Obstructive sleep apnea for the dental hygienist: Overview and parameters for interprofessional practice

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ABSTRACT

Objective: This literature review is intended to provide an overview of obstructive sleep apnea (OSA) to assist dental hygienists with developing an OSA screening protocol. This paper will also provide a review of legal and ethical considerations related to the diagnosis, treatment, referrals, and the circle of care for OSA clients. **Methods:** OSA is associated with concomitant conditions such as obesity, hypertension, and cardiovascular disease and thus requires a multidisciplinary client management strategy. A multidisciplinary search was conducted using databases that include: Academic Search Premier, Bioline International, Biological Sciences@ Scholars Portal, Biomed Central, Medline, PubMed Central, Scifinder, Scholars Portal and Scholars Portal E-Journals. In addition, physicians, dentists, and an oral myofunctional therapist provided clinical perspectives. **Discussion:** OSA is a potentially fatal disorder that affects 15–24% of the adult population; however 70–80% of cases are undiagnosed. Clinical signs and symptoms include loud snoring, witnessed apneas, and excessive daytime sleepiness. OSA has a myriad of cardiovascular, metabolic and neurocognitive consequences. Untreated OSA may lead to cognitive impairment, sexual dysfunction, and a poorer quality of life. Many of the comorbid conditions and risk factors associated with OSA can be identified through a comprehensive dental hygiene client assessment. **Conclusions:** OSA can have serious detrimental effects on client's overall health and quality of life. Dental hygienists are primary healthcare providers who can screen clients for OSA through comprehensive health histories and extra/intra oral assessments. Dental hygienists can facilitate a medical diagnosis by recognizing OSA signs, symptoms and risk factors, using OSA screening tools, and by providing the client with medical referrals. The dental hygienist may support clients with OSA by providing oral health education, and nutritional counselling.

RÉSUMÉ

Objet : Cette revue de la littérature présente à la profession de l'hygiène dentaire un aperçu général de l'apnée obstructive du sommeil (AOS) afin d'aider les hygiénistes dentaires à élaborer un protocole de dépistage de l'AOS. Cet article offre aussi une revue des aspects juridiques et éthiques du diagnostique, du traitement, de la soumission de cas et du cercle des soins pour les patients atteints de l'AOS. **Méthodes :** L'AOS est associée à des états concomitants, telles l'obésité, l'hypertension et la maladie cardiovasculaire; elle requiert ainsi une stratégie de gestion multidisciplinaire de la clientèle. Une recherche multidisciplinaire a été effectuée dans les bases de données suivantes : *Academic Search Premier*, *Bioline International*, Sciences biologiques dans *Scholars Portal*, *Biomed Central*, *Medline*, *PubMed Central*, *Scifinder*, *Scholars Portal* et les journaux électroniques de *Scholars Portal*. Et outre, des médecins, des dentistes et une thérapeute buccale myofonctionnelle ont apporté des perspectives cliniques. **Discussion :** L'AOS peut être un désordre funeste chez 15 à 24 % de la population adulte; toutefois 70 à 80 % des cas ne sont pas diagnostiqués. Les signes et les symptômes cliniques comprennent les ronflements bruyants, les apnées témoins et la somnolence diurne excessive. L'apnée obstructive du sommeil a une myriade de conséquences cardiovasculaires, métaboliques et neurocognitives. L'AOS non traitée peut mener à une déficience cognitive, à un dysfonctionnement sexuel et à un appauvrissement de la qualité de vie. Plusieurs des problèmes de comorbidités et des facteurs de risque associés à l'AOS peuvent être identifiés grâce à une évaluation complète de l'hygiène dentaire du patient. **Conclusions :** L'AOS peut affecter gravement la santé globale et la qualité de vie du client. Les hygiénistes dentaires sont les principaux fournisseurs de soins de santé qui examinent les patients en scrutant tous leurs antécédents de santé et en faisant des évaluations intra et extra buccales. Les hygiénistes dentaires peuvent faciliter le diagnostic médical en reconnaissant les signes, les symptômes et les facteurs de risque de l'AOS avec des outils d'examen de l'AOS et en orientant le patient vers le médecin. L'hygiéniste dentaire peut soutenir sa clientèle atteinte de l'AOS, par l'information sur la santé buccale et le conseil diététique.

Key words: obstructive sleep apnea, apnea–hypopnea Index (AHI), hypertension, continuous positive airway pressure (CPAP), sleep apnea questionnaires, obesity, gastroesophageal reflux disease (GERD), bruxism, oral appliances, systemic inflammation, cariovascular disease, atherosclerosis

OBJECTIVE AND BACKGROUND

This article is intended to provide an overview and summarize available knowledge about obstructive sleep apnea (OSA) to assist dental hygienists with developing an OSA screening protocol.

OSA is a potentially fatal disorder that is characterized by repetitive, complete or partial obstruction of the upper airway during sleep causing cessation in airflow.[1–4] Obstruction of the airway during sleep leads systemic oxygen deprivation, leading to potentially deleterious effects

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on organs such as the heart and brain. OSA occurs in both adults and children. OSA was reported to be prevalent in 9% of women and 24% of men in 1993.[5] Studies indicate a rise in OSA prevalence and report OSA in 15–25% of middle aged and older adults,[6–9] that may be associated with the rise in North American obesity rates.[10–13] In addition to the increase in OSA prevalence, 70–90% of OSA cases are undiagnosed.[5,8,14,15] Undiagnosed OSA may be attributed to the paucity in awareness of OSA in the medical and oral health professions and failure to recognize common OSA symptoms.[15–17]

It was estimated in 1990 that in the US, healthcare costs associated with diagnosed OSA amount to $275 million per year.[18] OSA sequelae include cardiovascular disease and other comorbidities; consequently undiagnosed and untreated OSA cases may engender an even more significant economic strain on the healthcare system. Superfluous healthcare costs associated with undiagnosed/untreated OSA are related to treating OSA sequelae and comorbidities without addressing OSA as the primary etiologic condition.

The two salient types of sleep apnea are central sleep apnea (CSA) and OSA. OSA is defined as "a cessation of airflow for at least 10 seconds…the event is obstructive if during apnea there is an effort to breathe".[4] Thus, OSA results when the airway is obstructed or collapses during sleep resulting in respiratory effort to restore breathing. CSA is defined as a "cessation in airflow for at least 10 seconds…the event is central during apnea if there is no effort to breathe".[4] CSA results from the brain's imbalance of respiratory control during sleep that leads to a decrease or absence in the effort to breathe. This paper will focus on OSA, as the discussion of CSA is beyond the scope of this paper. It is also important to distinguish between OSA and obstructive sleep apnea syndrome (OSAS). OSA is limited to the medical condition as defined above and may not include symptoms.[5] OSAS is the syndrome associated with OSA and is accompanied by symptoms such as excessive daytime sleepiness and snoring.[5] Clients with OSA may not necessarily exhibit symptoms of OSA.[5]

Research has identified OSA as a chronic and multifaceted disease, and when undiagnosed and untreated can result in an array of concurrent health disorders. As a result, this literature review will also provide an overview of the comorbidites associated with OSA. This article will also provide an overview of legal and ethical considerations related to the diagnosis, treatment, referral, and the circle of care for OSA clients.

METHODS

OSA is associated with numerous concomitant conditions; therefore, the literature search was conducted in multidisciplinary databases including Academic Search Premier, Bioline International, Biological Sciences@ Scholars Portal, Biomed Central, Medline, PubMed Central, Scifinder, Scholars Portal, and Scholars Portal E-Journals. Physicians, dentists and an oral myofunctional therapist provided clinical perspectives. Medical and dental disciplines searched included respirology, sleep medicine, anesthesiology, otorhinolaryngology, cardiology, gastroenterology, immunology, endocrinology, pediatric medicine, pediatric dentistry, general dentistry, prosthodontics, orthodontics, oral surgery, and orofacial myology. Key words used in the search include: obstructive sleep apnea, hypopnea, apnea-hypopnea Index (AHI), polysomnogram, hypertension, continuous positive airway pressure (CPAP), sleep apnea questionnaires, obesity, metabolic syndrome, mouth breathing, orofacial myology, asthma, gastroesophageal reflux disease (GERD), oral appliances, bruxism, adenotonsillar hypertrophy, systemic inflammation, cariovascular disease, and atherosclerosis. Inclusion criteria involved full text articles, randomized clinical trials, literature reviews, and systematic reviews. Non peer reviewed and unpublished papers were excluded in the search for this paper. The literature search involved an international search for articles in English. Based on the inclusion and exclusion criteria, 251 articles were included in this review.

Interviews with primary healthcare providers—a sleep medicine respirologist, an otolaryngologist and dentist, two general practice dentists who were trained to deliver oral appliance therapy for the treatment of OSA, a certified oral myofunctional therapist, and an orthodontist—were conducted to gain insight into the clinical management and treatment of clients with OSA (Fitzpatrick M. Telephone interview. 10 February 2011; Wade P. Personal interview. 10 January 2011; Priemer L. Personal interview. 30 September 2010; Shnall J. Personal interview. 21 January 2011; Moeller J. Telephone interview. 10 February 2011; Tovilo K. Personal interview. 14 February 2011). These health professionals were selected on their clinical and academic experience within their respective professions and knowledge of OSA.

DISCUSSION

The following sections of this article will discuss the pathophysiology of OSA, craniofacial clinical presentation of OSA, risk factors for OSA in adults, incidence, and risk factors for OSA in children, comorbid conditions that are associated with obstructive sleep apnea (OSA), diagnosis of OSA, treatment modalities, and the dental hygienist's role in screening, referring and supporting OSA clients.

OSA pathophyiology

The upper airway, which is also referred to as the pharyngeal airway, is the soft tissue region bordered by the nasopharynx, the epiglottis, the maxillomandibular complex (anteriorly) and the spinal column (posteriorly)[19–21] as depicted in figure 1. OSA arises from narrowing of the airway that results in pharyngeal airway collapse and occlusion. Obstruction of the upper airway can involve one or more components of the pharyngeal airway anatomy including the base of the tongue, the soft palate, uvula, hypertrophic adenoids and tonsils, and the nose.[22,23] The primary site of airway obstruction associated with OSA is considered to be in the oropharyngeal–hypopharyngeal area.[1,23] Thus, if nasal obstruction is involved in OSA, resolution of nasal obstruction may not necessarily resolve OSA if the primary site of obstruction is the oropharyngeal–hypopharyngeal area.[23,24]

The pharyngeal airway soft tissue is dilated during

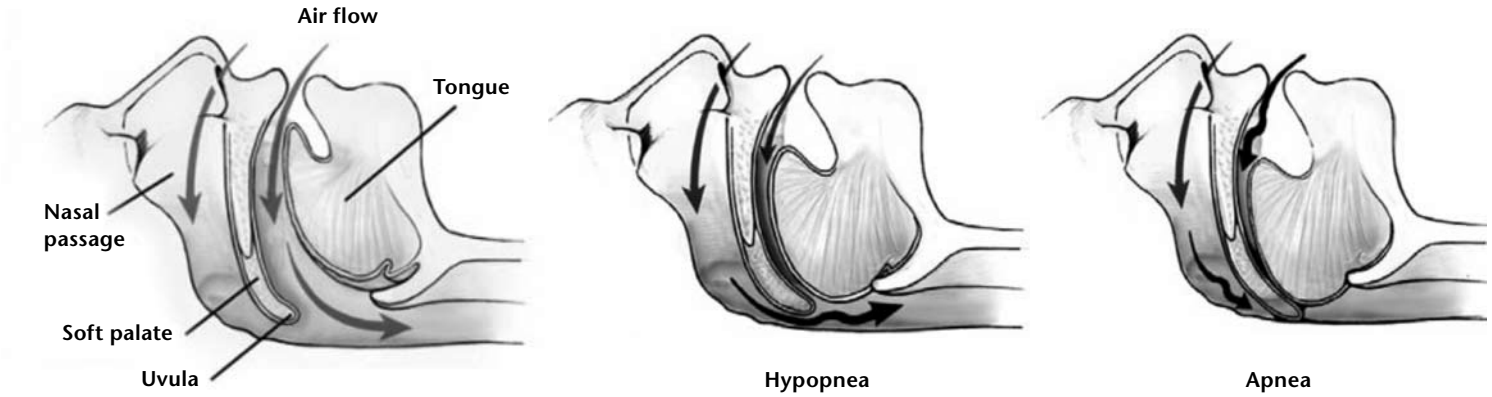


Figure 1. Partial and complete airway obstruction resulting in hypopnea and apnea, respectively. Reprinted from Hahn PY, Somers VK. Sleep apnea and hypertension. In: Lip GYH, Hall JE, eds. *Comprehensive Hypertension.* St. Louis, MO: Mosby; 2007:201–07. ©Elsevier, 2007. Reproduced with permission.

wakefulness but loses its tone during sleep.[19,21] OSA occurs as a sequel to the negative pressure of inspiration which draws soft tissue anatomy against the pharyngeal walls, narrowing or obstructing the airway,[21,23] resulting intermittent interruption of ventilation. The occurrence and severity of hypoventilation during sleep is measured by the apnea–hypopnea index (AHI). An apnea is defined as a cessation of airflow resulting from a complete blockage of the upper airway lasting for at least 10 seconds[4,8] as shown in Table 1. However, AHIs have been reported to last up to two minutes (Fitzpatrick M. Telephone interview. 10 February 2011). Hypopneas are defined as a partial obstruction of the upper airway resulting in a 20–50% decrease in airflow, and 3–4% oxygen desaturation.[25,26] Both apneas and hypopneas result in arterial hypoxemia and hypercapnia.[27–36] Arterial hypoxemia is considered a reduction in arterial blood oxygen and hypercapnia is considered an increase in blood carbon dioxide.[25,26] Recurrent reduction of systemic oxygen and elevated carbon dioxide may lead to metabolic disturbances[37–42] such as in obesity,[10–12] cardiac disease,[2] stroke and death.[7,8]

The AHI measures the number of apneas and hypopneas per hour during sleep. OSA is characterized by a minimum of five apneas and/or hypopneas during sleep.[8] The severity of OSAS is determined by the frequency of hypoventilation. The AHI quantifies the severity of OSA by calculating the number of apneas and hypopneas divided by the hours slept.[25] For adults, mild OSAS is scored at AHI> 5–15 hour, moderate OSAS is an AHI>15–30, and severe OSAS is an AHI >30.[26,29] For children, mild OSA is an AHI= 1–5, moderate is an AHI>=6–10, and severe is considered at an AHI of >10.[30] See Table 1.

OSA symptoms

It is important to distinguish between OSA and OSAS. OSA clients may present as asymptomatic. When a client with OSA exhibits symptoms, they are considered to have OSAS.[5] There is a greater percentage of the population which has OSA but does not exhibit symptoms of the syndrome.[5] Primary symptoms associated with OSAS are loud chronic snoring,[43–48] witnessed apneas and excessive daytime sleepiness.[5,43] Primary symptoms are those that are highly associated with OSA.[48] The presence of primary symptoms would warrant a medical referral for an OSA assessment.[48] A combination of common OSA symptoms in addition to other OSA risk factors would also warrant a referral for an OSA medical assessment.

Loud chronic snoring arises from the vibration of soft tissue obstruction during inspiration.[23,25] Snoring may be followed with periods of silence that may indicate the presences of an apneic event. Clients with OSA may awaken from apenic and hypopneic events in an inadvertent strenuous effort to restore respiration.[25] Awakening may be synchronous with sounds of snorting, gasping, and choking.[43,46] Apneic and hypopneic episodes can occur isochronously during sleep. Sleep fragmentation from intermittent arousals often results in excessive daytime sleepiness[43,47–54] that can lead to increased work and traffic accidents[55–62] and a poorer quality of life.[49,63] Quality of life is defined as "the functional effect of an illness and its consequent therapy upon the patient, as perceived by the patient".[63] Effects of OSA on quality of life may involve limitations in "physical and occupational function, psychological functions, social interaction, and somatic sensation".[63]

Other common symptoms of OSAS related to decreased systemic oxygen and sleep disturbances include nocturia,[64–67] morning headaches,[64–68] xerostomia,[37,66] impairment of cognitive function,[69–76] depression,[77–80] sexual dysfunction,[81,82] irritability,[66] and decreased concentration.[13,66] These OSA symptoms are related to the

Table 1. Apnea–hypopnea index (AHI): OSA severity for adults and children.

OSA severity	Mild	Moderate	Severe
Adults AHI score	>=5–15	15–30	>30
Children AHI score	1–5	6–10	>10

Table 2. Symptoms of obstructive sleep apnea syndrome (OSAS).

Primary symptoms	Common symptoms
• Excessive daytime sleepiness • Loud chronic snoring • Waking events accompanied by snorting, gasping choking sounds witnessed by another person (witnessed apneas)	• Morning headaches • Xerostomia • Depression • Memory impairment • Decreased concentration • Irritability • Nocturia • Sexual dysfunction/ impotence • Diminished quality of life

sequelae arising from systemic oxygen desaturation and sleep arousals.

OSA craniofacial presentations

Craniofacial features of OSA include intraoral and extraoral findings. Extraoral features identified as risk factors associated with adult OSA include a neck circumference >40 cm (15.75 inches).[83–86] Men who have a 43.18 cm (17 inches) neck circumference (or greater) are at higher risk for OSA.[43] A neck circumference of 40.64 cm (16 inches) or greater for women increases OSA risk.[43] Excess adipose tissue in the neck region increases the risk of upper airway soft tissue obstruction.

Other craniofacial characteristics of clients with OSA (as seen in Table 3) include a small retrognathic mandible,[87–89] and inferior displacement of the hyoid,[90,91] increased anterior face height,[92–96] a steep mandibular plane angle,[92,93,97] decrease in nasal patency,[92–98] and Class II malocclusion.[93,99–101] These craniofacial presentations may involve mandibular retrognathia that can lead to the posterior displacement of the oropharyngeal soft tissues during sleep. Posterior displacement of soft tissues can heighten the risk of OSA pharyngeal occlusion.

Table 3. Craniofacial–oral risk factors associated with OSA.

Extraoral–craniofacial presentations of clients with OSA	Common intraoral features of clients with OSA

• Small, retrognathic mandible
• Large neck circumference
  ○ Men >43.18 cm (17 inches) diameter
  ○ Women >40.64 cm (16 inches) diameter
• Increased anterior face height (long looking face)
• Steep mandibular plane angle
• Inferior displacement of hyoid
• Decreased nasal passage size
	• Large, low, flat, posteriorly positioned tongue
	• High vaulted narrow palate
	• Elongated soft palate and uvula
	• Hypertrophic tonsils/ adenoids
	• Class II malocclusion, crossbite
	• Reduced intermaxillary space
	• Edentulous (especially sleeping without dentures)
	• Dental erosion (associated with GERD)
	• Attrition associated with bruxism

OSA intraoral and extraoral clinical presentations and risk factors

Prominent intraoral features include a posteriorly inclined, low lying broad tongue,[101–106] a reduction in intermaxillary space,[103,104] and a narrow high vaulted palate.[93–99] The hard palate is the floor of the nasal cavity thus; a narrow hard palate can lead to a decreased nasal space,[107,108] and increased nasal resistance.[98,99,108] A normal vaulted palate allows sufficient intermaxillary space to accommodate the tongue. Thus, a narrow high palate/ maxillary constriction may not accommodate the tongue, resulting in a low and flat tongue posture and crowding of the pharyngeal airway.[99] Other intraoral risk factors for OSA include an elongated soft palate,[105,109] hypertrophic uvula,[66,105] and enlarged tonsils/adenoids.[101,102,105] These intraoral features may increase the probability of upper airway obstruction.

Other common intraoral features of clients with OSA may include dental attrition and erosion which relate to common concomitant OSA conditions such as GERD,[110–114,] and bruxism.[115–118] Bruxism leads to muscle activation in the upper airway—tongue, suprahyoid and masseter—and is often observed at the end of an apneic event, restoring tone to the airway, alleviating the obstruction, and terminating the apneic event.[115] Edentulism is also an OSA risk factor.[119–122] The absence of posterior teeth may lead to loss in vertical dimension and collapse of pharyngeal soft tissues,[119,121,122] resulting in an increase OSA risk. Accordingly, the severity of AHI has been shown to be higher in clients with OSA who sleep without dentures.[121,122]

Adult OSA risk factors

Independent risk factors for OSA include sex,[48,123] age,[48] and hypertension.[124,125] Men between the ages of 30 and 50 have twice or thrice higher incidence of OSA than women of the same age.[46,47,126] The link between OSA and hypertension is thought to be a causal relationship and OSA is considered an independent risk factor of hypertension.[125,126] Studies have also shown that both hypertension and OSA exacerbate each other,[127] and that about 50 per cent of clients with OSA develop systemic hypertension.[124] The relationship between OSA and hypertension may be attributed to an increase in sympathetic nerve activity and increased peripheral vascular resistance that results from a decrease in oxygen saturation during sleep.[128] The increase in peripheral resistance leads to vasoconstriction that may result in a 25 per cent raise in systemic blood pressure accompanied by further blood pressure surges at each apneic episode.[129]

Obesity is defined by a body mass index (BMI) greater than 30,[12] and is a recognized risk factor for OSA.[130–157] Obesity can lead to an increase in craniofacial adipose tissue that can cause upper airway obstruction.[43,84–89] However, it is important to distinguish that *not all adults with OSA are obese*.[47] Other associated risk factors include pregnancy,[139–144] tobacco,[47,84] alcohol use,[45,47] sedative use,[47,142] and genetics.[145,146] Weight gain and edema may increase OSA risk and pre-eclampsia during pregnancy.[139–144] Tobacco use is believed to cause inflammation of the pharyngeal soft tissues that may increase the risk for upper

airway occlusion during sleep and exacerbate OSA related cardiovascular risk. Alcohol and sedatives may decrease pharyngeal airway soft tissue tonicity, exacerbating the risk of pharyngeal occlusion.[37,139] The genetic risk for OSA is related to genetic biochemical markers and shared familial craniofacial architecture.[47,87,105] Clients who have a familial history of OSA have 2 to 4 times greater risk for OSA.[37,105] These risk factors for OSA should be considered as part of an overall OSA risk assessment during client care.

Concomitant conditions associated with OSA

OSA can be the progenitor and aggregator for a myriad of concomitant conditions. Concurring conditions associated with OSA include numerous cardiovascular diseases including arrhythmias,[147,148] atrial fibrillation,[149,150] stroke,[147,151] myocardial infarction,[152,153] heart failure,[153,155] systemic hypertension,[120,156–160] atherosclerosis,[161–167] pulmonary hypertension,[135,159] and systemic inflammation.[161–167] Other concurrent conditions associated with OSA include diabetes mellitus,[168,169] asthma,[170–172] allergic rhinitis,[23,173] fibromyalgia,[174] metabolic syndrome.[37–40] The mechanisms of interaction between concomitant conditions and OSA are complex, and discussion of all of these mechanisms is beyond the scope of this article. This article will provide a brief discussion on the mechanisms of OSA, systemic inflammation, atherosclerosis, and cardiovascular disease.

OSA, systemic inflammation, and cardiovascular disease

OSA is considered a risk factor for cardiovascular disease by the American Heart Association and the American College of Cardiology.[26] Systemic inflammation is suggested to be a sequela to OSA and an important factor in the pathogenesis of cardiovascular disease.[175,176] Accordingly, OSA clients are five times more likely to die from cardiovascular related causes.[176] Systemic inflammation that results from OSA is thought to exacerbate cardiovascular disorders such as hypertension, atherosclerosis and heart disease.[175–179] OSA related oxygen deprivation may lead to immuno-inflammatory alterations resulting in vascular changes and atherosclerotic remodelling. Studies suggest a positive correlation between atherosclerotic plaque volume and OSA.[176] OSA related atherosclerosis may also be exacerbated by hypertension related vascular hypertrophy and OSA related hormonometabolic changes such as insulin resistance and dyslipidemia.[176]

OSA also results in an increase of atherosclerotic pro-inflammatory mediators such as C-reactive protein (CRP). CRP is a salient pro-inflammatory mediator associated with OSA.[178,181–183] Elevation of CRP has been detected in clients with OSA independent of comorbidities such as obesity.[164] Chronic periodontal disease is also associated with an increase in systemic inflammatory mediators, atherosclerosis, and coronary heart disease.[184–190] CRP is also the inflammatory mediator associated with chronic periodontal disease.[187,190] A recent study has suggested that there may be an association between OSA and chronic periodontal disease.[191] The link between OSA and periodontal disease is suggested to be related to the chronic inflammatory nature of both conditions.[191] However, more research is needed to explore the nature of this association.

Pediatric OSA, epidemiology, and risk factors

OSA occurs in approximately 2 per cent of children.[192] Pediatric OSA is most often associated with hypertrophy of adenoid and tonisllar tissue[174,192–210] that may lead to pharyngeal occlusion. However, other common conditions and risk factors associated with pediatric OSA include loud chronic snoring,[211] obesity,[193,194,206] allergic rhinitis,[172,212,213] nasoseptal obstruction,[205,212,214] micrognathia,[32,205,212] retrognathia,[32,68] and genetics.[205] Relative mechanisms of pediatric OSA risk factors are similar to those discussed for adults.

Craniofacial–oral features of children with OSA are also similar to those of adults. Clinical presentation includes a low lying tongue position,[197] a high, narrow, vaulted palate and maxillary constriction,[215,216] increased soft palate volume,[216] retrognathia,[32,68,103] decreased nasal patency,[68,108] malocclusion, posterior crossbite,[217–219] anterior open bite,[211] and increased anterior facial height.[68,103] These risk factors are associated with an increased risk of pharyngeal obstruction during sleep.

Nasal obstruction and hypertrophic adenoids and tonsils may also lead to mouth breathing.[89,172] The typical facial architecture associated with mouth breathing is termed “adenoid face” or “long face” syndrome.[96,107,231] The adenoid face is characterized by an incompetent lip seal, a narrow upper dental arch, increased anterior face height, a steep mandibular plane angle, and a retrognathic mandible.[93] There are commonalities between the typical orofacial architecture of chronic mouth breathing and craniofacial risk factors for clients with OSA. Common orofacial findings of chronic mouth breathing include open lip posture,[94] Class II malocclusion,[107,218] crossbite,[107,218] an increased anterior facial height,[95,218] a hypertrophic soft palate,[108] a narrow, high, vaulted palate,[107,108,218] a low and flat tongue,[197,220] and a steep mandibular occlusal plane.[96,107,108] Craniofacial features of mouth breathing clients suggest the posterior inclination of the mandible and pharyngeal tissues which may be related to OSA risk. Although, chronic mouth breathing is not causally related to OSA,[104,108] the presentation of craniofacial architecture associated with mouth breathing may indicate latent craniofacial risk for OSA.[195,108]

Children with OSA often will experience detrimental neurobehavioural consequences,[201,205,212] a poorer quality of life,[205] and can negatively affect academic performance.[205,212] Some studies have shown that OSA in children may have a higher prevalence of attention deficit disorder (ADD).[32,69,76] Negative cognitive consequences of pediatric OSA may be attributed to oxygen deprivation during sleep and sleep arousals.[32,69] However, more research is needed to define the relative mechanisms between OSA and ADD.

Screening and medical diagnosis

In 2009, the American Academy of Sleep Medicine recommended, “Questions regarding OSA should be incorporated into routine health evaluations. Suspicion

Table 4. STOP-BANG questionnaire and the Epworth Sleepiness Scale.

STOP-BANG questionnaire	Epworth Sleepiness Scale
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 (231). **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 >30? **A:** Age >50? **N:** Neck circumference >40 cm? **G:** Gender male? **STOP-BANG score** _____	A score of 10 or more is considered sleepy. If you score 10 or more on this test, you may not be obtaining adequate sleep. These issues should be discussed with your physician. Use the following scale to choose the most appropriate number for each situation: 0 = would never doze or sleep 1 = slight chance of dozing or sleeping 2 = moderate chance of dozing or sleeping 3 = high chance of dozing or sleeping **Situation** **Chance of dozing or sleeping** Sitting and reading _____ Watching TV _____ Sitting inactive in a public place _____ Being a passenger in a motor vehicle for an hour or more _____ Lying down in the afternoon _____ Sitting and talking to someone _____ Sitting quietly after lunch (no alcohol) _____ Stopped for a few minutes in traffic while driving _____ **Total score** _____

of OSA should trigger a comprehensive sleep evaluation. The diagnostic strategy includes a sleep oriented history and physical exam, objective testing, and education of the client."[29] A medical diagnosis of OSA can be facilitated by routine screening initiatives by any primary healthcare provider. Screening for OSA may involve identifying comorbidities associated with OSA in the health history, adding related questions as part of the health history, identification of craniofacial–oral risk factors and clinical presentations associated with OSA, and use of screening questionnaires. A comprehensive health history assessment will reveal salient concomitant conditions associated with OSA such as obesity, hypertension, diabetes, depression, and GERD. Questions in the health history related to OSA can include the investigation of diagnosed OSA, and primary symptoms of OSA including snoring, witnessed apneas, and excessive daytime sleepiness.

There are various soft tissue obstruction grading systems such as the Friedman, Fujita and Mallampati scoring methods used to screen for OSA risk during the intraoral assessment.[101,102,220–222] When using such grading systems, it is important to recognize that these grading systems are not used to diagnose OSA, nor do these grading systems indicate absence or severity of OSA.[223]

Common screening questionnaires used to assess OSA risk include the STOP-BANG questionnaire,[224] and the Epworth sleepiness scale.[225] The STOP-BANG questionnaire is an acronym which is used to identify salient risk factors of OSA, those being S-snoring, T–tired during daytime, O–observed apneas, P–high blood pressure, B–BMI >30, A–age (middle age), N–neck circumference >40 cm (15.75 inches), G–gender (male). A simplified version is the STOP questionnaire that utilizes the first four questions for screening for OSA.[48] The Epworth sleepiness scale is commonly used to determine the extent of daytime sleepiness related to OSA as shown in Table 4. Questionnaires, health history findings, and clinical assessments should be used collectively to determine the need for a referral to the family physician for an assessment of OSA.

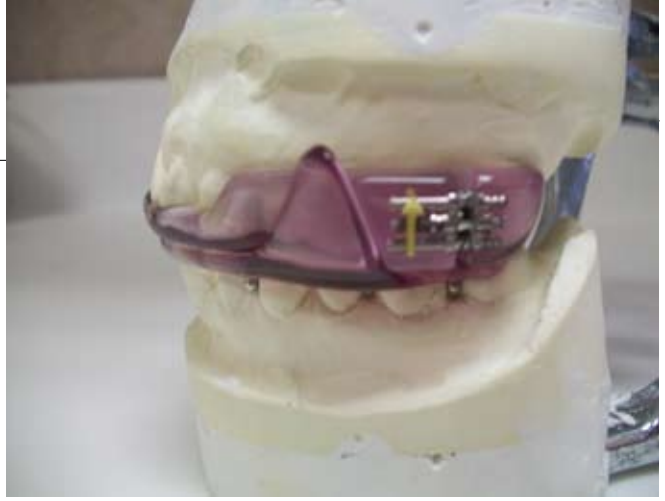
Medical diagnosis

The in-laboratory polysomnogram (PSG) is considered the gold standard for diagnosing OSA.[1,25,29] The PSG records the number of apneic and hypopneic events during the sleep cycle. The PSG also records other sleep mechanisms such as sleep latency, oxygen saturation and sleep position.[1,25,69] There are a number of other diagnostic methods such as portable home monitoring units and anecdotal questionnaires.[192,226–230] However, portable home monitoring units are recommended by the American Academy of Sleep Medicine[29] to be used as a pretesting evaluation to determine the need for a PSG. Thus, portable home monitoring units are not considered the primary diagnostic method for OSA.[29]

Medical treatment and management of clients with OSA

Continuous positive airway pressure (CPAP) is considered the first line treatment in the treatment of OSA that opens up the airway during sleep.[1,231] CPAP armamentarium usually consists of a face mask that covers both the nose and mouth. The mask is secured to a ventilating device as shown in Figure 2. The effectiveness of CPAP therapy in the treatment of OSA has been documented by numerous studies.[155,156,232,233] Although the CPAP therapy is the first line treatment for OSA, it has inadequate levels of client compliance,[234–236] and its non compliance is related to the cumbersome nature of the apparatus and poor fit of the nasal mask.[234–236] Compliance with the CPAP therapy ranges from 25.5–58%, with women being less

▲ **Figure 2.** Continuous Positive Airway Pressure (CPAP) is the first line treatment for OSA. Image of model wearing a full mask CPAP, courtesy of Resmed Company and the CPAP Clinic Company, Toronto. Reproduced with permission.



▲ **Figure 3.** Somnodent oral appliance for the treatment of OSA.

compliant than men.[215,216] Secondary medical treatment of OSA includes oral appliances, surgical management and behavioural modification.[29] Surgical modalities to treat OSA include adenotonsillectomy, uvulopalatopharyngealplasty, maxillomandibular advancement.[237,238] Studies have shown that surgical removal of adenoids and tonsils results in 75–100% resolution of OSA in children.[192]

Oral appliance therapy for the treatment of OSA

Oral appliance therapy is considered by the American Academy of Sleep Medicine to be a secondary line of treatment of OSA.[29,231] This oral appliance therapy is administered by a specially trained dentist using a medical device.[239,240] Oral appliances used to treat OSA involve the placement of a custom dual arch appliance. An OSA oral appliance is designed to advance the mandible anteriorly as shown in Figure 3. Mandibular advancement also protracts the base of the tongue forward, opening up the throat and airway. Oral appliances are generally indicated for mild to moderate OSA.[231,241] Client compliance with oral appliances is considered greater than client compliance with CPAP therapy.[241] If oral appliance therapy is implemented, a second follow up PSG is required to determine the efficacy of the oral appliance on the OSA condition.[29,231]

Behavioural modification and other conjunctional therapies for OSA

Behavioural modification to treat OSA focuses on managing risk factors and comorbidities related to OSA. Behavioural modification involves tobacco cessation for clients who use tobacco products, weight loss through diet and exercise,[230,242–244] positional therapy,[29,230] and abstinence from alcohol and sedative use prior to sleep.[29,243] Weight loss is usually difficult for clients with OSA[29] which may be related to excessive daytime sleepiness. Hence, clients with OSA may find it difficult to muster the energy to engage in physical activity. In addition, studies have suggested that clients with OSA and obesity have increased leptin resistance.[244] Leptin regulates appetite satiety,[135] and leptin resistance is associated with an unregulated appetite. Accordingly, OSA clients who have leptin resistance and excessive daytime sleepiness usually struggle with weight loss.[244]

OSA is often exacerbated when sleeping in the supine position. Positional therapy involves the use of a positioning device such as a pillow, backpack, or tennis ball that adheres to the client's back during sleep. These devices are used to prevent the client from assuming a supine position during sleep.[29,230] However, not all OSA relates to sleep position.[29] A diagnosis of a PSG will determine if positional therapy is indicated.[29]

A novel association has been made between orofacial myofunctional therapy and improvement in AHI scores.[245,246] Although orofacial myofunctional therapists in North America do not treat OSA, a recent Brazilian study has shown that orofacial myofunctional therapy techniques that use orofacial exercises can reduce the severity of AHI in OSA clients by increasing the tone of the soft tissues of the pharyngeal airway.[245,246] However, more research is needed to establish its efficacy.

Screening and supporting clients with OSA

In Canada, dental hygiene is a self regulated profession in most provinces. Dental hygiene standards and scope of practice will vary in each province, thus the parameters of supporting and referring clients with OSA may also vary in each province. Dental hygienists should be familiar with their regulatory body standards of practice within their respective provinces. The authors of this article reside in Ontario, thus the following discussion will be from an Ontario perspective. The College of Dental Hygienist of Ontario (CDHO) offers a professional practice advisory called the "Knowledge Network".[247] The CDHO Knowledge Network provides access to all Ontario registered dental hygienists as well as guest access. The CDHO knowledge network advisory on OSA suggests, "as part of their role in preventive healthcare, dental hygienists may identify in a patient/client what appear to be possible early warning signals of obstructive sleep apnea; in that event they should promptly arrange or advise referral of the patient/client to the family physician".[247] Thus, dental hygienists have a recognized seminal role in screening and facilitating the assessment of OSA. Dental hygienists can screen for OSA by identifying comorbid conditions associated with OSA, recognizing craniofacial and oral risk factors for OSA, using OSA related questionnaires, and referring to the family physician for a medical consultation for OSA. The dental hygienist can also help support and educate clients with OSA by discussing such behaviour modification

strategies as tobacco cessation for clients who use tobacco products, by providing nutritional counselling, and by educating the client about the oral–systemic link.

Parameters for interprofessional practice

If a client is suspected of having OSA, the client should be referred to his or her family physician for a sleep apnea risk assessment. The family physician will determine the need for a consultation with a specialist in sleep medicine to determine if PSG is indicated.[248] PSG is typically conducted in an overnight sleep laboratory, and results are diagnosed by the specialist physician. Sleep medicine physicians are typically respirologists/pulmonologists. Once a diagnosis of OSA has been made, the specialist will determine if CPAP therapy is required.[29] If CPAP therapy is declined, or the client is non adherent, the physician will determine whether alternative treatment for OSA is suitable.[29] If an oral appliance is indicated for the treatment of OSA, the physician and dentist will consult with one another to initiate oral appliance therapy.[249]

Clients with OSA and excessive daytime sleepiness are at higher risk for traffic accidents.[59,62] In Ontario, physicians are required to report clients whose medical conditions are considered "dangerous…to operate a motor vehicle"[250] to the Ministry of Transportation of Ontario (MTO). Criteria for physician reporting to the MTO include the presence of uncontrolled sleep apnea,[250] and/or if the client's medical condition impairs their ability to drive safely. The driver's licence can also be reinstated if the client is no longer considered a risk to road safety.[250] Disclosure of this contingency is the sole responsibility of the physician.[250] Dental hygienists should explore their provincial regulations in regards to physician reporting in their respective provinces.

If a client has had a previous sleep study and has been diagnosed with OSA but untreated, the client should also be referred back to his or her family physician to determine the OSA status[29,248] prior to oral appliance therapy or any other treatment modality.[249] In 2009, the American Academy of Sleep Medicine posited, "the presence or absence and severity of the OSA must be determined before initiating treatment in order to identify those clients at risk of developing the complications of sleep apnea, guide selection of appropriate treatment, and to provide a baseline to establish the effectiveness of subsequent treatment".[29] Once a treatment for OSA has been implemented, a second PSG is required to determine the efficacy of the treatment.[29,248]

Clients who request treatment for snoring must undergo a physician consultation prior to treatment consideration.[29,231,249] The Royal College of Dental Surgeons of Ontario postulates, "snoring may be symptomatic of a serious and sometimes life-threatening medical condition called obstructive sleep apnea (OSA). Before oral appliance therapy is considered, it is essential that the presence or absence of OSA be determined by means of a medically supervised sleep test."[249] Snoring may be attributed to various etiologies. A differential diagnosis by a physician must be made prior to any treatment of snoring.[29,231,249]

CONCLUSION

OSA is a life threatening condition that often remains undiagnosed. In 1992, the national commission on sleep disorders announced "an urgent need for physicians, nurses, all healthcare professionals to be able to identify and refer or treat clients with sleep disorders."[251] Dental hygienists have a significant role in the screening of clients with OSA and in facilitating a medical diagnosis through an interprofessional referral.

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