processes are often exacerbated by central sensitization, a condition characterized by increased reactivity of the nervous system that intensifies pain signals and contributes to the chronicity of the pain. [62]

Studies evaluating participants with sleep-disordered breathing have reported a higher prevalence of TMD signs and symptoms among individuals with OSA, compared with those without the disorder. [61] [63] [64]

A systematic review by Al-Jewair et al. (2022) on the relationship between sleep disorders and TMDs found a consistent association between poor sleep quality and TMD, suggesting sleep disturbance may contribute to the development or worsening of temporomandibular dysfunction. [65]

[12] Refer to Episodes 148 and 149 for additional information on headaches.

Additional observational evidence has further supported these findings, demonstrating a significantly higher prevalence of TMD symptoms among individuals with untreated OSA. [61] [66] [67]

The relationship between OSA and TMD is likely multifactorial. Recurrent arousals and sleep fragmentation in OSA may disrupt pain processing, promote central sensitization, and increase musculoskeletal pain sensitivity. Intermittent hypoxia may further contribute by triggering systemic inflammatory pathways and increasing pro-inflammatory cytokines, which can adversely affect joint and muscle tissues. [61]

A bidirectional Mendelian randomization study by Wang et al. (2024) suggested a possible causal effect of OSA on TMD risk, whereas TMD did not independently increase the risk of OSA. These findings suggest effective treatment of OSA may help prevent and alleviate TMD symptoms. [58]

A prospective cohort study by Alessandri-Bonetti et al. (2023) indicated pain-related TMD and headache attributed to TMD improved significantly after 18 months of OSA treatment. [59]

Individuals undergoing mandibular advancement device (MAD) therapy for OSA may experience changes in jaw position and altered occlusion, which could potentially contribute to the development or exacerbation of TMD symptoms. A systematic review by Langaliya et al. (2023) indicated MAD treatment for OSA had varying effects on TMD-related outcomes. According to a few studies, MAD therapy significantly reduced the severity and frequency of TMD symptoms. However, other studies did not observe significant changes in TMD symptoms or TMJ-related parameters. While the overall results suggest no significant effect of MAD treatment on TMD symptoms, future studies should use standardized approaches to address the differences in results. [68]

A cross-sectional study by Moradi et al. (2026) found individuals with TMD demonstrated a substantially elevated risk for OSA and concluded the findings support integrating routine sleep apnea screening, using tools such as the STOP-BANG questionnaire, into standard TMD clinical evaluation. [62]

Mandibular tori

Oral tori are benign bony outgrowths of the maxilla and mandible. Multiple risk factors have been associated with the development or growth of these tori, including bruxism. [69] [70]

Maxillary tori are bony exostosis located on the hard palate and mandibular tori are bony exostoses located on the lingual surface of the mandible. Large mandibular tori have been associated with the presence of OSA. [71] [72]

There are theories suggesting mandibular tori may compete with the tongue for space in the oral cavity, leading to the displacement of the tongue and its associated soft tissues. This displacement can narrow the airway and increase the likelihood of OSA. Following

this reasoning, large tori potentially can cause greater displacement, thereby increasing the severity of OSA. [69] [72]

The presence of mandibular tori may also impact the treatment of OSA. Individuals who are not compliant with continuous positive airway pressure (CPAP) therapy may be prescribed mandibular advancement devices (MADs). However, the fit and comfort of the MAD could be compromised by mandibular tori, affecting its efficacy. Additionally, some authors have suggested tori could influence the success of surgical treatments for OSA and tori removal be part of the treatment plan. [69] [73] [74]

A systematic review and meta-analysis by Yong et al. (2025) found individuals with mandibular tori were more likely to have OSA, and larger tori were generally associated with the presence of OSA. While larger tori are more likely to be associated with OSA, individuals with severe OSA tend to have smaller tori. The evidence is conflicted on whether tori are a risk factor for OSA. Nevertheless, removal of the tori may not be necessary for MAD therapy or soft-tissue OSA surgery to be effective. While tori removal can ameliorate OSA, the improvements are modest. More research is required to determine the link between tori and OSA. [69]

Assessment

Assessment of OSA in adults may include:

- Review of the medical history, symptoms, and sleep history.
- Physical assessment, including measuring the body mass index (BMI), blood pressure, and neck circumference, and examining the nasal cavity.
- Oral examination, including Mallampati classification.
- Sleep study (polysomnography) performed either in a sleep lab or at home. An in-laboratory polysomnogram is considered the gold standard for diagnosing OSA. [4] [13] [75]

Risk assessment tools

Tools to assist with conducting OSA risk assessment may include the STOP-BANG questionnaire, Epworth Sleepiness Scale, and the Modified Mallampati score.

STOP-BANG questionnaire

The STOP-BANG questionnaire is a useful tool to determine the risk of OSA in individuals who snore. The questionnaire consists of a brief set of yes-or-no questions. Each “yes” answer scores one point. The total score ranges from 0 to 8. The questionnaire does not diagnose sleep apnea, but it can help determine whether someone needs further testing. [13]

Each letter in STOP-BANG represents a risk factor for sleep apnea:

- **S**noring
- **T**iredness during the day
- **O**bserved pauses in breathing during sleep
- **P**ressure (treated for high blood pressure)
- **B**ody mass index (BMI) more than 35 kg/m^2

- **A**ge older than 50
- **N**eck circumference (40cm/16 in or larger)
- **G**ender (male)

STOP-BANG questionnaire [1] [13] [76]

Answer "Yes" or "No" to the following questions. A total of "yes" to 3 or more items on this questionnaire is considered high risk for OSA.

S: Do you snore loudly?
T: Do you often feel tired, fatigued, or sleepy during daytime?
O: Has anyone observed you stop breathing during your sleep?
P: Do you have high blood pressure?
B: BMI >35?
A: Age >50?
N: Neck circumference >40 cm/16 in?
G: Gender male?

STOP-BANG score _____

A score of 3 or higher suggests an increased risk for OSA. The STOP-BANG questionnaire is often a first step in identifying individuals who may have OSA. However, the tool is meant for screening only. A high score does not mean a person has OSA, and a low score does not rule it out completely. A formal sleep evaluation is still needed for a diagnosis. [77]

STOP questionnaire

A simplified version of the STOP-BANG questionnaire is the STOP questionnaire that utilizes the first four questions for screening for OSA. A person is considered at high risk for OSA if they answer "yes" to two or more of the questions. [4] [32]

STOP questionnaire [4] [32] [78]

Question	Yes/No
S: Do you **snore** loudly?	
T: Are you **tired** during the day?	
O: Has anyone **observed** you stop breathing while sleeping?	
P: Do you have high blood **pressure**?	

Epworth Sleepiness Scale

The Epworth Sleepiness Scale (ESS) can be used to quantify daytime sleepiness. Eight questions ask the person to rate, on a four-point scale (0-3), their usual chances of dozing off or falling asleep while engaged in different activities. The total score ranges from 0 to 24. The higher the ESS score, the higher their daytime sleepiness. Results

from 0 to 10 show average (normal) daytime sleepiness. A result from 11 to 24 indicates excessive (abnormal) daytime sleepiness. [13] [79] [80]

In general ESS scores may be interpreted as follows:

- 0 to 5: Low daytime sleepiness (normal).
- 6 to 10: High daytime sleepiness (normal).
- 11 to 12: Mild excessive daytime sleepiness.
- 13 to 15: Moderate excessive daytime sleepiness.
- 16 to 24: Severe excessive daytime sleepiness. [79] [80]

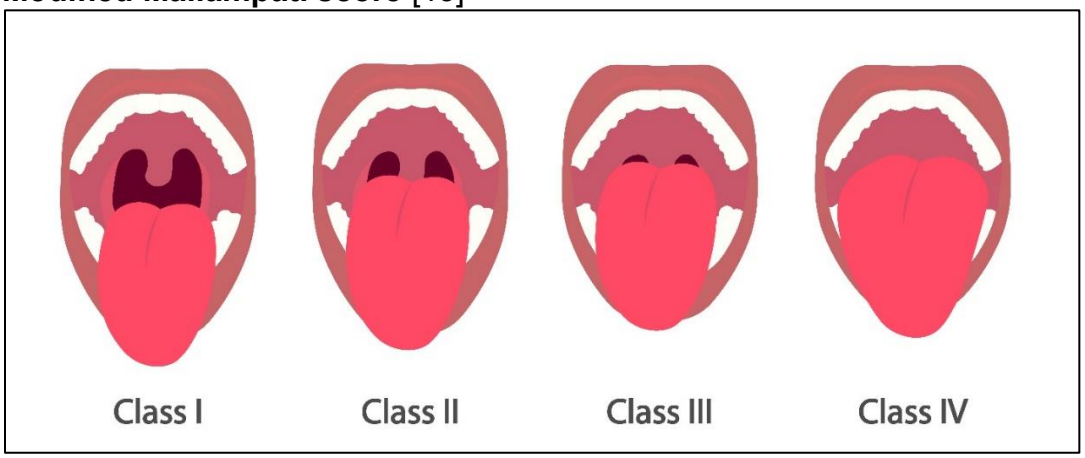
Epworth Sleepiness Scale [13]

Activity	**Chance of dozing** (0 = no chance; 1= slight chance; 2 = moderate chance; 3 = high chance)
Sitting and reading	
Watching TV	
Sitting inactive in a public place	
Sitting for an hour as a passenger in a car	
Lying down in the afternoon to rest	
Sitting and talking to someone	
Sitting quietly after a lunch without alcohol	
Sitting in a car, stopped for a few minutes due to traffic	
Total	

Modified Mallampati score

Modified Mallampati score can be used to assess the risk of OSA. A score of 3 or 4, where only the base or none of the uvula is visible during an oral examination, indicates an increased risk of OSA. [13] [14]

Modified Mallampati score [13]



Modified Mallampati scoring
Class I: Tonsils, uvula, and soft palate are fully visible.
Class II: Hard and soft palate, upper portion of tonsils, and uvula are visible.
Class III: Soft and hard palate and base of the uvula are visible.
Class IV: Only the hard palate is visible.

Screening for OSA in children

Oral health professionals have the opportunity to identify children at risk of OSA. Tonsillar enlargement and obesity are major risk factors for OSA in otherwise healthy children. With a history and careful clinical examination at each oral health visit, oral health professionals may identify signs and symptoms that may raise a concern for OSA. Assessment of tonsillar enlargement and the amount of airway obstruction by the modified Mallampati classification should be part of routine intraoral assessments. [12]

Validated screening tools are available for adult OSA (e.g., STOP-BANG, STOP, Epworth Sleepiness Scale). However, questionnaires for the pediatric population are not sensitive enough to detect the presence or severity of OSA. Nonetheless, the inclusion of sleep questions on the health history form may further help identify clients at risk.

Questions may include:

- Does your child snore loudly when sleeping?
- Does your child have trouble breathing while sleeping?
- Does your child stop breathing during sleep?
- Does your child occasionally wet the bed at night?
- Is your child hard to wake up in the morning?
- Does your child complain of headaches in the morning?
- Does your child tend to breathe through their mouth during the day?
- Have you or the teacher noted your child appears sleepy during the day?
- Does your child fall asleep quickly? [12]

Diagnosis

Information obtained from the medical history, as well as the extraoral and intraoral assessments, can reveal OSA risk factors. Clients suspected of being at risk for OSA should be referred to a medical professional (e.g., sleep medicine physician) for further evaluation and definitive diagnosis. Diagnostic confirmation requires polysomnography (sleep study). [4] [12] [30]

Treatment

Goals of OSA treatment include:

- Reducing symptoms,
- Improving quality of life,
- Reducing episodes of hypoxia and sleep fragmentation,
- Restoring sleep continuity, and

- Preventing potential complications (e.g., cardiovascular disease, motor vehicle accidents). [3] [29]

Effective treatments are available for OSA. Treatment depends on the severity of sleep apnea and may include:
- Controlling risk factors
- Positive airway pressure
- Oral appliances
- Surgery [3] [9]

Controlling risk factors
Lifestyle modifications can help control risk factors, reduce sleep apnea symptoms, and enhance treatment outcomes. These modifications may include:
- Improving healthy behaviours, such as balanced nutrition, regular physical activity, and sleep hygiene[13] may help reduce OSA severity by improving muscle tone, reducing weight, and increasing sleep quality. Gradual, sustainable changes will increase the likelihood of long-term maintenance.
- Weight loss if overweight. Excess weight can contribute to airway obstruction. Reducing body weight may lead to significant improvements in symptoms and a decrease in the severity of OSA. Some people still may need treatment for OSA after weight loss, although the symptoms are usually milder.
- Reducing alcohol use, as alcohol can exacerbate OSA symptoms by relaxing the muscles of the upper airway, which may lead to increased airway obstruction and more frequent apneas during sleep. Alcohol consumption, especially in the hours leading up to bedtime, can worsen OSA severity. Reducing alcohol intake can help improve sleep quality and the effectiveness of OSA treatment.
- Avoiding sedatives (e.g., sleeping pills), as these medications can excessively relax throat muscles and close off the airway and increase sleepiness.
- Sleeping on one side rather than the back may help reduce symptoms, particularly in cases of mild to moderate OSA. While sleeping on the back, gravity can impact the tissues at the back of the throat and neck, causing the upper airway to become narrow or collapse completely. Measures to encourage side sleeping may include attaching a tennis ball to the back of the nightshirt (e.g., in a pocket sewn to the back of the nightshirt) or sleeping with a pillow against the back to help prevent rolling onto the back.
- Elevating the head of the bed slightly may also assist in reducing OSA severity for individuals with mild to moderate OSA (e.g., placing blocks under two of the bed's legs to raise the head of the bed).
- Smoking cessation.[14]
- Treating nasal congestion (e.g., treating allergies) [2] [9] [18] [29] [75]

Weight loss and weight loss medications
Studies show weight loss medications glucagon-like peptide-1 receptor agonists (GLP-1RAs) can help improve symptoms of OSA in individuals with elevated body weight.

[13] Refer to Episode 156 for strategies to improve sleep hygiene.
[14] Refer to Episode 101 for additional information on tobacco cessation.

Further long-term studies are needed to confirm the sustained benefits and safety of GLP-1 RAs in OSA management. [75] [81] [82]

In 2024, the U.S. Food and Drug Administration (FDA) approved the weight-loss drug tirzepatide, marketed as Zepbound, in combination with diet and exercise, for the treatment of moderate to severe OSA in adults with obesity. [75] [83]

Currently in Canada, Zepbound (tirzepatide injection) is indicated for once-weekly administration for chronic weight management, including weight loss and weight maintenance, in combination with a calorie-reduced diet and increased physical activity, in adults with an initial BMI of:

- ≥30 kg/m^2 (obesity) or
- 27 kg/m^2 to <30 kg/m^2 (overweight) with at least one weight-related comorbid condition, such as OSA, hypertension, dyslipidemia, prediabetes, type 2 diabetes, or cardiovascular disease. [84]

Positive airway pressure

The therapy most studied and most often recommended for OSA is positive airway pressure (PAP). PAP systems for OSA treatment include continuous positive air pressure, auto-titrating positive airway pressure, and bilevel positive air pressure.

Continuous positive air pressure

Continuous positive airway pressure (CPAP) is the primary treatment for individuals with OSA. Research consistently shows CPAP reduces sleepiness and snoring while improving the sleep quality of bed partners.

CPAP involves wearing a mask over the nose or over the nose and mouth while asleep. The mask attaches to a small pump that supplies continuous pressurized air to hold the upper airway open. CPAP can be used with or without humidification. Some individuals find humidified air more comfortable and alleviate dry mouth often associated with CPAP use. Close follow-up by a medical provider is needed during the first 2 weeks of use to ensure proper mask fit and provide appropriate encouragement as the person learns to sleep with the mask.

However, many people find CPAP hard to tolerate, leading them to stop using it or use it inconsistently. Ensuring a proper fit and comfort is crucial for both effectiveness and adherence to the treatment. [2] [3] [85]

Auto-titrating positive airway pressure

Auto-titrating positive airway pressure (APAP), also called auto-CPAP, uses varying air pressure that automatically adjusts when the device senses changes in airway resistance. APAP can be helpful when the ideal fixed pressure for CPAP is difficult to identify. There is less evidence supporting APAP compared with CPAP, but it seems to offer equivalent results. [75] [86]

Bilevel positive air pressure
Bilevel positive air pressure (BiPAP) involves wearing a mask connected to a small pump. BiPAP provides two different pressures: one for inhaling and another for exhaling. BiPAP can be adjusted to deliver a breath if the user has not taken one within a certain number of seconds. BiPAP is less commonly used than CPAP or APAP. [75] [87]

Oral appliances
Oral appliances are a treatment option for OSA. Various oral appliances are available. These devices typically work by moving the mandible, tongue, and soft palate forward to increase pharyngeal airway space and reduce the risk of airway collapse. In general, adjustable oral appliances (i.e., titratable) are preferred over fixed devices. [2] [5]

The two main types of oral appliances are mandibular advancement devices (MADs) and tongue-retaining devices (TRDs). MADs engage the mandible and reposition it, and indirectly the tongue, in an anterior and forward position. They are the more commonly used option and are usually made of acrylic resin in two pieces that cover the upper and lower dental arches. TRDs directly engage the tongue and hold it forward. They are typically made of silicone and shaped like a bulb. The tongue is placed into the bulb, which is then squeezed and released to create suction that maintains the tongue in a forward position. [5]

Sleep physicians may prescribe MADs for primary snoring, mild-to-moderate obstructive sleep apnea, individuals unable or unwilling to use PAP therapy, and those with contraindications for surgery. However, this therapy is not recommended for individuals with TMDs, inability to sufficiently protrude the mandible (<10 mm), nasal obstruction, severe OSA, or central sleep apnea. Poor oral health (e.g., dental caries, abscesses, periodontal disease) should be managed before starting mandibular advancement therapy.[15] [88]

Before an oral appliance is prescribed or constructed by a dentist trained in sleep medicine, the client should undergo medical evaluation by a sleep physician to confirm the diagnosis and severity of OSA. This evaluation should also determine whether snoring can be managed independently by the dentist or whether treatment should proceed in collaboration with the attending physician because OSA is present. [5]

Although oral appliances are generally well tolerated, adverse effects are common. Most are minor, transient, and resolve quickly after the device is removed. Common adverse effects include TMJ pain, joint sounds, muscular pain, tooth pain, hypersalivation, gingival irritation, dry mouth, temporary chewing difficulties, and morning-after or permanent occlusal changes, such as tooth movement resulting in posterior open bite and/or decreased anterior overjet. [5]

[15] Listen to Episode 89 for discussion on oral self-care.

Clients should use their oral appliances every night, or during each sleep period, to optimize control of OSA symptoms. They should also be monitored over time for adverse effects related to appliance use. [2]

Persistent occlusal changes associated with oral appliance use may include proclination of the mandibular incisors, retroclination of the maxillary incisors, and posterior open bite. In some cases, these changes may require orthodontic treatment. [5]

Surgery
Surgery may be recommended for individuals who do not improve or respond to nonsurgical OSA therapies. Surgical procedures are used to adjust bone or soft tissues to reduce obstruction in the upper airways, to implant neurostimulator devices, or to reduce weight [2].

Surgical options include uvulopalatopharyngoplasty, maxillomandibular advancement surgery, tracheostomy, nerve stimulation implant surgery, nasal surgery, adenotonsillectomy, and bariatric surgery.

Uvulopalatopharyngoplasty
Uvulopalatopharyngoplasty (UPPP) removes soft tissue at the back of the throat to create more space in the oropharynx. This may include:
- All or part of the uvula
- Parts of the soft palate and tissue at the sides of the throat
- Tonsils and adenoids [89]

Some people who undergo UPPP surgery experience nasal regurgitation (i.e., movement of food or liquids backward into the nasal cavity during swallowing). [5]

Maxillomandibular advancement
Maxillomandibular advancement involves detaching both the upper and lower jaws and surgically advancing them anteriorly to increase space in the oropharynx to reduce airway obstruction. This procedure is most effective for individuals with retrognathia. It tends to be less successful in older adults or those with larger neck circumferences. [2] [90]

Tracheostomy
Tracheostomy is typically reserved for use as a potential interim treatment in select circumstances, such as severe life-threatening OSA, when other clinical options have failed or do not exist.

In this surgery, the surgeon creates a permanent opening in the neck to the trachea and inserts a tube to allow air in.
- A valve keeps the opening of the tube closed during the day, which allows the person to speak and breathe normally.
- At night, the individual opens the valve so that air bypasses the blockage in the throat and enters the lungs during sleep. [2] [75] [91]

Upper airway stimulation
Upper airway stimulation uses an implanted device to stimulate a branch of the hypoglossal nerve, activating the tongue and other muscles that help keep the airway open. It can be effective for some individuals with moderate to severe OSA who cannot tolerate CPAP and do not benefit from oral appliances. It may also be considered for those being evaluated for maxillomandibular advancement. [3] [85]

Nasal surgery
Nasal surgery can help reduce snoring and sleep apnea by clearing and enlarging the nasal passages. Types of nasal surgery include removing polyps, correcting a deviated septum (nasal septoplasty), and turbinate reduction. [5] [75]

Adenotonsillectomy
Adenotonsillectomy is the removal of tonsils and adenoids to create more space in the oropharynx. [3] [75]

Bariatric surgery
Bariatric surgery reduces the size of the stomach to help individuals lose weight. This surgery can help improve symptoms of OSA in people with obesity. [75]

Transoral neuromuscular electrical stimulation
Daytime neuromuscular electrical stimulation (NMES) of the tongue is a novel treatment for mild OSA and primary snoring. This method uses a silicone mouthpiece equipped with four electrodes that directly stimulate the tongue muscles with electrical currents. The goal is to strengthen these muscles and enhance their endurance, helping to prevent excessive relaxation during sleep.

The device is designed for use for 20 minutes each day for a duration of six weeks. After this initial period, it is used twice a week for 20 minutes to maintain muscle tone. Using daytime NMES alongside PAP therapy may further improve OSA symptoms. The device, known as eXciteOSA® has received approval from both the FDA and Health Canada. [92] [93] [94] [95] [96] [97]

OSA treatment in children
Treatment for OSA may be accomplished with either nonsurgical or surgical options, depending on its severity and etiology. Treatment of OSA in children may include:

- Sometimes watchful waiting for up to 6 months may be appropriate in young healthy children with mild OSA who do not exhibit daytime symptoms or severe abnormalities on polysomnography.
- Adenotonsillectomy is usually effective for children with OSA who are otherwise healthy and have enlarged tonsils and/or adenoids. This surgery may also improve some behaviours, quality of life, and blood pressure compared with watchful waiting. However, adenoidectomy alone is often ineffective. The risk of perioperative airway obstruction is higher in children with OSA than those without OSA undergoing adenotonsillectomy, so close monitoring is essential.
- In addition to adenotonsillectomy, children with OSA may benefit from orthodontic treatment, rapid maxillary expansion, or transverse palatal expansion.

- Alternate surgical procedures may be indicated depending on the anatomic abnormality causing OSA, such as uvulopalatopharyngoplasty, or tongue or midface surgeries and correction of congenital micrognathia.
- CPAP can be used for children who are not candidates for corrective surgery or who continue to have OSA after adenotonsillectomy.
- Weight loss can help reduce OSA severity and provide additional health benefits. However, it is rarely sufficient as a standalone treatment for OSA in the long term.
- Nocturnal oxygen supplementation may help prevent hypoxemia until definitive treatment is provided.
- Treatment of allergic rhinitis. However, corticosteroids and antibiotics are not usually indicated. [5] [12] [23] [24]

OSA awareness

Research suggests a lack of knowledge within dentistry and dental hygiene as well as dental and dental hygiene students about OSA and its management. This highlights the need to incorporate OSA into dental and dental hygiene educational curriculum and through ongoing professional development for oral health professionals. [98] [99] [100] [101]

Role of oral health professionals

Oral health professionals play an essential role in the multidisciplinary care of clients with OSA. Performing a comprehensive assessment is essential. Reviewing medical histories and asking questions about:

- Symptoms (e.g., excessive daytime sleepiness, snoring, witnessed apneas),
- Risk factors (e.g., tobacco, alcohol, and sedative use, and
- Concomitant conditions can help identify clients who may be affected by or at risk for OSA.

Evaluating risk factors, such as hypertension[16] and obesity and performing thorough extraoral and intraoral assessments, noting any clinical features associated with the disorder, such as large neck circumference, small, recessed mandible, large tonsils, tooth wear, etc. Findings from the medical history, clinical assessment, and screening can help identify clients requiring referral to an appropriate medical provider for further evaluation. Referrals can lead to a definitive diagnosis, which can improve the management and prognosis of these clients.

Oral health professionals can also educate clients about the health risks, symptoms, and treatment options associated with OSA. Education should also focus on lifestyle modifications, such as smoking cessation. [4] [30] [102] [103]

Novel therapies for sleep disorders

Oral healthcare has an increasingly important role in the management of sleep health and sleep disorders. Novel dental therapies may serve as effective adjunctive treatment options for managing OSA and snoring. Simmons et al. (2026) synthesized the 2024 American Academy of Dental Sleep Medicine (AADSM) consensus report on the

[16] Refer to Episodes 135 and 136 for additional information on hypertension.

evidence supporting new and emerging dental interventions for sleep health and sleep-related disorders. The analysis reviewed the AADSM consensus panel report and its cited peer-reviewed studies, along with additional literature on dental treatments for OSA and snoring without OSA.

Areas of focus included premolar extraction for orthodontics, expansion orthodontics, myofunctional therapy, frenum release, and laser therapies for OSA and snoring.

No emerging dental therapy met the criteria to serve as a standalone substitute for mandibular advancement devices for treating OSA or snoring. Some approaches may play adjunctive or individualized roles when established therapies are ineffective or declined. Evidence did not support orthodontic extractions as either a risk factor for OSA or a therapeutic option, while expansion orthodontics showed limited potential benefit in some pediatric cases without consensus for first-line use.

The authors stated that myofunctional therapy is:

> *Considered a low-risk intervention with some supportive evidence for managing snoring and OSA, but not curing or preventing these disorders, particularly as an adjunct with potentially more impact at an early age. Patient satisfaction is often high, but standardized protocols and stronger trials are required. Demand times of treatment from 30 through 60 minutes of daily myofunctional exercise will likely limit practicality in adults.*[17]

Frenum release and ablative laser therapies were strongly discouraged due to lack of evidence and potential risks, while nonablative laser treatments showed only short-term promise for snoring.

However, this study was limited by inconsistent study designs and narrow sample diversity, highlighting the need for standardized protocols and more diverse, clearly defined cohorts to improve clinical relevance.

The authors concluded that oral health professionals contribute to multidisciplinary sleep care through established therapies, selective use of emerging interventions, and early recognition of airway growth issues. Adoption of new therapies should follow expert consensus and robust evidence until stronger data are available. [104]

Novel research

AD109 is a once-nightly oral medication being studied for OSA. In a large phase III trial involving 646 adults with mild to severe OSA who could not tolerate or declined CPAP, the drug improved breathing during sleep, reduced oxygen desaturation, and lowered hypoxic burden. Over 40% of participants improved their OSA severity category, and 18% achieved complete disease control.

[17] Note: Registered dental hygienists practising myofunctional therapy should contact a CDHO practice advisor to understand myofunctional therapy's role in snoring and OSA to ensure they remain within the dental hygiene scope of practice.

AD109 combines aroxybutynin and atomoxetine to help keep throat muscles active and reduce airway collapse during sleep. Participants taking AD109 experienced about a 44% reduction in apnea-hypoxia index compared with 18% in the control group. Side effects were generally mild and included dry mouth, nausea, and insomnia, although about 21% of participants discontinued treatment because of side effects. AD109 has received FDA Fast Track designation, and its manufacturer has submitted it for review, with a possible FDA decision expected in early 2027. [105] [106] [107]

Key messages:

- Untreated OSA can lead to daytime sleepiness and trouble thinking clearly, and increases the risk of vehicle and workplace accidents, depression, vascular dementia, hypertension, cardiac arrhythmias, stroke, and heart attack.
- OSA increases the risk of morbidity and mortality, underscoring the need for interprofessional collaboration in diagnosing and managing this condition.
- Oral health professionals are uniquely situated to help detect early signs of OSA and identify clients who may benefit from seeing a sleep specialist. Early recognition, referral, and treatment of OSA can help prevent adverse health consequences.
- By screening for OSA and collaborating with other healthcare providers, oral health professionals can improve long-term health outcomes for their clients.
- Educating clients about OSA risk factors, concomitant issues, and treatment options is crucial for managing OSA as it transforms clients into active partners in their own care. Without a clear understanding of the risks, related medical conditions, and available interventions, clients are far less likely to recognize the severity of their symptoms or adhere to long-term therapies.

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