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Obstructive sleep apnoea and the need for its introduction into dental curricula

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Abstract

Obstructive sleep apnoea (OSA) is a major health problem which causes blood oxygen desaturation that may initiate a cascade of events via inflammatory cytokines and adrenocorticotrophic hormone that may have impact upon quality of life and lead to potential life-threatening events. Even though OSA affects an increasing number of individuals, the role of dental practitioners in recognition, screening and management has not developed accordingly. The goal of this article was to provide updated information to dental practitioners on pathophysiology, consequences and treatment options of OSA with a focused discussion on oral appliance (OA) therapy, as this topic is not routinely included in current dental curricula of many dental schools. Additionally, we present a template dental curriculum for predoctoral and/or postdoctoral students in education regarding sleep disordered breathing.

Introduction

Sleep disordered breathing (SDB) has several presentations with one being obstructive sleep apnoea (OSA) (1). OSA involves episodes of both obstructive apnoea and hypopnoeic events (2). Arousal (micro-awakening) is defined as a brief awakening characterised with an abrupt shift in the electroencephalogram/electromyogram lasting longer than 3 s (3). The arousal may be spontaneous or a result of movement (such as periodic limb movements) or due to respiratory factors. The relationship between SDB and arousal is linear in that the greater the SDB severity, the more numerous are the arousals (arousal index >6 per hour may make people chronically sleepy) (4).

In the adult population, OSA may be due to collapse of the pharynx and intermittent partial (hypopnoea) or complete (apnoea) obstruction of the upper airway (5–7) (Fig. 1). Common characteristics of OSA include repetitive apnoea/hypopnoea, sleep fragmentation and excessive daytime sleepiness (7, 8). Various maxillofacial skeletal and soft tissue abnormalities (6, 7, 9, 10), endocrine disorders (11), smoking, obesity and genetics (12), alcohol consumption (13) and positional factors (8) have been suggested as the contributing components of OSA. Individuals often seek treatment for undiagnosed OSA due to snoring, which is often a major complaint originating from their sleep partner (14). Amongst those aforementioned characteristics, high body mass index (BMI) is most common (7–9), and due to the current obesity epidemic, both the prevalence and severity of SDB have been increasing (3%–9% females; 10%–17% males in ages 30–49 to 50–70 years of age) (15, 16). In contrast, the prevalence of OSA in paediatric patients has been reported from 0–1% to 4–5.7% (17, 18).

The gold standard for the diagnosis of OSA is overnight polysomnography (PSG), which includes synchronised observation and audio-visual recordings of both sleep and respiration (7, 19). Additionally, electroencephalogram (EEG), electrocardiogram (EKG), electrooculogram (EOG) and electromyography (EMG) along with respiration rate, tidal volume, and inspiration and expiration volumes are utilised in diagnosis of OSA (19–21). Epworth Sleepiness Scale (ESS) and respiratory disturbance index (RDI) [-average number of apnoeas, hypopnoeas and respiratory effort-related arousals (RERAs) per hour of sleep] are also used to assess daytime sleepiness and OSA, respectively (22). The severity of OSA is determined with the apnoea/hypopnoea index (AHI), which is the number of

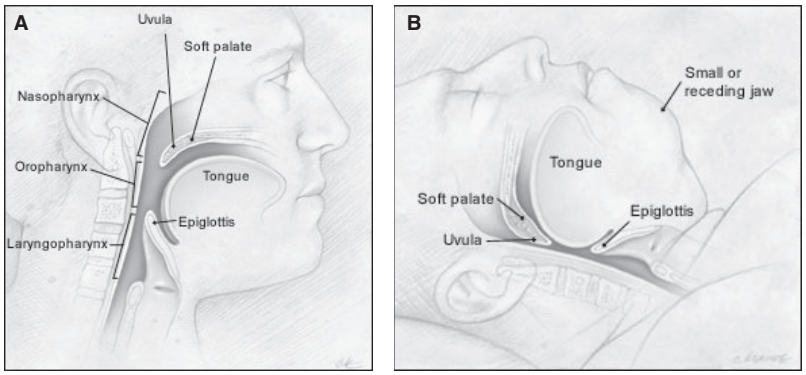

Fig. 1. (A) Normal airway: The soft palate, uvula and the tongue are normal in length and size; the tongue is angled forward. The upper airway at the level of the nasopharynx, oropharynx and hypopharynx is normal in size and contour, (B) Abnormal airway during sleep: Multiple sites of obstruction are present; elongated and enlarged soft palate blocks the posterior airway at the level of the nasopharynx and oropharynx. Due to a retruding jaw, the enlarged tongue is pushed posteriorly to impinge on the hypopharyngeal space (http://www.aafp.org/afp/1999/1115/p2279.html; accessed on 11,11.2015).

respiratory events per hour of sleep. An AHI score of five or greater is considered diagnostic for OSA (7, 8, 10, 14); it is considered mild when AHI >5–14/h, moderate when AHI = 15–29/h, and severe when AHI >30/h (8, 14). In children, AHI scores for OSA differ from those of adults: 1–5 score refers to mild, 6–10 moderate, and greater than 10 AHI score signifies severe OSA (18).

The mechanisms of OSA remain to be fully identified (23), but the loss of pharyngeal dilator muscle tone appears responsible for the narrowing of the airway (8, 14, 24). Additionally, posterior positioning of the mandible or constriction of pharyngeal walls by fat pads in obese individuals (BMI $\geq$30 kg/m^2) (20) may result in neurophysiological activation (8, 25). This activation leads to abrupt mid-sleep awakening in the form of arousal, resulting in sleep fragmentation and daytime sleepiness (24, 25).

Systemic consequences of OSA

In untreated OSA, oxygen desaturation is often reported with an elevated AHI index (6, 8) resulting in neurophysiological reactivation which is primarily responsible for the development of adverse outcomes (7, 26). The adverse outcomes include an increase in systemic inflammatory response (26–31), plasma adrenocorticotrophic hormone (ACTH), urinary norepinephrine and decreased growth hormone concentrations (28). The production and release of these inflammatory mediators and hormonal factors are closely related to life-threatening outcomes such as hypertension, cerebrovascular accident (CVA), congestive heart failure and atrial fibrillation, increased risk of motor vehicle accidents, excessive daytime sleepiness, and impaired quality of life including social life (32–37).

One of the serious disorders linked to OSA is type 2 diabetes mellitus (DM) (28, 38–40), and a bidirectional correlation between OSA and type 2 DM has been suggested (28, 38, 41–43).

Similarly, associations between OSA and hypertension, coronary artery diseases and CVA have been investigated (44, 45). The mechanisms responsible for these interactions involve disturbances in sleep quantity or quality which may lead to oxidative stress and increased plasma norepinephrine levels and subsequent sympathetic nerve activity upon the vasculature. These processes may contribute to the development or progression of hypertension, increase in heart rate, and rapid rise and fall in cerebral blood flow (46, 47). Additionally, the chronic presence of inflammatory cytokines in the peripheral circulation of patients with OSA may activate immune and inflammatory pathways, leading to subsequent neuroinflammatory/neurodegenerative diseases such as chronic fatigue syndrome (48), impaired learning and memory (49), cognition (50) and Alzheimer's disease (51).

The National Asthma Education and Prevention Program Expert Panel includes OSA as one of the contributors to uncontrolled asthma (52, 53), because of the effects of cytokines and superoxide radicals related to hypoxia (54). Individuals with OSA are 3.6 times more likely to have uncontrolled asthma (53), and moderate-to-severe asthma is observed in 34% of patients with OSA who had an AHI >5 events/h (55).

An association between OSA and cancer has been the subject of interest in recent years (56, 57). Accelerated tumour angiogenesis and elevated oxidative stress observed in nocturnal hypoxia may damage DNA and RNA and potentially promote tumorigenesis (58–60). Also, increased cancer-related mortality correlating with the severity of OSA has been noted (61).

Management options for OSA

Recommendations for treatment of OSA begins with weight loss, tobacco and alcohol cessation, and includes continuous positive airway pressure (CPAP) therapy, medical therapy, surgical intervention to modify the upper airway, electrical upper airway muscle stimulation and oral appliances (OAs) (6, 11).

CPAP therapy

CPAP, suggested as the gold standard in the management for OSA (11, 62), is a device with a motor unit that pushes filtered air through a facial mask (involving the nose, mouth or both)

at a positive pressure (8). The pressure inside the mask remains positive throughout the respiratory cycle, with the goal of maintaining a patent upper airway whilst the patient self ventilates (8). A major obstacle for CPAP is poor patient adherence (7, 63, 64). Nasal expiratory positive airway pressure (EPAP) uses the patients' own breathing in order to produce positive airway pressure to prevent obstructed breathing (65), although the efficacy of EPAP is not documented (66–68).

Medical therapy

The medications that have been advocated to manage mild-to-moderate OSA include progestogens, acetazolamide (impacts respiratory muscle strength and endurance) (69), theophyllines (increases ventilator drive) (63), antidepressants (stimulates central and inhibits peripheral serotonin receptors) (70) and serotonin reuptake inhibitors (elevates respiratory arousal threshold) (71). Some studies report significant (70) or partial success (71) with medical therapy of OSA, but the outcome of medical treatment for OSA remains inconclusive (24, 63).

Surgery

Mild-to-moderate patients with OSA who are CPAP resistant may be appropriate for surgical interventions such as uvuloplasty, bipolar radiofrequency surgery of the tongue base (7, 14, 72, 73) and correction of nasal pathologies (74). Complicated upper airway surgeries such as bimaxillary orthognathic procedures (17), anterior inferior mandibular sagittal osteotomy, genioplasty, maxillomandibular advancement osteotomies (10, 72) and uvulopalatopharyngoplasty (7, 17) are less frequently offered due to lower than 50% significant improvement in AHI in some patients (7) and potentially serious post-operative complications (14). The indications and efficacy of surgical treatment of OSA for patients who fail to respond to CPAP therapy are yet to be determined (6).

Electrical stimulation of the hypoglossal nerve to trigger the genioglossus muscle (75) and the tensor veli palatini muscle has been suggested to improve upper airway patency (76). To date, several means of electrical stimulation of the muscles to enlarge the pharyngeal air space have been reported with varying degrees of effectiveness (77–80).

Oral appliances

OAs are recommended for patients with: mild-to-moderate OSA, with severe OSA but intolerant of CPAP therapy, and who are not surgical candidates (14, 62, 81–84). These appliances are designed to enhance the patency of the posterior pharynx (11, 18, 85) and have been shown to decrease AHI and elevate oxygen saturation (84). OAs may be considered prior to surgical procedures as they are a reversible intervention.

A thorough history and clinical evaluation of the patient including evaluation of temporomandibular joint (TMJ) and masticatory muscle function and review of a completed sleep study (provided by a medical practitioner) are needed prior to determining the appropriate OA (18, 81, 83, 86). There are several types of OAs based upon their mechanism of action. OAs function by either advancing the mandible (mandibular repositioning appliance/mandibular advancement device), by sustaining the tongue in an anterior position (tongue-retaining device – TRD) especially in edentulous patients (11, 14, 18, 82, 86) or by supporting the soft palate (soft palate lifters) (11, 18, 82). Typical predictors for success with OAs include less severe disease, younger age, female sex, lower body mass index, smaller neck circumference and more positional (supine-dependent) OSA. Radiographic predictors of success using cephalometric parameters include a short palate, large retro-palatal airway space, narrow anterior posterior position of mandible (small SNB angle) and
higher anterior posterior position of the maxilla (large SNA angle) (21, 87). OAs have less effect on reducing AHI than CPAP, but do improve OSA when compared to the patients who receive no treatment (62, 81, 82, 84). Temporary adverse effects associated with OAs are mostly observed during the initial stages of therapy (14, 88) and include excessive or diminished salivation, mucosal dryness, transient discomfort/pain in teeth, gingiva, masticatory muscles, TMJ and headache (14, 21, 62, 88). The potential long-term side effects depend upon OA type, design and duration of use, and may include potential mesial migration of lower dentition and distal migration of upper dentition (89–91).

The contraindications for OA use may include severe periodontal disease, presence of temporomandibular disorders (TMD), severe gag reflex and incomplete dentition (less than 6 teeth on the maxillary/mandibular arches). In edentulous individuals, advanced bone loss and/or poor denture retention may limit effects of OAs. Severe hypoxia, growing children, protrusive range of the mandible <7 mm, mouth opening restricted to 30 mm or less, unmotivated patients and presence of severe comorbidities may impact the effect of OAs. (14, 90).

OAs may be commercially purchased or custom made by dental practitioners. Whilst a custom OA is an elaborate procedure associated with time and expense (62), the efficacy of over-the-counter OAs is limited when compared to the custom-made design (14, 62, 82), and data of their effectiveness are limited.

Patient adherence with OAs ranges from 51% to 88%, depending on the type used with adherence being highest for a mandibular advancement device (MAD) as compared to the other appliances (89, 91).

The designs of OAs vary with respect to their allowance for lateral jaw movement, the coupling mechanisms between maxillary and mandibular components, the ability to adjust the degree of advancement, vertical opening and the occlusal coverage (14, 21, 62, 82).

Recently, an oral negative pressure device that uses a mouthpiece connected to a suction mechanism to create an intra-oral vacuum to pull the tongue anteriorly has been presented (92). However, efficacy of this approach has not been established (63).

Types of oral appliances

The Dental Division of Food and Drug Administration (FDA) requires OAs to have a label informing patients about contraindications and potential side effects (86). The American

Academy of Sleep Medicine (AASM), Canadian Academy of Dental Sleep Medicine and Canadian Sleep Society suggested that OAs be administered by qualified and trained dental personnel who are experienced in the care of oral health, the TMJ, dental occlusion and related oral structures, as OAs may worsen an existing TMD problem and/or cause dental misalignment and discomfort (81, 83).

Following a sleep study performed by a qualified medical practitioner, a thorough history and orofacial examination including dental structures are required (86). If the patient is a suitable candidate, then, depending on the type of OA chosen, the dental practitioner will obtain dental impressions of the maxillary and mandibular arches, custom bite registration in centric occlusion and determine the appropriate advanced position (21). Even though the types and shapes of OAs vary widely, a design that provides appropriate mandibular advancement or tongue protrusion without excessive mouth opening and with stability of tooth position is applicable (84).

Amongst OAs, the MAD is most commonly preferred. It functions by bringing the mandible forward, thereby increasing the airway volume (85). It can be either fixed (predetermined advancement), adjustable or either a one-piece (mono-bloc) or a two-piece device (bi-bloc) (14, 82, 85). Increased TMD is reported with one-piece MAD as compared to other devices (85). The adjustable MAD incorporates a mechanism that allows progressive advancement of the mandible after initial fabrication until the optimal mandibular position is achieved. Typically, a MAD is advanced to 75% of maximum mandibular protrusion (62); however, a greater protrusion may be necessary especially in severe cases (62), possibly resulting in more risk for TMD and occlusal problems (82, 85, 88). As the degree of vertical opening has not been shown to impact efficacy (82), it is suggested to keep the vertical to a minimum in order to increase patient adherence (62). A standard method for the patient-specific protrusion and MAD design is yet to be defined (82, 85), and therefore, adjustable device design is frequently chosen.

The TRD is suggested for patients with an excessively large tongue, when the use of a MAD is limited due to edentulous ridges, the presence of periodontal problems or lack of an adequate number of teeth for device retention (18, 82). Soft palate lifters are removable maxillary appliances which cover the mucosal surfaces of the hard–soft palatal area and the lingual sides of the teeth, so they can support and lift the soft palate (18).

The subjective therapeutic efficacy of OAs can be assessed by the dental practitioner during patient management, and necessary measures can be initiated for improved outcomes (Fig. 2) (20).

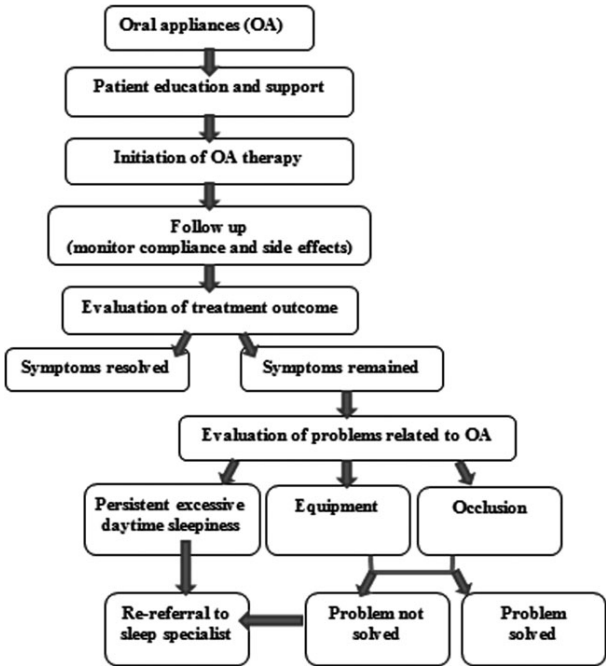

Fig. 2. The steps of initiation, follow-up and management of OA treatment in patients with OSA.

Dental practitioners' role in OSA diagnosis and management

Many patients may not recognise or appreciate the role of dentistry in OSA. Therefore, patients may fail to report their signs and symptoms associated with this disorder or knowledge of their diagnosis to their dental practitioner (93). However, dental practitioners have the opportunity to inquire about the possibility of OSA and to examine the entrance to the nasopharyngeal area in addition to routine investigation of oro-dental hard and soft tissues (8, 21, 94). During the initial interview, dental practitioners may enquire about signs and symptoms of OSA (Table 1). Dental examination should include soft and hard tissue and masticatory muscle and TMJ assessment, evaluation of occlusion and determination of bruxism (20, 86). Any potential dental pathosis diagnosed must be treated (81, 83). In certain cases, cephalometric analysis may be performed by the trained professional (20, 81). Following this, as per the guidelines of the Adult OSA Task Force of The American Academy of Sleep Medicine (20), the patients with suspected OSA should be referred to specialised centres for further evaluation and diagnosis (14, 95). When needed, dental practitioners may seek the assistance of other professional colleagues for assessment of daytime sleepiness by the Epworth Sleepiness Scale (ESS), (96), risk for OSA using the STOP-BANG questionnaire (97) or the Berlin questionnaire (98), for possible depressive symptoms by the Beck Depression Inventory (BDI) (99), and subjective sleep quality with the Pittsburgh Sleep Quality Index (PSQI) (100).

Orofacial and dental features associated with the presence of OSA are as follows: increased neck circumference (≥16 inch in females, ≥17 inch in males), body mass index ≥30 kg/m^2 (20), Mallampati score of 3–4 (9, 20), retrognathic mandible, macroglossia (20), longer face height, scalloped tongue, tonsillar hypertrophy and decreased lateral peritonsillar space, elongated uvula, deep hard palate, nasal anomalies and excessive overjet (20, 101). If a dental practitioner is not comfortable in following the appropriate information gathering procedures and examination, referral to a knowledgeable colleague may serve as a vital step in both assessing and management of OSA (14, 15).

TABLE 1. Questions and examination findings to be considered in the screening for OSA (8)

Questions
Do you snore?
Does snoring occur in a particular position?
Have you ever had upper airway surgery?
Has anyone ever witnessed you stop breathing or have choking episodes in your sleep?
For how long do you sleep?
Do you feel refreshed on waking?
Do you experience daytime sleepiness, poor concentration or poor memory?
Have you ever had any road traffic incidents or near misses when driving?
Do you have to use the toilet in the night?
Do you have headaches in the early morning?
Do you have loss of libido?
Examination
Body mass index, neck circumference
Blood pressure
Thyroid assessment
Assessment for nasal obstruction and retrognathia
Oral examination for tonsillar enlargement and oropharyngeal crowding
Additional assessment should be made regarding medication use, comorbidities, alcohol and tobacco consumption

Once a diagnosis is provided by a medical practitioner and if appropriate (mild-to-moderate OSA or CPAP intolerant), the patient may be managed by a knowledgeable dental practitioner who has specific education and training regarding OSA (81, 83). The dental practitioner can then observe the patient at regular intervals (recall at 6, 12 months; yearly thereafter) to assess patient adherence (≥4 h use for ≥70% the nights), monitor OA efficacy and monitor occurrence of side effects and modify the OA as needed (20, 81, 95) (Table 2). Thus, dental practitioners should be a part of the multidisciplinary/interdisciplinary team for patients with OSA (14), which includes medical sleep specialists, referring physicians, nurses, respiratory therapists and sleep technologists (86).

The selection and fabrication of custom-made OAs should be made only by the dental practitioner who considers the patient-specific needs, the OSA severity, and availability and affordability of management alternatives (14, 86). In refractory patients, these patients should be referred to the appropriate health practitioner for a different approach in order to provide improved general health and quality of life (14, 18, 102). This is especially important for patients with systemic conditions such as cardiovascular, respiratory, cognitive and endocrine (type 2 diabetes mellitus) disorders (38, 40). Clearly, collaboration between medical and dental practitioners is vital for screening, diagnosis, management and follow-up of OSA (14, 18, 81, 93, 95).

TABLE 2. The role of the dental practitioner in OSA (13, 94)

Assessment of patients with OSA before intervention (once diagnosis provided by medical practitioner)
Informing patients about management modalities
Management of present oral problems
Counselling on oral hygiene and OA maintenance
Recalling every 4-6 weeks to control patient adherence and treatment efficacy
Management of OA-related complications/adverse effects
Providing communication between sleep specialists about the outcome of OA treatment
Determination of the need to change the OSA treatment
Establishment of protocols on OA titration, personnel training, official procedures (i.e. insurance coverage)
Participation in multidisciplinary/interdisciplinary meetings
Organising routine patient follow-up care

Educational need

Dental practitioners may be reluctant to engage in management of OSA due to concerns of litigation and malpractice (15), as they report a lack of adequate training in the screening for OSA and its subsequent management in providing OAs (86, 93, 94). The training that is currently available is mainly derived from courses offered by OA manufacturers, scientific meetings and information garnered from the literature (94, 95). It is reported that in 49 USA dental schools, the mean total predoctoral sleep curriculum time is 2.96 h (94). Additionally, only prosthodontics, oral medicine and orofacial pain postdoctoral programs include sleep-related disorders in their curricula (94). In Middle Eastern universities, the total average hours dedicated to teaching sleep medicine was 1.2 h and this was less than half of the average of that in North American dental schools (103). A recent database search resulted in only two papers related to the education of OSA in dental curricula; however, these did not involve European institutions.

Bian et al. (104) reported that because of the scant curricular time, resources, and teaching facilities, dental schools lacked the appropriate training regarding sleep disorders and over half of the dental practitioners failed to recognise OSA in their patients. However, dental practitioners who have receive some kind of education regarding OSA report managing patients with OSA more frequently (104).

Considering that dental practitioners will have patients with OSA either knowingly or unknowingly, they should have appropriate background knowledge about OSA and related morbidities and be competent to screen for SBDs and OSA. Unfortunately, current dental curricula have failed to respond to this health trend and, at best, provide an introduction regarding OSA usually without an appropriate dental approach for these patients (15, 94). It is prudent for dental institutions to be at the forefront of educating in this subject matter. Inclusion of specific courses and/or lectures related to sleep disorders and specifically to OAs and the role of the dental practitioner is warranted with emphasis directed towards recognition, screening and management of OSA as part of the health care team.

The previous sections of this paper highlight the background information required to be provided in educating predoctoral and/or postdoctoral students regarding OSA. Table 3 provides a template for topics to be considered as part of dental curricula regarding OSA and outlines suggestions for preparation of course material for this purpose.

TABLE 3. Topics to be considered in dental curricula towards the education of OSA

Proposed topics to be included into a dental curriculum	Contents
1. Epidemiology of sleep issues in society	Prevalence of sleep issues in adults/children, gender differences and association with other medical disorders
2. Normal sleep	The components and periods of normal sleep pattern
3. Prevalence of sleep disorders	The number of children and adult patients with sleep disorders and future trends in global prevalence of sleep-related problems
4. Anatomy and physiology of the upper airway	The hard and soft tissues of the related region and their roles in normal physiological functioning
5. Terminology	The definitions of sleep-related disorders, obstructive sleep apnoea, hypopnoea, apnoea/hypopnoea index (AHI), respiratory effort-related arousals, polysomnography, respiratory disturbance index
6. Signs and symptoms of obstructive sleep apnoea (OSA)	Patient report and clinical presentation of sleep-related problems, that is daytime sleepiness, excessive tiredness, sleep fragmentation
7. Physiologic effects of OSA	The impact of molecular changes such as low oxygen saturation via production and release of inflammatory and hormonal factors; disease states such as autoimmune disorders, cognitive disabilities, hypertension, cerebrovascular accident, congestive heart failure and atrial fibrillation, poor cognitive and functional status, dementia, Alzheimer's disease, bronchial asthma; and tumorigenesis due to altered physiologic processes associated with OSA
8. Risk factors/predisposing factors for OSA	Age, obesity, sleeping position, tobacco and alcohol use, type 2 diabetes mellitus, asthma, increased neck circumference (≥16 inch in females, ≥17 inch in males), body mass index ≥30 kg/m^2, Mallampati score of 3-4, retrognathic mandible, macroglossia, longer face height, scalloped tongue, tonsillar hypertrophy and decreased lateral peritonsillar space, elongated uvula, deep hard palate, nasal anomalies and exaggerated overjet
9. Medical assessment for OSA	Health history and symptom history related to snoring and OSA, head and neck examination and PSG evaluation and diagnosis(es)
10. Treatment options for snoring and OSA	Behavioural options and avoidance of risk factors to include weight loss, tobacco and alcohol cessation, over-the-counter products for snoring, positive airway pressure therapies, oral negative pressure devices, medical therapy, surgical intervention to modify the upper airway, electrical upper airway muscle stimulation and oral appliances
11. Dental practitioners role in snoring and OSA	
a. Patient evaluation	A thorough history and clinical extra-oral–intra-oral evaluation of the patient including the masticatory muscles and temporomandibular joints (TMJ), evaluation of occlusion and determination of bruxism, screening/assessment and/or review (as provided by professional colleagues) of sleep disturbance with appropriate means, that is Epworth Sleepiness Scale, Berlin Questionnaire, STOP-BANG questionnaire, Pittsburgh Sleep Quality Index, Multiple Sleep Latency Test, Beck Depression Inventory and dental models for reviewing occlusal schema
b. Imaging	Bitewing, periapical, cephalometric and panoramic radiographs, orthognathic analyses, MRI, CBCT (use of images when required)
c. Oral appliance therapy (OA)	
i. Indications for use	Patients with mild-to-moderate OSA, smaller neck circumference, more positional (supine-dependent) OSA, cephalometric parameters such as short palate, large retro-palatal airway space, narrow anterior posterior position of mandible (small SNB angle) and higher anterior posterior position of the maxilla
ii. Classification of OA	Mandibular advancement devices (fixed or predetermined advancement), adjustable or either a one-piece or a two-piece device, tongue-retaining devices
iii. Mechanism of action	Bringing the mandible forward, thereby increasing the airway volume, holding the tongue in a protruded position to enlarge the pharyngeal airway space
Iv. Evidence for use of OA	Literature review following an evidence-based approach
V. Management with OA	Protocol for OA use to include appliance design variations, selection criteria, potential candidates of OA therapy, contraindications for use
Vi. Potential side effects and complications	Minor side effects and more significant side effects, prevalence of side effects/complications and understanding/managing side effects to include excessive/diminished salivation, mucosal dryness, transient discomfort/pain in teeth, gingiva, masticatory muscles and TMJ, headache, potential mesial migration of lower dentition and distal migration of upper dentition
Vii. Multidisciplinary/ interdisciplinary approach	The role of dental practitioners and those of the sleep specialists, referring physicians, nurses, respiratory therapists and sleep technologists in management of OSA

The authors of this paper suggest that emphasis should be placed on an interprofessional model of collaborative practice with various health practitioners, as previously proposed (15). Therefore, dental institutions need to have access to qualified faculty who are knowledgeable in providing didactic and clinical material on this subject, in addition to having adequate clinical laboratories, courses and hands-on experiences regarding OSA, otherwise preparing future dental practitioners will be incomplete (94). This approach will facilitate best management of patients with OSA (86).

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