# Dental Comorbidities and Risk Factors of Sleep-Disordered Breathing

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G. Gary Demerjian, Pooja Goel, Mayoor Patel, Anthony Sims, Rachel-Marie Demerjian, and André Barkhordarian

## Abbreviations

| | |
|---|---|
| AHI | Apnea-hypopnea index |
| BMI | Body mass index |
| DSA | Dental sleep appliance |
| HH | Hypnic headache |
| OAT | Oral appliance therapy |
| OHS | Obstructive hypopnea syndrome |
| OSA | Obstructive sleep apnea |
| RDI | Respiratory distress index |
| REM | Rapid eye movement |
| RERA | Respiratory effort-related arousal |
| RME | Rapid maxillary expansion |
| SB | Sleep bruxism |

G. G. Demerjian (✉)
Center for TMJ and Sleep Therapy, Glendora, CA, USA
e-mail: drd@tmjdemerjian.com

P. Goel
Smile for life Dental Group, Santa Clara, CA, USA

M. Patel
Craniofacial Pain and Dental Sleep Center, Atlanta, GA, USA

A. Sims
Maryland Center for Craniofacial TMJ and Dental Sleep Disorders, Ellicott City, MD, USA

R.-M. Demerjian
Center for TMJ and Sleep Therapy, Glendora, CA, USA

A. Barkhordarian
Department of Oral Biology and Medicine, UCLA School of Dentistry, Los Angeles, CA, USA



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| SRBD | Sleep-related breathing disorder |
| T&A | Adenotonsillectomy |
| TMD | Temporomandibular joint dysfunction |
| TTH | Tension-type headache |
| UARS | Upper airway resistance syndrome |
| VDO | Vertical dimension of occlusion |

## 6.1 Introduction

Sleep-related breathing disorders (SRBDs) refer to several pathologies which include snoring, upper airway resistance syndrome (UARS), obstructive hypopnea syndrome (OHS), and obstructive sleep apnea (OSA). OSA occurs in approximately 5–15% of women of the population. The pathophysiology of OSA is characterized by repetitive oropharyngeal collapse and occlusions during sleep, which obstructs the airway. It is associated with sleep fragmentation, hypoxemia, hypercapnia, marked swings in intrathoracic pressure, increased sympathetic activity, and cardiovascular complications [1, 2].

OSA causes oxyhemoglobin desaturation, persistent inspiratory efforts due to the occluded airway, and termination by arousal from sleep. OSA is associated with fatigue and daytime sleepiness, due to fragmented sleep caused by recurrent arousals. Sleep deprivation impairs host defense mechanisms, and consequently, it might be associated with changes that affect the components and responses of the immune system. OSA was associated with increased levels of inflammatory markers and cytokines in the blood such as C-reactive protein (CRP), IL-8, IL-6, and TNF-α [3–8]. Evidence shows that patients with OSA have an increased risk of developing several medical conditions such as incidence of hypertension, implicated in stroke and transient ischemic attacks, coronary heart disease, heart failure, and cardiac arrhythmias. If patients have comorbid conditions of OSA and preexisting pulmonary disease, they may develop pulmonary hypertension as a result [2]. The actual cause linking OSA with cardiovascular disease is unknown, but evidence shows that OSA is associated with pro-inflammatory and prothrombotic factors which have been identified in the development of atherosclerosis. OSA is associated with an increase in daytime and nocturnal sympathetic activity. Autonomic abnormalities seen in OSA patients include increase in resting heart rate and blood pressure variability. Furthermore, OSA and atherosclerosis are associated with endothelial dysfunction indicated by an increase in C-reactive protein, fibrinogen, interleukin 6, and reduction of plasminogen and fibrinolytic activity. The prevalence of OSA in the adult population is estimated to be between 2 and 4%, with the major factors being age, sex, and weight [9–11]. The Wisconsin Sleep Cohort Study reported that the prevalence of AHI greater than five per hour in 30–60-year-old men is 24% and women is 9% [12].

There are multiple structural, orthopedic, and physical contributing factors that a dental healthcare physician looks at on a daily basis in the field of dental sleep

medicine during a physical examination. When looking at the craniofacial evaluation, neck size, and intraoral structures, there are common factors that contribute to the collapse of the oropharyngeal structures. Such factors include the elongation of the soft palate and uvula from the pulling forces that have been put on it from snoring and loss of vertical dimension resulting in a shortening of the lower one-third of the face. This can be due to bruxism resulting in attrition of teeth, clenching or extraction of teeth causing a loss in jaw support [13], increase in tongue size due to weight gain and fat deposition in the tongue [14, 15], and constriction of dental arches [16] due to improper tongue position, extraction of first bicuspids when wearing braces and headgear, and negative transmural pressure gradient and tissue weight.

## 6.2 Causes of OSA

Oropharyngeal patency is dependent on several factors, tongue size and position, tongue space in the oral cavity during occlusion, and balance between collapsing and dilating forces of the oropharynx. Contraction of dilator muscles causes stiffening of oropharyngeal tissues resulting in dilation but can still occur in patients with OSA during an obstructive event [17]. Studies show that tension produced during contraction of the dilator muscle is higher due to OSA [18, 19]. Subjects with OSA who snore have a higher rate of uvular stiffness, when compared with non-OSA subjects [19]. Recurrent or chronic OSA can lead to development of an inflammation, causing histologic alterations of oropharyngeal tissues, leading to alteration in the integrity of the extracellular matrix, and interferes with the mechanical properties of soft tissues [1]. Inflammation caused by plasma cell infiltration and interstitial edema is present in the uvula mucosa of OSA patients, suggesting that soft palate inflammation contributes to upper airway occlusion observed during sleep in these patients [20].

OSA is the most common form of sleep apnea. There are various forms of sleep apnea, which are obstructive, central, and complex sleep apneas. OSA is a chronic clinical syndrome characterized by snoring, apnea during sleep (episodes of oropharyngeal collapse), hypoxemia (low oxygen levels) during sleep, and daytime hypersomnolence (sleepiness) [21, 22]. The disorder is characterized by repetitive collapse (apnea) or partial collapse (hypopnea) of the pharyngeal airway during sleep [23]. OSA is classified as cessation of breath for ≥10 s. In 2007, there were some changes made by the task force in the respiratory scoring rules. Apnea in adults is scored when there is a drop in airflow by ≥90% from normal airflow for ≥10 s. A hypopnea in adults is when there is a drop in airflow by ≥30% for more than ≥10 s in association with either ≥4% arterial oxygen desaturation or an arousal.

The numbers of both event types such as apnea and hypopneas are ultimately combined to compute an apnea-hypopnea index (AHI) [23]. OSA is defined as AHI or respiratory distress index (RDI) greater than five events in an hour and is associated with symptoms such as excessive daytime sleepiness, impaired cognition,

mood disorders, insomnia, hypertension, ischemic heart diseases, or history of stroke.

There are multiple risk factors for patients diagnosed with OSA. Among genetic and social factors, patients with OSA have a narrow oropharyngeal airway, which is commonly due to being overweight and absence of tongue space in adults and enlarged tonsils in children. During rapid eye movement (REM) sleep, the muscles of the oropharynx and tongue relax and therefore cause the oropharyngeal airway to narrow and collapse during intervals of OSA [2]. Risk factors of OSA, from the dental perspective, can be as a result from attrition of teeth; clenching or extraction of teeth causing a loss in jaw support [13]; increase in tongue size due to weight gain and fat deposition in the tongue [14], which is due to weight gain [15]; and constriction of dental arches [16] due to improper tongue position, extraction of first bicuspids when wearing braces and headgear, and negative transmural pressure gradient and tissue weight.

## 6.3 Orofacial Risk Factors

### 6.3.1 Obesity

When a patient enters the dental office with an assessment and diagnosis of OSA from a physician and wants dental sleep appliance (DSA) therapy also known as oral appliance therapy, the dentist should make a mental note of obesity as it is the most common risk factor of obstructive sleep apnea. Patients who are overweight have a higher chance of developing symptoms for OSA. Obesity relates to OSA due to the excess fatty tissue, thickening of the walls, and decreased lung volume [24]. If a patient is overweight, thickening of the lateral walls occurs compromising the airway passage, which may cause choking during or fragmented sleep. Thickening of the lateral walls can be seen in a computerized tomography (CT) scan or magnetic resonance imaging (MRI). When body weight increases, excess fat starts to develop on the muscular tissue, which narrows the airway. Obesity also contributes indirectly to upper airway narrowing, due to hypotonic airway during sleep. Lung volume reduces due to a combination of increased abdominal fat mass and the recumbent posture [25].

### 6.3.2 Narrow Airway Passages

Narrow airways hinder normal breathing during sleep, which can lead to respiratory effort-related arousals (RERAs), hypopneas, and apneas. The primary factor of a narrow airway leading to OSA can be a result of craniofacial skeletal deficiency. Improper development of the maxillary and/or mandibular bones can result in a narrow airway [24]. A narrowed airway causes snoring, a common symptom of OSA. An airway can be narrowed by increase or enlargement of the soft tissue [2,

25]. Narrowing of the airway can also be caused by aging, as soft tissue and muscles sag. Furthermore, hormonal factors such as the presence of testosterone or the absence of progesterone can cause airway narrowing [26].

### 6.3.3 Nasal Congestion/Obstruction

A small nostril size, narrow nasal valves, and nasal congestion increase the risk of both snoring and OSA. Breathing through the nasal airway is important and idealistic for improved sleep. If the nasal airway is constricted or congested, the patient is forced to breathe through his or her mouth [27]. Nasal congestion is a risk factor due to allergic rhinitis or an acute upper airway infection. Nasal congestion is commonly related to anatomical abnormalities such as deviated septum, conchal hypertrophy, and nasal polyps [28]. Nasal breathing is better for the patient as the lungs will absorb more nitric oxides, due to the back pressure from the resistance air flowing out of the sinuses, when compared to no resistance when breathing through the mouth [29].

### 6.3.4 Mouth Breathing

Since SRBD has serious consequences for long-term health and quality of life, early diagnosis of SRBD is essential. Healthcare professionals can play an important role in the early diagnosis of SRBD by recognizing distinct facial morphologies such as long face, reduced nose prominence, and retrognathic mandible and referring these children to specialists for further assessment of SRBD clinical symptoms. There are several studies worldwide that show the prevalence of mouth breathing as a risk factor and/or the perpetuation factor of sleep apnea. Mouth breathing can be commonly seen in patients with some nasal obstruction due to pharyngeal lymphoid tissue hypertrophy and intranasal deformities such as nasal septum deviation, polyps, tumor, and allergic rhinitis [30].

Habitual mouth breathers have the habit of sleeping with their mouth open, without a correlation to a medical condition. Both habit and nasal obstruction-related mouth breathing may cause facial muscle imbalance and craniofacial changes. Facial musculature imbalance occurs as a result of mouth breathing, which causes changes in tongue position, tooth positioning, lips, palate, and jaws, so as to counterbalance the new breathing pattern [31]. The most common findings in people with mouth breathing are lack of lip seal, incompetent lips, postural changes (forward posture of the head to facilitate better breathing), dark circles around the eyes because of the sagging and hypofunction of the orbicularis oris muscle, long face due to the downward growth of the mandible, anterior open bite due to proclination of maxillary and mandibular incisors, and high vaulted and atresia of the palate because of imbalance of forces (Fig. 6.1). The tongue can take a low and forward position, which is common in the presence of hypertrophic palatine tonsils, as an

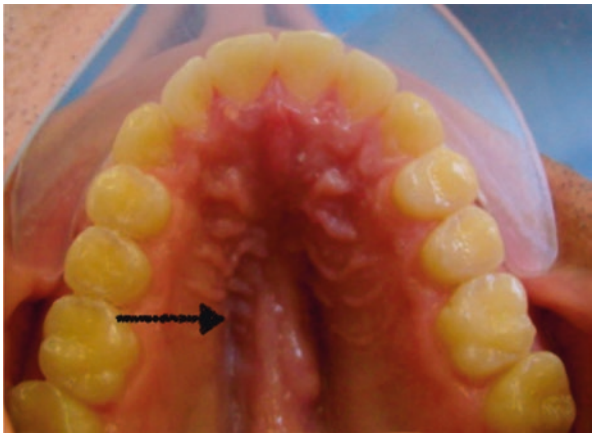

**Fig. 6.1** Narrow/vaulted maxilla. Bicuspids and first molars are more palatal in relation to the second molars. Arrow points to a high palatal arch. (Figure reprinted with permission [2])

attempt to increase posterior airway space and ease breathing [31]. The low position of the tongue decreases internal pressure in the upper arch, increasing the external pressure of perioral muscles and causing abnormal narrowing of the palate. The proper balance between bones, muscles, and dental structures is essential to avoid anatomical and functional changes resulting in an imbalance. All this cascade of events lead to an underdeveloped lower jaw, which is pointed downward, and long face syndrome, ultimately leading to adenotonsillar hypertrophy and narrowing of the upper airway [32]. Among mouth breathers, it is also common to find the possibility of OSA. OSA is common among 7–10% of children between the ages of 1 and 10. OSA in children is a disease characterized by partial prolonged and/or complete obstruction of the upper airways, impairing normal ventilation. The signs and symptoms of OSA include snoring, fragmented sleep, and neurocognitive and behavioral disorders such as learning disorders, behavioral changes, and attention deficit hyperactivity disorder (ADHD) [33, 34]. The major complications of the OSA include growth and developmental delays, mental retardation, and cor pulmonale [35]. Chapter 14 will discuss at length the subject of pediatric OSA.

### 6.3.5 Large Tongue

The tongue is known to be the most important pharyngeal dilator muscle, which is unique and moves freely, unlike other muscles. The oropharynx is a highly collapsible area and lacks rigid supporting structures, but the dilating pharynx muscles, especially the genioglossus, prevent the tendency of the pharynx to collapse. Macroglossia can be associated with a wide range of congenital and acquired conditions, or it can occur as an isolated feature (with no other abnormalities).

The prevalence of OSA is increasing among the general population in correlation with the rise in weight gain, as obesity is a major risk factor of developing OSA [36, 37]. A human autopsy study demonstrated that the tongue has a high percentage of fat localized at the tongue base and the tongue weight and fat percentage correlated to the degree of obesity [38]. Animal studies had similar results [39].

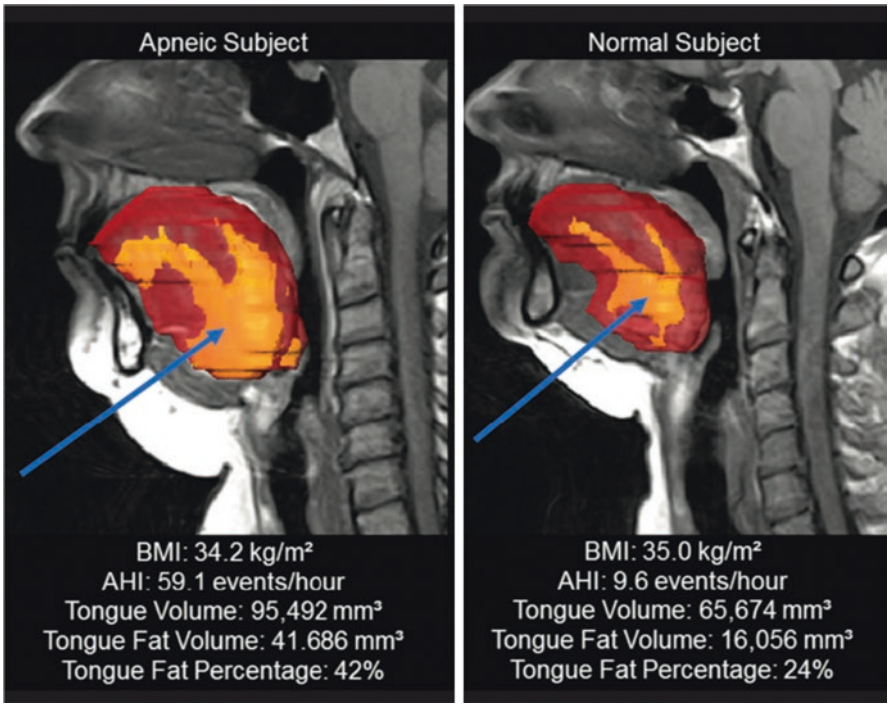


**Fig. 6.2** Tongue size/fat deposition. MRI cross section of a patient with OSA versus normal (non-OSA). Notice the shape of the tongue. Arrow points to fat deposition in the tongue. (Figure adapted from [14])

During an MRI study, the volumetric analysis of soft tissues and intramuscular fat in the tongue and masseter were measured. Patients with more obesity had a larger tongue as well as higher percentage of tongue fat. There were significant correlations (0.44; $P < 0.0001$) between visceral fat in the abdomen and tongue fat (Fig. 6.2) [14]. This study had four conclusions: (1) apneics have enlarged tongue volumes and increased fat within the tongue compared to control subjects; (2) the tongue contained more fat than the masseter muscle in both apneics and controls; (3) tongue fat percentage was higher in apneics, with the greatest among far located in the retroglossal region; and (4) tongue fat volume correlates with AHI and BMI [14].

Changes in tongue size due secondary to fat increase in the tongue can alter airway collapsibility and Pcrit (closing pressure) [14]. Eckert and colleagues showed in a test that tongue force fatigability (tongue protrusion) occurred more rapidly in patients with OSA than control in the control group [40].

Parapharyngeal fat pads have also been shown to be enlarged in apneics contributing to velopharyngeal narrowing [41]. Statistically, the size of the parapharyngeal fat pads was not significantly different among apneics and normal subjects [42]. This suggested that obesity compromises the airway in apneics through other mechanisms, not through fat deposition in the parapharyngeal fat pads. Li and colleagues

showed that apneics have increased fat deposition within the soft palate compared to controls, depending on BMI when standard T1-weighted spin echo MRI is used [43]. Lastly, weight loss or myofunctional therapy improves OSA and may decrease tongue fat [44].

### 6.3.6 Tongue Scalloping

Tongue scalloping is defined as multiple lateral glossal indentations resulting from molar compression. This condition is secondary to either glossopalatal disproportion alone or in combination with macroglossia. In one study, it was found that tongue scalloping showed a positive predictive value of 67% for abnormal AHI, 89% for apnea or hypopnea, and 89% for nocturnal desaturation (Figs. 6.3 and 6.4) [45].

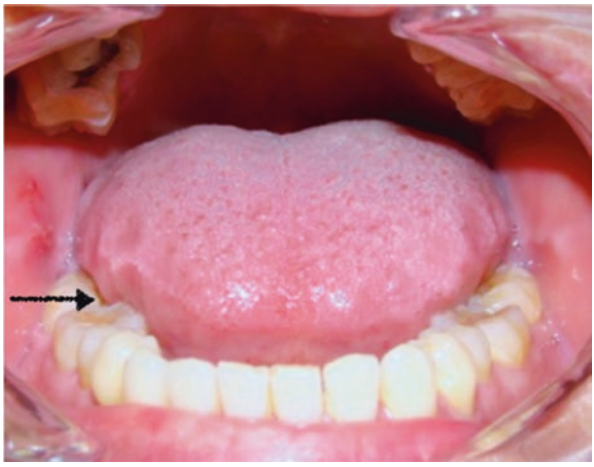

**Fig. 6.3** Large and scalloped tongue. Large tongue is resting above the occlusal plane of teeth. Arrow on the side of the tongue is pointing to the scalloping where the tongue is taking the shape of the teeth. (Figure reprinted with permission [2])

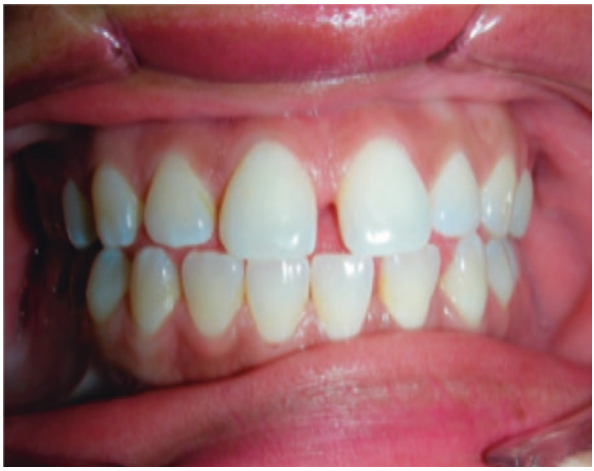

**Fig. 6.4** Large tongue/interproximal spacing. Space developing between the teeth due to the tongue is pushing when swallowing due to the limited tongue space. (Figure reprinted with permission [2])

## 6.4 Bruxism and Related Conditions

Bruxism is of great interest to researchers and clinicians in the fields of dentistry and sleep medicine. The etiology remains largely unknown, but current evidence supports the hypothesis of a multifactorial etiology. This involves sleep arousal mechanisms, autonomic sympathetic cardiac activation, sleep-related respiratory conditions, and genetic and various psychological exogenous factors. The role of SRBD for sleep bruxism (SB) has gained much attention in recent times. SRBDs, such as snoring (odds ratio 1.4) and obstructive sleep apnea (odds ratio 1.8), are reported to slightly increase the risk of SB [46]. There have been numerous discussions, classifications, and definitions of bruxism over the past several decades. In March of 2017, an international consensus meeting was held regarding the assessment of bruxism status, with bruxism experts from around the world. The aim of the consensus meeting was (a) to clarify the definition of bruxism, by separating definition into sleep bruxism and awake bruxism; (b) to determine the status of bruxism, whether it should be considered a disorder or behavior; (c) to review the assessment of bruxism; and (d) to develop a research agenda for future studies on bruxism topics [47].

As sleep and awake bruxism are considered two different behaviors, the single definition of bruxism was "retired" observed during sleep and wakefulness, respectively; the single definition for bruxism is recommended to be "retired" in favor of two separate definitions:

1. Sleep bruxism is a masticatory muscle activity during sleep that is characterized as rhythmic (phasic) or nonrhythmic (tonic) and is not a movement disorder or a sleep disorder in otherwise healthy individuals.
2. Awake bruxism is a masticatory muscle activity during wakefulness that is characterized by repetitive or sustained tooth contact and/or by bracing or thrusting of the mandible and is not a movement disorder in otherwise healthy individuals.

Both definitions of bruxism emphasize the role of the masticatory muscle as the source of potential clinical consequence and should not be limited and can include medical measures from sleep studies (e.g., heart rate variability, respiratory parameters, audio–video recordings). Due to the ending of both definitions of "in otherwise healthy individuals," they conclude that bruxism is not a disorder but a sign of a disorder such as people having rapid eye movement (REM) behavior disorder.

The *Orofacial Pain, Guidelines and Assessment, Diagnosis and Management* (*Sixth Edition*) defines bruxism as "a repetitive jaw-muscle activity characterized by clenching or grinding of the teeth and/or by bracing or thrusting of the mandible; can occur during sleep (sleep bruxism) or during wakefulness (awake bruxism)" [48].

Common clinical symptoms associated with bruxism are attrition (tooth wear), loss of vertical dimension, recession including bone loss around the dentition, pulpitis, bone overgrowth (tori, exostosis), failing or fracturing dental restorations, and orofacial/craniofacial pain (TMJ pain, myalgia, myofascial pain).

## 6.5 Dental Clinical Signs of Bruxism

### 6.5.1 Attrition

Dental attrition is considered the most visible sign of functional wear on dentition due to bruxism. Parafunctional habits of bruxism and clenching are a major concern for dental professionals (Fig. 6.5) [49].

### 6.5.2 Abfraction

Abfraction is thought to take place when excessive cyclic, non-axial tooth loading (bruxism) leads to cusp flexure and stress concentration in the vulnerable cervical region of teeth (Fig. 6.6). Elevator muscles in particular the masseter have been shown to activate during inspiratory resistance loading (mimicking an obstruction in the airway) [50]. Yet in another study, masseter muscle activation followed hypercapnia (seen in OSA). Additionally, recruitment of the muscle increased linearly with increasing carbon dioxide concentrations [51]. Activation of masseters is then believed to directly or indirectly contribute loading to teeth leading to the loss of cervical tooth substance. Clinical studies have shown associations between abfraction lesions, bruxism, and occlusal factors, such as premature contacts and wear facets, but these investigations do not confirm causal relationships [52].

### 6.5.3 Tori and Buccal Exostosis

It has been suggested that maxillary and mandibular tori are markers of increased craniofacial muscle activity such as bruxism, discussed in detail in this chapter. The concept of bone remodeling or growth as it adapts to mechanical forces is called Wolff's law [54]. The two maxillary bones come together at the midpalatal suture to form the palate. This suture remains patent well into adulthood. Due to daytime and sleep bruxism, heavy repetitive forces may lead to flexing and buckling of the

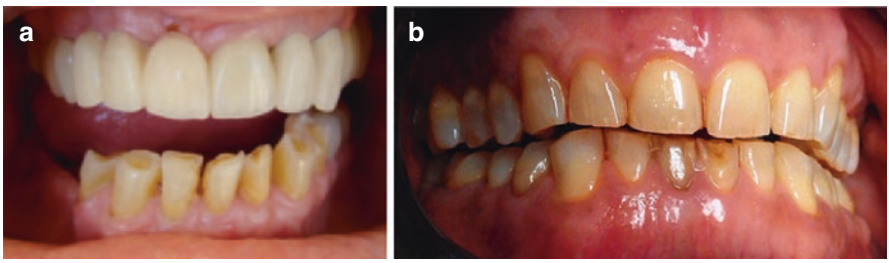


**Fig. 6.5** Attrition/worn dentition: (**a**) Attrition of lower dentition due to upper restoration being more abrasive. (**b**) Maxillary and mandibular incisal edges have similar wear due to attrition. (Figure reprinted with permission [2])

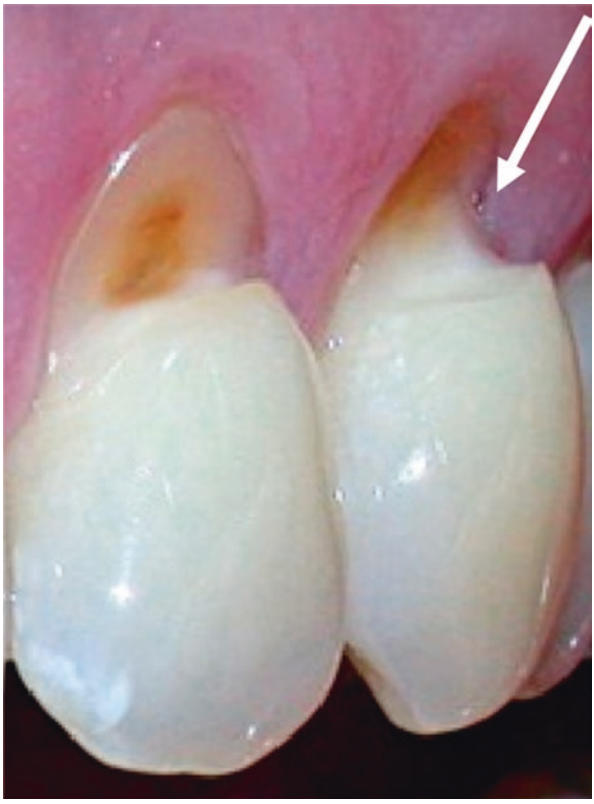

**Fig. 6.6** Abfraction is a concavity of the tooth structure at the gumline caused by lateral forces placed on the teeth. Arrow points to the abfraction area on the tooth. (Figure adapted from [53])

maxilla at the weakest point being at the midline (midpalatal suture). This intermittently tension leads to new bone formation localized to the midline, causing the formation of the maxillary tori [55]. Sleep bruxism can lead to hypertrophy of bilateral masseters and tendinous insertions at the angle of the mandible, resulting in antegonial notch. This is often present in patients with symptoms of temporomandibular joint dysfunction. These consequences may also be explained by the functional matrix hypothesis [55, 56].

Regarding the osteogenic-periosteal stretch hypothesis, the chin is prevented from undergoing excessive deformation due to the mental process. Humans lack the simian shelf seen in other mammals but instead have a developed chin to strengthen the weakest part of the mandible. Therefore, due to this morphology, the forces on the mandible are localized to the weakest point, being at the premolar region. The body of the mandible flexes medially due to muscular compression and tooth orientation directed by the maxilla. When the teeth are in full occlusion, the buccal overjet and curve of Spee ensure that mandibular dentition bends medially [55]. It has been suggested that due to heavy mastication forces, a protective mechanism of microfractures develops in the bone causing osteoblastic activity in order to repair

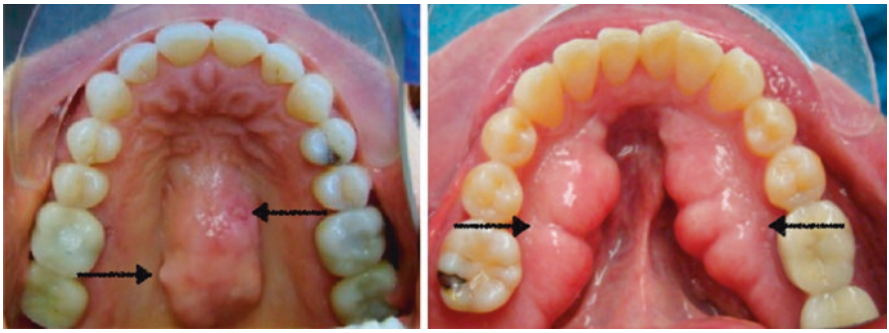

**Fig. 6.7** Tori: an overgrowth of the bone typically seen in the lingual aspect of the teeth, either at the middle of the palate or on the premolar section of the mandible. (Figure reprinted with permission [2])

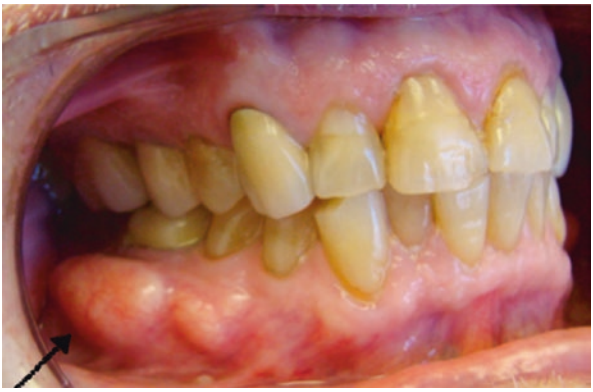

**Fig. 6.8** Buccal exostosis: overgrowth of the bone on the cheek side of the teeth. (Figure reprinted with permission [2])

the microfracture, thus causing an overgrowth of the bone [57]. See Figs. 6.7 and 6.8 for various overgrowths of the bone as explained above.

It has been hypothesized that mandibular tori can intrude on the space in the upper airway and promote sleep apneas [58, 59]. Maxillary and mandibular tori can sometimes reach a size at which they interfere with the space for the tongue and lead to decreasing volume of space within the oral cavity. Hence, the tongue falls back due to crowding, leading to impingement in the oropharyngeal region and upper airway obstruction leading to OSA. In a 2016 study, it was concluded that if mandibular tori is larger than 2 cm, then there is a possibility of having OSA [60].

### 6.5.4 Loss of Vertical Dimension

There are several studies aimed at the association between decreased vertical dimension and loss of oropharyngeal space by the collapse of orofacial structures. Loss of vertical dimension can affect the pharyngeal airway passage (Fig. 6.9). Vertical dimension of occlusion (VDO) is the relationship of the maxilla and the mandible when the teeth are occluded in maximum intercuspation. Loss of vertical dimension can be due to several factors including loss or absence of posterior dentition and bruxism followed by severe attrition [61]. This can influence oropharyngeal size and function as well as reduce lower face height and mandibular rotation [61]. There are several studies aimed at the association between decreased vertical dimension and loss of oropharyngeal space by the collapse of orofacial structures. Loss of vertical dimension constricts the tongue causing it to retract into oropharyngeal airway space. Evidence indicates that having an acceptable VDO can increase oropharyngeal space and improve OSA [62, 63].

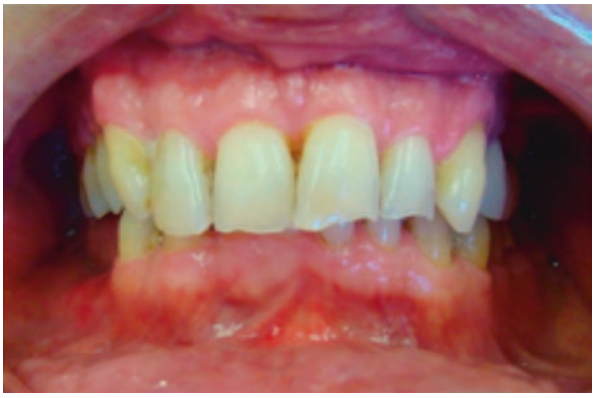

**Fig. 6.9** Loss of vertical dimension/deep bite. Maxillary incisal edges are worn down. Mandibular incisors almost fully covered by maxillary incisors due to deep overbite. (Figure reprinted with permission [2])

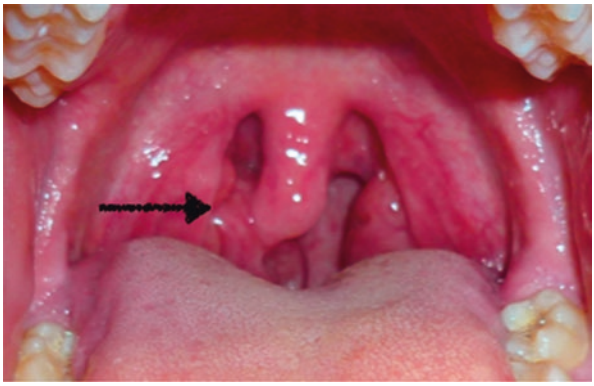

**Fig. 6.10** Elongated uvula. (Figure reprinted with permission [2])

### 6.5.5 Soft Palate and Elongated Uvula

The narrowest area of the airway between the posterior nasal opening and the epiglottis is located in the oropharynx. This is the site of airway obstruction due to the collapsibility. Some studies revealed that patients with OSA had a significantly longer soft palate length in proportion to their oropharyngeal airway when compared to controls, As well as men compared to women than controls (Fig. 6.10). Soft palate length increases with age in males and is smaller in females after adjusting for body mass index (BMI) and OSA status. This can be used to identify patients at risk for OSA in combination with their age [64]. Elongation of the soft palate and enlarged uvula may further compromise the airway by impinging on the nasopharynx and oropharynx. Chang et al. included a systematic review that revealed the relationship between uvula size, snoring, and OSA. Large uvulas were associated with more severe snoring and OSA [65].

### 6.5.6 Tonsils

OSA affects 2–3% of all children [66]. Adenotonsillar hypertrophy is the major pathophysiological contributor in children who have OSA [67], and adenotonsillectomy (T&A) remains as the first line of treatment [68]. Untreated OSA can result in several morbid consequences affecting cognition, behavior, and cardiovascular systems [33, 69]. Upper airway obstruction due to enlarged tonsils results in limited airflow. Such limitation is caused by a mechanical blockage that obstructs airflow, leading to mouth breathing. See Fig. 6.11 for a visual grading scale of tonsillar tissues.

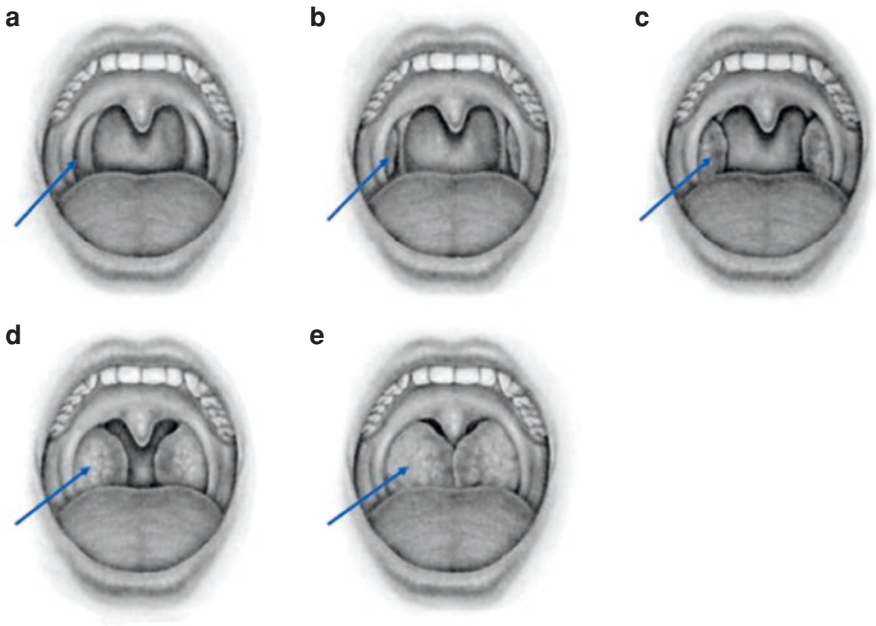


**Fig. 6.11** Tonsil grades 0–4. Blue arrow pointing to tonsil. Tonsils were classified by degree according to hypertrophy as follows: (**a**) Grade 0, tonsils inside the tonsillar fossa lateral to posterior pillars or previous tonsillectomy. (**b**) Grade 1, tonsils occupying 25% of the oropharynx. (**c**) Grade 2, tonsils occupying 50% of the oropharynx. (**d**) Grade 3, tonsils occupying 75% or more of the oropharynx. (**e**) Grade 4, almost meeting in the midline. (Figure adapted from [70])

## 6.6 Malocclusion

Malocclusion is an irregularity that tends to make a subject breathe through their mouth more prominently as compared to nasal breathing [71]. Increasing evidence demonstrates that OSA patients have dentofacial/skeletal characteristics associated with a narrow upper airway. In turn, that leads to the downward and backward rotation of the mandible and tongue and occlusion into the retropalatal (velopharynx) and retroglossal (oropharynx) [71, 72].

Class I occlusion is known as normal occlusion. When the jaw and the molars are in normal alignment, the teeth may be crowded/rotated or missing. The normal position of the tongue is when it is resting against the palate, posing a balancing force on the teeth between tongue and cheek muscles. However, if the mandibular angle is not high, but there is attrition of the teeth and loss of VDO, there is a potential risk of sleep apnea.

Class II occlusion is known as retrognathia of the mandible. As the mandible is deficient, the maxilla will protrude over the mandible. There is the presence of an increase in overjet/overbite and inability to close the lips, with increased tension in the orbicularis oris, buccinator, and constrictor superior muscle. The ring of muscles mentioned above plays a crucial role in the physiology of breathing in human beings. These sequelae of events can lead to narrowing of the airway and decrease in posterior airway space and contribute to OSA in patients [73].

In Class III occlusion, the mandible is larger than the maxilla, which causes the anterior teeth to be edge to edge or present with an underbite leading to a concave profile. Most cases of skeletal discrepancy are due to insufficient growth of the maxilla or overgrowth of the mandible. Studies have demonstrated that maxillary or mandibular abnormalities change the volume of the oral cavity and affect the morphology of the upper airway [74]. Class III malocclusion patients with the craniofacial anomalies usually have constriction of the velopharynx and nasal cavity, nasal obstruction, or choanal stenosis, which is caused by the severe maxillary hypoplasia which may impact nasal breathing [75, 76].

## 6.7 Bicuspid Extractions and Maxillary Expansion

There has been significant controversy regarding the role of orthodontics potentially contributing or causing OSA, in both children and adults. The belief was that four-bicuspid extraction and retraction of the incisors would contribute by crowding the tongue and decreasing oropharyngeal airway. A study in 2010 showed that there was no statistical change in the upper airway volume between those patients who underwent either extraction or non-extraction [77]. A study was done in 2015, where 5585 medical and dental records of adults were reviewed by health partners of Minnesota. Half of the patients had four bicuspids missing and were assumed to have had orthodontic treatment earlier in life. The data analysis was controlled for age, gender, BMI, premolars missing or not missing, and the diagnosis of OSA confirmed by PSG [78]. This record review determined that 267 of those without missing bicuspids had received a diagnosis of OSA and 299 subjects with missing bicuspids had received a diagnosis of OSA. The prevalence of OSA was therefore not significantly different between the two groups [79].

The most common cause of OSA in children is enlarged tonsils and adenoids. Therefore, the primary treatment for children is adenotonsillectomy. Unfortunately, there is a large subset of children with residual OSA after surgery [80]. A deficiency in the maxilla and/or mandible can predispose children to SBD caused by nasal airflow deficiency and mouth breathing. Mouth breathing causes maladaptation of tongue position and oropharyngeal volume. Rapid maxillary expansion (RME) increases nasal volume, creates room for proper tongue posture, improves muscle tone, and allows nasal breathing. In a study, 14 children were chosen who had a malocclusion and OSA confirmed with a PSG. Ten children were treated with RME over a 12-month period. Two of the children had failed to expand. Of the other eight children, the apnea AHI decreased by the end of the treatment period and the

symptoms had resolved. Two years after the resolution of treatment, no significant changes in the AHI were found [81].

Perilli et al. demonstrated that a subgroup of OSA children with isolated maxillary narrowing initially treated with RME were stable at the 12-year follow-up, a long-term result of post-RME treatment for pediatric OSA. The maxillary base width and the distance of the pterygoid processes measured using CT imaging stayed stable. The clinical evaluations, including orthodontic and otolaryngologic examinations and questionnaire scores, were consistently normal over time, and PSG showed a good response with a decrease in AHI and long-term resolution of their SDB [82, 83]. Another study showed that a significant number of children who underwent bimaxillary expansion had worsening of their SDB [84]. Overweight children are mistreated by either therapeutic approach, because weight loss is also an important part of therapy, as fat deposition increases in the tongue due to weight gain (discussed in Sect. 6.3.5). This is why there must be a coordinated team of healthcare professionals. Several studies found that surgical maxillary expansion helps to reduce AHI in those with transverse deficiencies [85, 86]. These authors believe that if OSA cases are treated in the early developmental phase, we can potentially help develop patients skeletally in the dentofacial region when they are in mixed dentition, to possibly avoid extraction of permanent teeth and widen the dental arches to create more room for the tongue long term [2, 83]. In skeletal discrepancy cases, such as Class II or Class III, there is usually underdevelopment of the mandible or maxilla. If there is any underdevelopment skeletally, we believe that when teeth are extracted in order to close that space, the anterior teeth have to be retracted, thus resulting in reduction of space for the tongue. Furthermore, as children grow into adults, all of the hard and soft tissues continue to grow and develop including the tongue due to fat deposition, except the size and shape of the teeth [2].

## 6.8 Temporomandibular Disorders and OSA

The treatment of OSA with DSA has been associated with temporomandibular joint disorder (TMD) symptoms. The dental healthcare practitioner should have a good knowledge base of these conditions in order to advise the patient on whether they need TMD treatment and/or to proceed with OAT. If there are underlying TMD conditions, risks and benefits need to be discussed.

When OSA occurs, the body's automatic reflexes and response are to open up the airway by pushing the jaw forward. This repetitive movement puts pressure on the TMJ throughout the night which causes a lot of stress and tension in the jaw joint. Some TMD conditions encountered are capsulitis, myalgia, myofascial pain, arthralgia, disk disorders (disk displacement with and without reduction), and arthritis [87]. The examination of the TMJ will be discussed in detail in Chaps. 8 and 10 will discuss how to resolve acute TMD symptoms during DSA treatment.

Subjective sleep disturbance has been consistently reported in TMD patients [88, 89]. An emergent body of fact and information proposes that OSA is related to chronic pain disorders including TMD [90–92]. In a cohort study of adults without

TMD at baseline, OSA signs/symptoms were associated with increased incidence upon the first onset of TMD. Men and women with two or more signs/symptoms of OSA had 73% greater incidence of first-onset TMD, independently of age, gender, race/ethnicity, obesity, smoking history, and autonomic parameters. In a case-control study, chronic TMD was three times more frequent among adults with the likelihood of OSA, independently of these same factors [91].

TMD is a musculoskeletal disorder indicated by sustaining pain in the temporomandibular joint, in the periauricular region, and in the masticatory muscles. Current evidence of a relationship between OSA and TMD is constricted to certain findings within a clinical setting [91, 93]. The correlation of pain and sleep is bidirectional. Dubrovsky and colleagues used PSG studies to investigate sleep and respiratory parameters in women with TMD pain demonstrating that TMD cases with chronic myofascial pain have a mild degree of objective sleep disturbance and a mild increase in upper airway resistance during sleep, both of which appear to relate to acute levels of myofascial pain at night [94]. The use of the OAT may cause transient TMD symptoms when the appliance is first worn, but these manifestations disappear within a few days. If the manifestations become persistent, treatment of these symptoms should become the center of attention [87]. One cannot estimate the strength of the interrelationship or determine the temporal order of the interconnection between OSA and pain [91].

As the prevalence of TMD and OSA is high in the general population, many patients may complain of TMD pain during DSA. Cunali and colleagues evaluated the prevalence of pain with TMD in OSA patients who were referred for DSA, 52% of patients presented symptoms of TMD, and 75% of the patients presented chronic pain related to TMD, categorized as low-grade disability. The most common TMD diagnosis was myofascial pain with and without limited mouth opening and arthralgia (50%) [90].

SDB was reported to be six times higher in children with TMD pain upon awakening than children without SDB. Therefore, sleep bruxism may be implicated in development of TMD in children [95]. This relationship between SDB and TMD implies that children with TMD should be routinely checked for SDB, and those with a higher development of sleep problems should be considered for referral and comprehensive sleep study and/or evaluation [96].

## 6.9 Headaches

Due to the lack of evidence, previously there has not been enough studies to establish correlation between OSA and headaches [97]. However, there are numerous recent studies which have mixed conclusions about OSA and headaches being directly related. There are two major findings for sleep-related headaches distinguished by the International Classification of Headache Disorders, one is sleep apnea headache and the other is hypnic headache (HH).

Sleep apnea headache is a morning headache, usually bilateral, occurring more than 15 days/month, lasting less than 4 h, caused by OSA and resolving with successful OSA treatment [98]. HH is a rare disorder characterized by frequently recurring headache attacks starting during sleep, causing wakening and lasting from 15 min to 4 h, occurring at least 10 days per month for more than 3 months, without cranial autonomic symptoms and not attributed to other pathologies [98]. HH is more common in women (male/female ratio 1:1.5) and usually begins after the age of 50 years with pain usually bilateral and mild to moderate in intensity [99, 100].

Tension-type headache (TTH) is another headache known to be perpetuated with OSA [101]. Pain is a typically bilateral, pressing, or tightening feeling in quality and of mild to moderate intensity, lasting minutes to days, and does not worsen with routine physical activity. It usually is not associated with nausea, but photophobia or phonophobia may be present [98]. There is evidence of dysfunction of serum serotonin levels in patients with OSA. In a study conducted in 2015, 4759 patients who were diagnosed with OSA were tested for TTH. TTH were noticed in 10.2% of patients with OSA and 7.7% of patients without OSA. The study concluded that patients who have OSA also have higher chances of getting TTH [101]. In a polysomnographic (PSG) study, 50% of children with TTH had SDB versus the normal group where 2.4% of children with non-tension-type headaches [102].

The most commonly described sleep apnea headaches are the recurrent morning headaches found to be three times more prevalent upon awakening in heavy snorers and OSA patients [102, 103]. A retrospective study reported that out of 82 chronic headache patients with migraine, TTH, or both, 52 patients (63%) also had OSA. When the patients were treated with continuous positive airway pressure (CPAP) therapy, the headaches improved by 49% [104]. More than 70% of cluster headache patients report nocturnal attacks, often waking them from sleep [105]. Furthermore, in patients diagnosed with cluster headaches, oxygen desaturation below 89% during sleep preceded 8 out of 14 attacks among 10 patients, showing positive support for the relationship of cluster headache and OSA [106].

CPAP and other treatment modalities such as DSA therapy not only treat the OSA but have led to resolution and improvement in headaches from time to time. Treating OSA might not only improve headaches but also leads to decreased comorbidity [107]. Children with headaches complain about sleep quality and experience excessive daytime sleepiness [108]. It has well been observed that frequent headaches are associated with sleep bruxism, in both adults and children [102, 109]. In Fernandes et al. (2016), an association among patients with sleep bruxism has been shown between painful TMD and headache diagnoses. The magnitude of association was greater for chronic migraine, since 100% of the patients presented TMD and sleep bruxism, followed by episodic migraine (95.3%) and episodic tension-type headache (91.3%) [110].

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