IAOMT BIOLOGICAL DENTAL HYGIENE ACCREDITATION--
Checklist for Completing Unit 6:
Sleep-Disordered Breathing, Myofunctional Therapy, and Ankyloglossia

CONTENT FOR UNIT 6: SLEEP-DISORDERED BREATHING, MYOFUNCTIONAL THERAPY, AND ANKYLOGLOSSIA

☐ Read the “Obstructive sleep apnea for the dental hygienist: Overview and parameters for interprofessional practice” review by An and Ranson.
   Click here to go to pages 3-18.

☐ Read the “Association of obstructive sleep apnea with hypertension: A systematic review and meta-analysis” by Hou et al.
   Click here to go to pages 19-28.

☐ View the “Pediatric Sleep-Disordered Breathing” presentation by Rosellini at https://iaomt.org/airways-video-activity.
   Click here to go to page 29 for more information about this video.

☐ Read the “Myofunctional therapy: a novel treatment of pediatric sleep-disordered breathing” article by Moeller, Coceani, and Gelb.
   Click here to go to pages 30-38.

☐ View the “Tongue Tie and Surgery” video by Hornsby at https://www.youtube.com/watch?v=3TozTlenWEI.
   Click here to go to page 39 for more information about this video.

☐ Read the “Management of Infants Presenting with Ankyloglossia” IAOMT Scientific Review by Abramczyk.
   Click here to go to pages 40-50.

☐ Read the “Supplementary Resources for Sleep-Disordered Breathing, Myofunctional Therapy, and Ankyloglossia.”
   Click here to go to pages 51-52.

CONTINUED ON NEXT PAGE…
TEST FOR UNIT 6: SLEEP-DISORDERED BREATHING, MYOFUNCTIONAL THERAPY, AND ANKYLOGLOSSIA

☐ Take the Post-test for Unit 6 at https://www.cvent.com/d/mhq1j5.
  Click here to go to page 53 for more information about this test.

☐ You’re now ready to proceed to Unit 7!
  Click here to access Unit 7.
Obstructive sleep apnea for the dental hygienist: Overview and parameters for interprofessional practice

Soo-Lyun An, DipDH; Catherine Ranson, DipDH, BHA

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, Scilinder, 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.

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, cardiovascular 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...
on organs such as the heart. OSA occurs in both adults and children. OSA was reported to be prevalent in 9% of women and 24% of men in 1993. Studies indicate a rise in OSA prevalence and report OSA in 15–25% of middle aged and older adults that may be associated with the rise in North American obesity rates. In addition to the increase in OSA prevalence, 70–90% of OSA cases are undiagnosed. 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.

It was estimated in 1990 that in the US, healthcare costs associated with diagnosed OSA amount to $275 million per year. 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/unfreated 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” 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”. 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. OSAS is the syndrome associated with OSA and is accompanied by symptoms such as excessive daytime sleepiness and snoring. Clients with OSA may not necessarily exhibit symptoms of OSA.

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 comorbidities 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, anesthesia, 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, cardiovascular 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 OSA, diagnosis of OSA, treatment modalities, and the dental hygienist’s role in screening, referring and supporting OSA clients.

**OSA Pathophysiology**

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) 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. The primary site of airway obstruction associated with OSA is considered to be in the oropharyngeal-hypopharyngeal area. 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.

The pharyngeal airway soft tissue is dilated during...
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, resulting in intermittent interruption of ventilation. The occurrence and severity of hypoventilation during sleep is measured by the apnea–hypopnea index (AHI).

**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. There is a greater percentage of the population which has OSA but does not exhibit symptoms of the syndrome.

Primary symptoms associated with OSAS are loud chronic snoring, witnessed apneas and excessive daytime sleepiness. Primary symptoms are those that are highly associated with OSA. The presence of primary symptoms would warrant a medical referral for an OSA assessment. 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. Snoring may be followed with periods of silence that may indicate the presence of an apneic event. Clients with OSA may awaken from apenic and hypopneic events in an inadvertent strenuous effort to restore respiration. Awakening may be synchronous with sounds of snorting, gasping, and choking. Apneic and hypopneic episodes can occur isochronously during sleep. Sleep fragmentation from intermittent arousals often results in excessive daytime sleepiness that can lead to increased work and traffic accidents and a poorer quality of life. Quality of life is defined as “the functional effect of an illness and its consequent therapy upon the patient, as perceived by the patient.” Effects of OSA on quality of life may involve limitations in “physical and occupational function, psychological functions, social interaction, and somatic sensation.”

Other common symptoms of OSAS related to decreased systemic oxygen and sleep disturbances include nocturia, morning headaches, xerostomia, impairment of cognitive function, sexual dysfunction, irritability, and decreased concentration. These OSA symptoms are related to the sequelae arising from systemic oxygen desaturation and sleep arousals.
Table 2. Symptoms of obstructive sleep apnea syndrome (OSAS).

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

OSA craniofacial presentations

Craniofacial features of OSA include intraoral and extra-oral findings. Extraoral features identified as risk factors associated with adult OSA include a neck circumference >40 cm (15.75 inches). Men who have a 43.18 cm (17 inches) neck circumference (or greater) are at higher risk for OSA. A neck circumference of 40.64 cm (16 inches) or greater for women increases OSA risk. 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-retroglossal mandible, and inferior displacement of the hyoid. Increased anterior face height, a steep mandibular plane angle, decrease in nasal patency, and Class II malocclusion. 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.

<table>
<thead>
<tr>
<th>Extraoral–craniofacial presentations of clients with OSA</th>
<th>Common intraoral features of clients with OSA</th>
</tr>
</thead>
<tbody>
<tr>
<td>• Small, retroglossal mandible</td>
<td>• Large, low, flat, posteriorly positioned tongue</td>
</tr>
<tr>
<td>• Large neck circumference</td>
<td>• High vaulted narrow palate</td>
</tr>
<tr>
<td>❍ Men &gt;43.18 cm (17 inches) diameter</td>
<td>• Elongated soft palate and uvula</td>
</tr>
<tr>
<td>❍ Women &gt;40.64 cm (16 inches) diameter</td>
<td>• Hypertrophic tonsils/adenoids</td>
</tr>
<tr>
<td>• Increased anterior face height (long looking face)</td>
<td>• Class II malocclusion, crossbite</td>
</tr>
<tr>
<td>• Steep mandibular plane angle</td>
<td>• Reduced intermaxillary space</td>
</tr>
<tr>
<td>• Inferior displacement of hyoid</td>
<td>• Edentulous (especially sleeping without dentures)</td>
</tr>
<tr>
<td>• Decreased nasal passage size</td>
<td>• Dental erosion (associated with GERD)</td>
</tr>
<tr>
<td></td>
<td>• Attrition associated with bruxism</td>
</tr>
</tbody>
</table>

OSA intraoral and extraoral clinical presentations and risk factors

Prominent intraoral features include a posteriorly inclined, low lying broad tongue, a reduction in intermaxillary space, and a narrow high vaulted palate. The hard palate is the floor of the nasal cavity thus; a narrow hard palate can lead to a decreased nasal space, and increased nasal resistance. 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. Other intraoral risk factors for OSA include an elongated soft palate, hypertrophic uvula, and enlarged tonsils/adenoids. 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, and bruxism. Bruxism leads to muscle activation in the upper airway—tongue, suprahoid 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. Edentulism is also an OSA risk factor. The absence of posterior teeth may lead to loss in vertical dimension and collapse of pharyngeal soft tissues, 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.

Adult OSA risk factors

Independent risk factors for OSA include sex, age, and hypertension. Men between the ages of 30 and 50 have twice or thrice higher incidence of OSA than women of the same age. The link between OSA and hypertension is thought to be a causal relationship and OSA is considered an independent risk factor of hypertension. Studies have also shown that both hypertension and OSA exacerbate each other, and that about 50 per cent of clients with OSA develop systemic hypertension. 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. 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.

Obesity is defined by a body mass index (BMI) greater than 30, and is a recognized risk factor for OSA. Obesity can lead to an increase in craniofacial adipose tissue that can cause upper airway obstruction. However, it is important to distinguish that not all adults with OSA are obese. Other associated risk factors include pregnancy, tobacco use, alcohol use, and sedative use, and genetics. Weight gain and edema may increase OSA risk and pre-eclampsia during pregnancy. 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. The genetic risk for OSA is related to genetic biochemical markers and shared familial craniofacial architecture. Clients who have a familial history of OSA have a 2 to 4 times greater risk for OSA. 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, atrial fibrillation, stroke, myocardial infarction, heart failure, systemic hypertension, atherosclerosis, pulmonary hypertension, and systemic inflammation. Other concurrent conditions associated with OSA include diabetes mellitus, asthma, allergic rhinitis, fibromyalgia, metabolic syndrome, and chronic snoring. Elevation of C-reactive protein (CRP) has been detected in OSA clients with an increase in systemic inflammatory mediators, such as CRP. CRP is a salient pro-inflammatory mediator associated with chronic inflammatory nature of both conditions. 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. Pediatric OSA is most often associated with hypertrophy of adenoid and tonsillar tissue that may lead to pharyngeal occlusion. However, other common conditions and risk factors associated with pediatric OSA include loud chronic snoring, obesity, allergic rhinitis, naso- and oropharyngeal obstruction, retrognathia, micrognathia, and genetics. 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, a high, narrow, vaulted palate and maxillary constriction, increased soft palate volume, retrognathia, decreased nasal patency, malocclusion, posterior crossbite, anterior open bite, and increased anterior facial height. 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. The typical facial architecture associated with mouth breathing is termed “adenoid face” or “long face” syndrome. 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. 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, Class II malocclusion, crossbite, an increased anterior facial height, a hypertrophic soft palate, a narrow, high, vaulted palate, a low and flat tongue, and a steep mandibular occlusal plane. 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, the presentation of craniofacial architecture associated with mouth breathing may indicate latent craniofacial risk for OSA.

Children with OSA often will experience detrimental neurobehavioural consequences, a poorer quality of life, and can negatively affect academic performance. Some studies have shown that OSA in children may have a higher prevalence of attention deficit disorder (ADD). Negative cognitive consequences of pediatric OSA may be attributed to oxygen deprivation during sleep and sleep arousals. 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. Suspi-
The diagnostic strategy includes a sleep oriented history and physical exam, objective testing, and education of the client. 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. 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.

Common screening questionnaires used to assess OSA risk include the STOP-BANG questionnaire, and the Epworth sleepiness scale. 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. The Epworth sleepiness scale is commonly used to determine the extent of daytime sleepiness related to OSA as shown in Table 4.

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

<table>
<thead>
<tr>
<th>STOP-BANG questionnaire</th>
<th>Epworth Sleepiness Scale</th>
</tr>
</thead>
<tbody>
<tr>
<td>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</td>
<td></td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Situation</th>
<th>Chance of dozing or sleeping</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sitting and reading</td>
<td>............................... 0 = would never doze or sleep</td>
</tr>
<tr>
<td>Watching TV</td>
<td>............................... 0 = would never doze or sleep</td>
</tr>
<tr>
<td>Sitting in a public place</td>
<td>............................... 0 = would never doze or sleep</td>
</tr>
<tr>
<td>Being a passenger in a motor vehicle for an hour or more</td>
<td>............................... 0 = would never doze or sleep</td>
</tr>
<tr>
<td>Lying down in the afternoon</td>
<td>............................... 0 = would never doze or sleep</td>
</tr>
<tr>
<td>Sitting and talking to someone</td>
<td>............................... 0 = would never doze or sleep</td>
</tr>
<tr>
<td>Sitting quietly after lunch (no alcohol)</td>
<td>............................... 0 = would never doze or sleep</td>
</tr>
<tr>
<td>Stopped for a few minutes in traffic while driving</td>
<td>............................... 0 = would never doze or sleep</td>
</tr>
<tr>
<td>Total score</td>
<td>............................... 0 = would never doze or sleep</td>
</tr>
</tbody>
</table>

The in-laboratory polysomnogram (PSG) is considered the gold standard for diagnosing OSA. 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. There are a number of other diagnostic methods such as portable home monitoring units and anecdotal questionnaires. However, portable home monitoring units are recommended by the American Academy of Sleep Medicine 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.

### 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. 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.
Weight loss is usually difficult for clients with OSA. Behavioural modification involves tobacco cessation for OSA. 

Osborne and Ranson 

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, positional therapy, and abstinence from alcohol and sedative use prior to sleep. Weight loss is usually difficult for clients with OSA 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. Leptin regulates appetite satiety, 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.

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. However, not all OSA relates to sleep position. A diagnosis of a PSG will determine if positional therapy is indicated.

A novel association has been made between orofacial myofunctional therapy and improvement in AHI scores. 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. 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”. 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”. 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. 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. If CPAP therapy is declined, or the client is non adherent, the physician will determine whether alternative treatment for OSA is suitable. 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.

Clients with OSA and excessive daytime sleepiness are at higher risk for traffic accidents. In Ontario, physicians are required to report clients whose medical conditions are considered “dangerous...to operate a motor vehicle” to the Ministry of Transportation of Ontario (MTO). Criteria for physician reporting to the MTO include the presence of uncontrolled sleep apnea, 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. Disclosure of this contingency is the sole responsibility of the physician. 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 prior to oral appliance therapy or any other treatment modality. 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”. Once a treatment for OSA has been implemented, a second PSG is required to determine the efficacy of the treatment.

Clients who request treatment for snoring must undergo a physician consultation prior to treatment consideration. 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.” Snoring may be attributed to various etiologies. A differential diagnosis by a physician must be made prior to any treatment of snoring.

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.” Dental hygienists have a significant role in the screening of clients with OSA and in facilitating a medical diagnosis through an interprofessional referral.

Acknowledgements

The authors would like to acknowledge all those who assisted with this literature review. A special appreciation to Dr. Les Priemer, DDS; Dr. Jeff Shnall, DDS; Dr. Philip Wade, MD, DDS, FRCSC (C); Dr. Michael Fitzpatrick, MD, FRCPI, FRCP, D.ABSM; Joy Moeller, BS, RDH COM; Dr. Kruno Tovilo, DDS, Elsevier Publishing, and to Alex Novodvorets at the “CPAP Clinic” company for all their assistance and beneficence.

REFERENCES


Obstructive sleep apnea and the dental hygienist


Association of obstructive sleep apnea with hypertension: A systematic review and meta-analysis

Haifeng Hou1,2,*, Yange Zhao3,*, Wenqing Yu3,*, Hualei Dong4, Xiaotong Xue4, Jian Ding4, Weijia Xing1,*, Wei Wang1,2*

1 School of Public Health, Taishan Medical University, Taian, China
2 School of Medical and Health Sciences, Edith Cowan University, Perth, Australia
3 School of Basic Medical Science, Taishan Medical University, Taian, China
4 Taishan Hospital of Shandong Province, Taian, China
* These authors contributed equally to the article

Correspondence to:
Wei Wang
School of Medical and Health Sciences
Edith Cowan University
Perth, WA 6027
Australia
wei.wang@ecu.edu.au
Weijia Xing
School of Public Health
Taishan Medical University
2 Yingsheng East Road,
Taian 271000
PR China
71206930@qq.com

Obstructive sleep apnea (OSA) is a sleep disorder characterized as complete or partial upper airflow cessation during sleep, which mainly results from narrowed oropharyngeal or velopharyngeal anatomy [1,2]. OSA is reported to affect approximately 17% of American adults [3]. Hereditable dimension of craniofacial skeleton, obesity, and aging are considered risk factors for OSA, and the lack of neurophysiological regulation on airway dilator muscles also contributes to the causality of OSA [4]. Apart from causing uncomfortable symptomatology, untreated OSA is widely acknowledged to be associated with diabetes, cardiovascular disease and cerebrovascular disease [5]. Observational studies have illustrated that the prevalence of OSA is over 30% among hypertension patients and nearly 80% among resistant hypertensive patients [6,7]. Although the association between OSA and hypertension is considered obvious, the published results regarding this relation-

Background Obstructive sleep apnea (OSA) is a sleep disorder characterized as complete or partial upper airflow cessation during sleep. Although it has been widely accepted that OSA is a risk factor for the development of hypertension, the studies focusing on this topic revealed inconsistent results. We aimed to clarify the association between OSA and hypertension, including essential and medication-resistant hypertension.

Methods The Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA) was followed. PubMed and Embase databases were used for searching the relevant studies published up to December 31, 2016. A quantitative approach of meta-analysis was performed to estimate the pooled odds ratio (OR) and 95% confidence interval (CI).

Results Twenty-six studies with 51,623 participants (28,314 men, 23,309 women; mean age 51.8 years) met inclusion criteria and were included in this study. Among them, six studies showed a significant association between OSA and resistant hypertension (pooled OR = 2.842, 95% CI = 1.703–3.980, P < 0.05). Meanwhile, the combination of 20 original studies on the association of OSA with essential hypertension also presented significant results with the pooled ORs of 1.184 (95% CI = 1.093–1.274, P < 0.05) for mild OSA, 1.316 (95% CI = 1.197–1.433, P < 0.05) for moderate OSA and 1.561 (95% CI = 1.287–1.835, P < 0.05) for severe OSA.

Conclusions Our findings indicated that OSA is related to an increased risk of resistant hypertension. Mild, moderate and severe OSA are associated essential hypertension, as well a dose-response manner relationship is manifested. The associations are relatively stronger among Caucasians and male OSA patients.
ship are not consistent [3]. More than eight studies reported that OSA is not associated with hypertension, arousing skepticism of the effect of OSA on the risk for hypertension [2,8]. To our knowledge, the effect of continuous positive airway pressure (CPAP) therapy on blood pressure (BP) reduction improves the establishment of the causal association of OSA with hypertension and cardiovascular diseases [8]. However, a recent meta-analysis demonstrated that CPAP intervention does not reduce cardiovascular risk [9]. Besides another meta-analysis on CPAP treatment trials found a low reduction of HP (2.6 mm Hg for systolic BP and 2.0 mm Hg for diastolic BP) among OSA participants [10]. It has been manifested that CPAP may not be a sole hypertension intervention option for OSA patients [11]. To determine if OSA plays an independent causal role in hypertension, we conducted this systematic review and meta-analysis, and synthesized the studies on the association of OSA with essential hypertension and medication-resistant hypertension.

MATERIALS AND METHODS

We followed the criteria of the Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA). The reported PRISMA Checklist is provided in Table S1 in Online Supplementary Document. The protocol of this study has been registered in PROSPERO (No. CRD42017064336), available at https://www.crd.york.ac.uk/PROSPERO/.

Search strategy

The databases of PubMed and Embase were searched for literature published up to December 31, 2016. The search strategy was designed as a combination of the following key words: “obstructive sleep apnea” or “obstructive sleep apnea syndrome” or “sleep apnea” or “sleep disordered breathing” or “OSA” or “OSAS” or “SDB” and “hypertension” or “HTN”. In addition, studies cited in the references at the retrieved articles were further screened.

Selection criteria and quality assessment

Two authors independently reviewed the title, abstract and full text of the publications to determine the suitability for inclusion. Eligible studies were included based on the following criteria: 1) studies conducted on human populations; 2) studies conducted to investigate the relationships between OSA and essential and/or resistant hypertension; 3) case-control studies, case-control comparison of baseline data in cohort populations or clinical populations, and cohort studies; 4) odds ratios (OR) and 95% confidence interval (CI) available or can be calculated; 5) OSA patients diagnosed with polysomnography, and classified with apnea-hypopnea index (AHI); 6) studies published in English. We evaluated the methodological quality of each study according to the quality assessment scale by reference to PRISMA statement and MOOSE guideline (Table S2 and Table S3 in Online Supplementary Document).

Data extraction

Original data were retrieved independently from the eligible publications by two authors. The following data were extracted: author’s name, year of publication, ethnicity of participants, type of study design, sample size, characteristics of the participants. If results derived from the same population were published more than once, the most recent publication or the one with the largest population size was enrolled. If a publication investigated the baseline data, and also studied the association of recent-onset hypertension with OSA among different numbers of participants through a follow-up, the investigations were treated as two independent studies. Data in all subgroups were collected when the authors reported results with age or gender stratification. The adjusted ORs were extracted from articles.

Statistical analysis

All statistical analyses were performed with the Stata14.0 software (Stata Corp, College Station, TX, USA). The pooled ORs and 95% CIs were calculated to assess the strength of the association between OSA and hypertension. Q test and I² statistic were used to evaluate heterogeneity across the involved studies. When I²<50% and P>0.10, the fixed effect model was used to combine data sets. Otherwise, the random effect model was applied. Moreover, we conducted subgroup meta-analysis based on ethnicity of the population when heterogeneity was ultra (I²>50% or P<0.10). To assess the robustness of the combined results and evaluate the effect of individual studies on this meta-analysis, we carried out sensitivity analysis by
results of the study selection.

**Figure 1.** Flowchart of the study selection.

**Association between OSA and resistant hypertension**

We included six studies that explored the relationship between OSA and resistant hypertension [1,12-16], with five studies conducted among Caucasians and one study was among Asians. The synthesized results showed that OSA was significantly associated with resistant hypertension with a pooled OR of 2.842 (95% CI = 1.703-3.980, \( P < 0.05 \)) (Figure 2 and Table 2), which indicated that OSA participants
### Table 1. Characteristics of included studies

<table>
<thead>
<tr>
<th>Author</th>
<th>Year</th>
<th>Type of HTN</th>
<th>Country</th>
<th>Case</th>
<th>Mean Age</th>
<th>Matched Control</th>
<th>Gender</th>
<th>Duration of Follow-up</th>
</tr>
</thead>
<tbody>
<tr>
<td>Walia [1]</td>
<td>2014</td>
<td>R-HTN</td>
<td>United States</td>
<td>Hospital-based OSA</td>
<td>63.4 ± 6.9</td>
<td>NO</td>
<td>Case: 81/28</td>
<td>126/49</td>
</tr>
<tr>
<td>Wu [12]</td>
<td>2016</td>
<td>R-HTN</td>
<td>China</td>
<td>Hospital-based OSA</td>
<td>54.3 ± 3.0</td>
<td>NO</td>
<td>Case: 36/57</td>
<td>166/60</td>
</tr>
<tr>
<td>Drager [14]</td>
<td>2009</td>
<td>R-HTN</td>
<td>Brazil</td>
<td>Hospital-based OSA</td>
<td>51 ± 10</td>
<td>NO</td>
<td>Case: 30/25</td>
<td>22/22</td>
</tr>
<tr>
<td>Ruttenaumpawan [16]</td>
<td>2009</td>
<td>R-HTN</td>
<td>Canada</td>
<td>Hospital-based R-HTN</td>
<td>60.1 ± 1.8</td>
<td>YES</td>
<td>Case: 12/10</td>
<td>26/16</td>
</tr>
<tr>
<td>Bartel [17]</td>
<td>1995</td>
<td>E-HTN</td>
<td>South Africa</td>
<td>Hospital-based E-HTN</td>
<td>50.6 ± 8.0</td>
<td>YES</td>
<td>Case: 41/6</td>
<td>4/16</td>
</tr>
<tr>
<td>Crocker [18]</td>
<td>1989</td>
<td>E-HTN</td>
<td>Australia</td>
<td>Hospital-based OSA</td>
<td>51.5 ± 14</td>
<td>NO</td>
<td>Case: 9/10</td>
<td>78/22</td>
</tr>
<tr>
<td>Peppard [19]</td>
<td>2000</td>
<td>E-HTN</td>
<td>Australia</td>
<td>Hospital-based OSA</td>
<td>51 ± 8</td>
<td>NO</td>
<td>504/389</td>
<td>4 years</td>
</tr>
<tr>
<td>Guillot [21]</td>
<td>2013</td>
<td>E-HTN</td>
<td>France</td>
<td>Community-based OSA</td>
<td>68.0 ± 1.1</td>
<td>NO</td>
<td>Case: 34/39</td>
<td>3 years</td>
</tr>
<tr>
<td>Haas [22]</td>
<td>2005</td>
<td>E-HTN</td>
<td>United States</td>
<td>Community-based OSA</td>
<td>68.1 ± 0.9</td>
<td>NO</td>
<td>Case: 10/9</td>
<td>134/94</td>
</tr>
<tr>
<td>Haas [22]</td>
<td>2005</td>
<td>E-HTN</td>
<td>United States</td>
<td>Community-based OSA</td>
<td>52.2 ± 5.3</td>
<td>NO</td>
<td>1132/1345</td>
<td>NA*</td>
</tr>
<tr>
<td>Appleton [8] (study-a)</td>
<td>2016</td>
<td>E-HTN</td>
<td>Australia</td>
<td>Community-based OSA</td>
<td>58.0 ± 10.7</td>
<td>NO</td>
<td>448/0</td>
<td>NA*</td>
</tr>
<tr>
<td>Appleton [8] (study-b)</td>
<td>2016</td>
<td>E-HTN</td>
<td>Australia</td>
<td>Community-based OSA</td>
<td>62.6 ± 10.3</td>
<td>NO</td>
<td>2853/3192</td>
<td>NA*</td>
</tr>
<tr>
<td>Kapur [23]</td>
<td>2010</td>
<td>E-HTN</td>
<td>United States</td>
<td>Community-based E-HTN</td>
<td>53 ± 10</td>
<td>NO</td>
<td>420/182</td>
<td>NA*</td>
</tr>
<tr>
<td>Li [24]</td>
<td>2015</td>
<td>E-HTN</td>
<td>China</td>
<td>Hospital-based OSA</td>
<td>43.0 ± 12.1</td>
<td>NO</td>
<td>409/451</td>
<td>NA*</td>
</tr>
<tr>
<td>Priou [26]</td>
<td>2014</td>
<td>E-HTN</td>
<td>France</td>
<td>Community-based OSA</td>
<td>54.3 ± 13.5</td>
<td>NO</td>
<td>955/544</td>
<td>NA*</td>
</tr>
<tr>
<td>Redline [27]</td>
<td>2014</td>
<td>E-HTN</td>
<td>United States</td>
<td>Community-based OSA</td>
<td>46.7 ± 10.8, F: 50.5 ± 6.0</td>
<td>NO</td>
<td>7547/8693</td>
<td>NA*</td>
</tr>
<tr>
<td>Smith [28]</td>
<td>2014</td>
<td>E-HTN</td>
<td>Canada</td>
<td>Community-based OSA</td>
<td>46.7 ± 10.8, F: 50.5 ± 6.0</td>
<td>YES</td>
<td>Case: 599/174</td>
<td>599/174</td>
</tr>
<tr>
<td>Xie [29]</td>
<td>2011</td>
<td>E-HTN</td>
<td>United States</td>
<td>OSA from drivers</td>
<td>46.7 ± 9.82</td>
<td>NO</td>
<td>109/80</td>
<td>165/50</td>
</tr>
<tr>
<td>Young [30]</td>
<td>1997</td>
<td>E-HTN</td>
<td>United States</td>
<td>Community-based OSA</td>
<td>30 ± 60</td>
<td>NO</td>
<td>617/443</td>
<td>NA*</td>
</tr>
<tr>
<td>Yusoff [31]</td>
<td>2010</td>
<td>E-HTN</td>
<td>Malaysia</td>
<td>OSA from drivers</td>
<td>45.4 ± 7.0</td>
<td>NO</td>
<td>128/0</td>
<td>161/0</td>
</tr>
<tr>
<td>Zou [32]</td>
<td>2012</td>
<td>E-HTN</td>
<td>United States</td>
<td>E-Community-based HTN</td>
<td>60 ± 7</td>
<td>NO</td>
<td>97/886</td>
<td>3 years</td>
</tr>
<tr>
<td>O’Connor [33]</td>
<td>2009</td>
<td>E-HTN</td>
<td>United States</td>
<td>Community-based OSA</td>
<td>59.6 ± 10.3</td>
<td>NO</td>
<td>1103/1367</td>
<td>5 years</td>
</tr>
<tr>
<td>Chan [34]</td>
<td>2016</td>
<td>E-HTN</td>
<td>Singapore</td>
<td>Hospital-based OSA</td>
<td>57.1 ± 9.7</td>
<td>NO</td>
<td>534/53</td>
<td>2.5 years</td>
</tr>
</tbody>
</table>

R-HTN – Resistant hypertension, E-HTN – Essential hypertension, OSA – obstructive sleep apnea, Con – control group

*Baseline data in cohorts.

†Cross-sectional designed case-control study.
Association of obstructive sleep apnea with hypertension

Table 2. Meta-analysis of association between OSA and hypertension

<table>
<thead>
<tr>
<th>Type of HTN</th>
<th>Category of OSA</th>
<th>Subgroup</th>
<th>Effect size</th>
<th>Heterogeneity test</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>OSA/non-OSA</td>
<td>Asian</td>
<td>Pooled OR</td>
<td>95% CI</td>
</tr>
<tr>
<td>Resistant HTN</td>
<td></td>
<td></td>
<td>2.460</td>
<td>1.500-4.040</td>
</tr>
<tr>
<td></td>
<td>OSA/non-OSA</td>
<td>Caucasian</td>
<td>4.406</td>
<td>1.835-6.977</td>
</tr>
<tr>
<td></td>
<td>OSA/non-OSA</td>
<td>Overall</td>
<td>2.842</td>
<td>1.703-3.980</td>
</tr>
<tr>
<td>Essential HTN</td>
<td>Mild OSA</td>
<td>Prospective</td>
<td>1.038</td>
<td>0.808-1.267</td>
</tr>
<tr>
<td></td>
<td>Mild OSA</td>
<td>Non-Prospective</td>
<td>1.210</td>
<td>1.112-1.309</td>
</tr>
<tr>
<td></td>
<td>Mild OSA</td>
<td>Overall</td>
<td>1.184</td>
<td>1.093-1.274</td>
</tr>
<tr>
<td></td>
<td>Moderate OSA</td>
<td>Caucasian</td>
<td>1.315</td>
<td>1.197-1.433</td>
</tr>
<tr>
<td></td>
<td>Moderate OSA</td>
<td>Prospective</td>
<td>1.224</td>
<td>0.917-1.530</td>
</tr>
<tr>
<td></td>
<td>Moderate OSA</td>
<td>Non-Prospective</td>
<td>1.332</td>
<td>1.204-1.456</td>
</tr>
<tr>
<td></td>
<td>Moderate OSA</td>
<td>Overall</td>
<td>1.316</td>
<td>1.198-1.434</td>
</tr>
<tr>
<td></td>
<td>Severe OSA</td>
<td>Caucasian</td>
<td>1.549</td>
<td>1.275-1.824</td>
</tr>
<tr>
<td></td>
<td>Severe OSA</td>
<td>Prospective</td>
<td>1.505</td>
<td>0.998-2.013</td>
</tr>
<tr>
<td></td>
<td>Severe OSA</td>
<td>Non-Prospective</td>
<td>1.584</td>
<td>1.258-1.909</td>
</tr>
<tr>
<td></td>
<td>Severe OSA</td>
<td>Overall</td>
<td>1.561</td>
<td>1.287-1.835</td>
</tr>
<tr>
<td>Essential HTN</td>
<td>OSA/non-OSA*</td>
<td>Asian</td>
<td>1.583</td>
<td>1.160-2.007</td>
</tr>
<tr>
<td></td>
<td>OSA/non-OSA*</td>
<td>Caucasian</td>
<td>1.928</td>
<td>1.600-2.256</td>
</tr>
<tr>
<td></td>
<td>OSA/non-OSA*</td>
<td>Prospective</td>
<td>1.475</td>
<td>1.033-1.914</td>
</tr>
<tr>
<td></td>
<td>OSA/non-OSA*</td>
<td>Non-Prospective</td>
<td>1.972</td>
<td>1.651-2.294</td>
</tr>
<tr>
<td></td>
<td>OSA/non-OSA*</td>
<td>Overall</td>
<td>1.799</td>
<td>1.339-2.058</td>
</tr>
</tbody>
</table>

HTN – hypertension, OSA – obstructive sleep apnea, OR – odds ratio; CI – confidence interval

*Meta-analysis of six studies only divided subjects into OSA and non-OSA groups.

had an extra 1.842-fold risk for resistant hypertension prevalence compared to non-OSA participants. Subgroup analysis explicated that this association was more outstanding among Caucasian populations (OR = 4.406, 95% CI = 1.835-6.977).

The heterogeneity test showed no significant heterogeneity generated across all primary studies (P² = 0%, Q = 2.23, P = 0.816).

**Association between OSA and essential hypertension**

Twenty studies investigated the relationship between OSA and essential hypertension [2,8,17-34]. Among them, 16 studies classified OSA patients into three grades based on AHI their scores, which were mild OSA (AHI>5), moderate OSA (AHI>15) and severe OSA (AHI>30). Since natural differences exists between mild OSA, moderate OSA and severe OSA, results were interpreted separately. As shown in Table 2 and Figures 3 to 5, essential hypertension was significantly associated with OSA, in terms of mild OSA (OR = 1.184, 95% CI = 1.093-1.274, P < 0.05), moderate OSA (OR = 1.316, 95% CI = 1.198-1.434, P < 0.05), and severe OSA (OR = 1.561, 95% CI = 1.287-1.835, P < 0.05). These results showed the trend of that the more serious OSA is, the higher hypertension risk occurs. Subgroup analyses were conducted based on study design (prospective/non-prospective) and ethnicity. As shown in Table 2, no significant results were found in studies with prospective study design, which outlined the pooled ORs (95% CIs) of 1.038 (0.808-1.267) for mild OSA, 1.224 (0.917-1.530) for moderate OSA, and 1.505 (0.998-2.013) for severe OSA.

Figure 3. Forest plots of the association between essential hypertension and mild obstructive sleep apnea (OSA).
heterogeneity test illustrated that $I^2$ values were 10.1% ($P=0.348$) in mild OSA, 3.2% ($P=0.416$) in moderate OSA, and 0% ($P=0.546$) in severe OSA meta-analyses, indicating no significant heterogeneity in each group.

For the seven studies dividing participants only into OSA and non-OSA groups [8,24,26,28,29,31,34], the pooled OR was 1.799 (95% CI = 1.539-2.058, $P<0.05$), demonstrating that OSA was associated with essential hypertension (Figure S1 and Table S2 in Online Supplementary Document). The subgroup analysis based on ethnicity showed significant results among Asians (OR = 1.583, 95% CI = 1.160-2.007) and Caucasians (OR = 1.928, 95% CI = 1.600-2.256). Additionally, no significant heterogeneity was found ($I^2 = 26.0\%$, $P=0.221$). Four studies in the three articles reported the relationship between OSA and essential hypertension among male participants [2,8,33]. The combined result of these four studies suggested that the association between OSA and essential hypertension was relatively stronger among males (OR = 1.698, 95% CI = 1.319-2.077).

**Publication bias**

The funnel plot analysis, followed by Egger’s test, was applied to detect the potential publication bias. Significant publication bias existed in the meta-analyses of moderate OSA and severe OSA participants, and the meta-analysis of resistant hypertension (Table 3 and Figures S7-S11 in Online Supplementary Document). The Trim and Fill analysis was performed to eliminate the effect of publication bias on meta-analysis. As shown in Table 3 and Figures S12-S14 in Online Supplementary Document, the pooled ORs (95% CIs) were 1.327 (1.222-1.441) for moderate OSA and 1.593 (1.369-1.853) for severe OSA meta-analyses, and 1.575 (1.117-2.221) for resistant hypertension.

**Table 3.** Analysis on publication bias and results of Trim and Fill method analysis

<table>
<thead>
<tr>
<th>Type of HTN</th>
<th>Category of OSA</th>
<th>Egger’s Test</th>
<th>$t$</th>
<th>$p$</th>
<th>Pooled OR</th>
<th>95% CI</th>
<th>Trim and Fill Method Analysis</th>
<th>$P$</th>
</tr>
</thead>
<tbody>
<tr>
<td>Resistant HTN</td>
<td>OSA/non-OSA</td>
<td></td>
<td>2.36</td>
<td>0.078</td>
<td>1.575</td>
<td>1.117-2.221</td>
<td>0.010</td>
<td></td>
</tr>
<tr>
<td>Essential HTN</td>
<td>Mild OSA</td>
<td></td>
<td>0.92</td>
<td>0.380</td>
<td>1.187</td>
<td>1.103-1.276</td>
<td>&lt;0.001</td>
<td></td>
</tr>
<tr>
<td>Essential HTN</td>
<td>Moderate OSA</td>
<td></td>
<td>3.91</td>
<td>0.001</td>
<td>1.327</td>
<td>1.222-1.441</td>
<td>&lt;0.001</td>
<td></td>
</tr>
<tr>
<td>Essential HTN</td>
<td>Severe OSA</td>
<td></td>
<td>1.98</td>
<td>0.079</td>
<td>1.593</td>
<td>1.369-1.853</td>
<td>&lt;0.001</td>
<td></td>
</tr>
<tr>
<td>Essential HTN</td>
<td>OSA/non-OSA</td>
<td></td>
<td>0.87</td>
<td>0.420</td>
<td>1.356</td>
<td>1.184-1.554</td>
<td>&lt;0.001</td>
<td></td>
</tr>
</tbody>
</table>

HTN – hypertension, OSA – obstructive sleep apnea, OR – odds ratio, CI – confidence interval
Association of obstructive sleep apnea with hypertension

Sensitivity analysis
To evaluate the robustness of the results, sensitivity analyses were undertaken by sequentially removing each study. Consequently, no obvious change was generated on omission of any individual study (Figure S2-S6 in Online Supplementary Document), confirming that the results of our meta-analyses were stable. This coincided with the results of heterogeneity tests.

DISCUSSION
This systematic review and meta-analysis was conducted in an effort to clarify the association between OSA and hypertension, including both essential and resistant hypertension. Our findings suggest that OSA confers a significant association with both essential and resistant hypertension. Moreover, OSA is associated with mild OSA, moderate OSA and severe OSA with a stepwise increased degree.

OSA are acknowledged to be associated with hypertension [6,7]. Nevertheless, the potential mechanisms underlying the associations have not been well elucidated. Several potential explanations may help us to understand the association of OSA and hypertension. OSA induces intermittent hypoxemia, similarly to hypoxia/reperfusion injury, and causes oxidative stress, leading to dysfunction of vascular endothelium [35]. Meanwhile, excessive outflow of sympathetic vasoconstrictor together with diminished nitric oxide bioavailability plays a role in blood pressure elevation [36,37]. In addition, episodes of OSA up-regulate sympathetic excitation which acts on the chemoreflex and may consequently result in hypertension [10,38]. In clinical observations, sympathetic nervous activity, reflected by 24-hour urinary catecholamine excretion, is increased in individuals with sleep-disordered breathing [39]. The data from relevant surveys revealed that the prevalence rate of OSA is 70-83% in resistant hypertensive patients [40]. It is also suggested that untreated OSA may lead to reduced effectiveness of medications through pharmacokinetic or chronotherapeutic effects, activating a pathway of resistance to antihypertensive drugs [1]. In the present study, we found that OSA strongly predisposes to resistant hypertension by combining six relevant studies. The pooled OR of the causal association in Caucasians is 4.406, which indicates Caucasians with OSA suffer more from uncontrolled hypertension.

OSA is also considered to be associated with essential hypertension and the consequences of cardiovascular diseases. However, the results are not entirely consistent, meanwhile more than eight studies have generated non-significant results [2,8,13,17,20,22-23,33]. This meta-analysis, involving 20 original studies in 19 articles, demonstrates that OSA increases the risk of essential hypertension in a dose-response manner. Although the dose-response meta-analysis has not been conducted owing to the lack of current available data, the trend of pooled ORs shows obviously that the more serious OSA is, the higher hypertension risk occurs.

Gender is indispensable factor in the analysis of demographic data. Nonetheless, seldom studies reported the association between OSA and hypertension in specific gender. O’Connor et al. reported that female patients with moderate and severe OSA have higher prevalence of essential hypertension than male [33], while, Nieto et al reported a lower OR in severe OSA women before [2]. After combining four studies reported in three articles [8,29,31], our finding enlightens that men suffer from higher incidence of essential hypertension than women, as well the association in Caucasians is greater than that in Asians. These differences may be explained by the larger proportion of overweight and obesity among Caucasians and also among men [18]. This finding is consistent with a recent study by Cano-Pumarega et al., despite the fact that their study did not diagnose OSA with polysomnography and AHI [41]. Regarding ethnic subgroup analysis, the different results may subject to genetic backgrounds, environmental factors and genetic-environmental interaction [42,43].

Heterogeneity may result from diversity of clinical study design or statistical methodology, and the detection of heterogeneity is essential for meta-analysis. No significant heterogeneity was found in the present study, indicating high compatibility of our results. Care must be taken in the adjustment of potential confounding factors in the statistical analysis of clinical studies. Obviously, obesity and age are both contributing factors to OSA and hypertension. Although investigators suggested that the impact of body mass index (BMI) on OSA is quite modest and adjustment for BMI might be an over-adjustment [2,32], we have synthesized the OR adjusted for both age and BMI in order to estimate the risk more precisely.

Publication bias, a critical issue in systematic review, mainly results from the tendency that studies with positive results are more likely to be published than those with negative findings. Significant publication
bias exists in our meta-analyses of moderate OSA, severe OSA and essential hypertension. To reduce potential confounding of publication bias, we carried out the Trim and Fill analysis. According to the results of Trim and Fill analysis, the pooled ORs remained statistically significant for the association between moderate OSA and essential hypertension, the association between severe OSA and essential hypertension, as well as the association between OSA and resistant hypertension.

Comparing to low specificity of sleep questionnaires, AHI is an accurate parameter for determining OSA [14], which is defined as the number of episodes of apneas or hypopneas in 1 hour of sleep, recorded with polysomnography test [2]. One of inclusion criteria in this study is the usage of AHI as OSA diagnostic index, this obviously limits the number of studies to be included.

This study has certain limitations that are common in systematic reviews meta-analyses of descriptive studies: First, most of the original studies were based on cross-sectional designed investigation, which may limit the argumentation intensity of our conclusion. Second, the subgroup analyses based on age were not performed because of no primary data available from the included studies. Only 5 prospective studies [8,19,21,33,34] meeting the inclusion criteria were included, and the pooled results of the studies showed no statistical significance of association between OSA and hypertension. However, the number of included studies/patients is small. More prospective studies need to be conducted to clarify the relationship between OSA and hypertension.

The present study demonstrates that OSA is related to an increased risk of resistant hypertension, with a stronger association among Caucasian populations and male OSA patients. Mild, moderate and severe OSA are associated essential hypertension, as well a dose-response manner is manifested.

Our interpretation of synthesized epidemiological evidence strengthens the acceptance of OSA as a risk factor of hypertension, in term of essential hypertension and resistant hypertension. Given the underlying mechanisms have been explained, the key remaining questions are how does OSA mediate hypertension and how can we implement specific interventions to reduce hypertension induced by OSA. More studies should be conducted to examine the mechanisms of hypertension regulation by OSA, especially among medication-resistant hypertension patients.

Acknowledgements: This study was supported by the grants from the Teachers’ Training Project of Taishan Medical University, the National Natural Science Foundation of China (No. 81372586, 81370083, 81273170), and Shandong Provincial Natural Science Foundation, China (No. ZR2017MH100). We thank Enoch Odame Anto, Edith Cowan University, Australia for his English editing on this manuscript.

Authorship declaration: HH, WX and WW conceived and designed the study. HH, XX, and WW contributed to the professional review of medical approaches in the publications. HH and HD analyzed the data. HH, WX, and WW contributed to the writing of the manuscript. All authors read and approved the final manuscript.

Ethics approval: Not applicable.

Competing interests: All authors have completed the ICMJE uniform disclosure form at http://www.icmje.org/coi_disclosure.pdf (available upon request from the corresponding author) and declare no conflicts of interest.

Association of obstructive sleep apnea with hypertension

References


YOU NOW NEED TO VISIT IAOMT’S YOUTUBE CHANNEL TO WATCH THE “PEDIATRIC SLEEP-DISORDERED BREATHING” PRESENTATION BY BETH ROSELLINI, DDS, AIAOMT, AT https://youtu.be/7twXc5y7j2M.

UPON COMPLETION OF THE “PEDIATRIC SLEEP-DISORDERED BREATHING” VIDEO, YOU WILL NEED TO CONTINUE WITH THE ADDITIONAL REQUIREMENTS FOR UNIT 6, WHICH INCLUDE MORE READINGS AND ANOTHER VIDEO, AS WELL AS COMPLETING THE UNIT 6 TEST.
**Myofunctional Therapy**  
A Novel Treatment of Pediatric Sleep-Disordered Breathing

Joy L. Moeller, BS, RDH\(^a\),*  
Licia Coceani Paskay, MS, CCC-SLP\(^a\)  
Michael L. Gelb, DDS, MS\(^b\)

**INTRODUCTION**

Orofacial myofunctional therapy (OMT) is defined as the treatment of dysfunctions of the muscles of the face and mouth, with the purpose of correcting orofacial functions, such as chewing and swallowing, and promoting nasal breathing. OMT has been used for many years to repattern and change the function of the oral and facial muscles and to eliminate oral habits, such as prolonged thumb-sucking and nail biting, tongue thrusting, open mouth at rest posture, incorrect mastication, and poor oral rest postures of the tongue and lips.\(^1\) Physicians, dentists, and orthodontists have also used myofunctional therapy as an adjunctive noninvasive option for the treatment of temporomandibular joint disorders (TMJD).

In the last few years\(^2,3\) myofunctional therapy has also been proposed as a potentially important component of the multidisciplinary treatment of obstructive sleep apnea (OSA). The use of OMT as a noninvasive option for the treatment of sleep-disordered breathing (SDB) in children has the potential to become an important alternative to other available nonsurgical treatment modalities, such as positive airway pressure and

---

**KEY POINTS**

- Orofacial myofunctional therapy (OMT) is a noninvasive option for the treatment of sleep-disordered breathing (SDB) in children.
- OMT has the potential to become an important alternative to other available nonsurgical treatment modalities.
- Early identification and correction of mouth breathing are recommended as early as the first year of life.
- Removing the tonsils and adenoids does not always change the breathing pattern from oral to nasal, if the habit of mouth breathing has not been corrected.
- Myofunctional therapists use a variety of supportive techniques to promote self-awareness and positive habits and to prevent the dysfunctions that characterize pediatric SDB.

---

Disclosures: Paid lecturer for the Academy of Orofacial Myofunctional Therapy (AOMT), personally related to the AOMT Managing Director a main shareholder, Marc Moeller; Vice-president of the Academy of the 501(c)3 Academy of Applied Myofunctional Sciences (AAMS) (J.L. Moeller); Licia Coceani Paskay is a paid lecturer for the AOMT and President of the 501(c)3 AAMS (L.C. Paskay); No conflicts of interest (M.L. Gelb).

\(^a\) Academy of Orofacial Myofunctional Therapy (AOMT), 910 Via de la Paz #106, Pacific Palisades, CA 90272, USA; \(^b\) Department of Oral Medicine and Pathology, Tufts University School of Dental Medicine, NYU, 635 Madison Avenue, 19th Floor B/W: 59th & 60th Street, New York, NY 10022, USA

* Corresponding author.

E-mail address: joyleamoeller@aol.com

Sleep Med Clin 9 (2014) 235–243

http://dx.doi.org/10.1016/j.jsmc.2014.03.002

1556-407X/14/$ – see front matter © 2014 Elsevier Inc. All rights reserved.
oral appliances. This article outlines the development and clinical application of OMT, discusses the rationale for its application to SDB, and presents evidence supporting this treatment as it relates to prevention, assessment, and treatment of pediatric SDB.

**HISTORY OF OMT**

The history of myofunctional therapy in the United States goes back to the early 1900s and parallels orthodontic treatment. In the 1950s to 1960s, Walter Straub, an orthodontist, wrote numerous articles on malfunctions of the tongue and abnormal swallowing habits and their relationship to orthodontics and speech. He thought a major cause of oral problems was bottle-feeding. Inspired by the work of Walter Straub, Roy Langer, Marvin Hanson, and Richard Barrett in the 1970s and 1980s, Daniel Garliner was the first to recommend a therapeutic routine for nighttime sleeping consisting of keeping the lips together and the tongue up on the palate. Subsequently, 2 speech pathologists from Brazil, Irene Marchesan and Ester Bianchini, studied with Daniel Garliner in the 1980s and went back to Brazil, where they created a university program for speech pathologists centered on treating orofacial myofunctional disorders. Today, there are over 30 universities with PhD programs in myofunctional therapy and many programs that focus on sleep disorders and myofunctional therapy.

**RATIONALE: DEVELOPMENT OF THE UPPER AIRWAY**

As man evolved to an upright posture, the larynx descended, the forebrain grew, and the facial framework retreated, as the nasal airway became diminished in size and function. This evolution is one reason humans do not have the olfactory ability of other mammals. As the cranial base angle flexed, the maxilla was compressed and the para-nasal sinus size was reduced, creating millions of sinus sufferers as well as other facial changes. The flattened maxilla and longer face is a relatively recent phenomenon seen in humans, differentiating man from primates. The decrease in nose volume associated with cranial base flexing may have increased high upper airway resistance and increased the potential for collapse further down in the oropharynx. Man was no longer an obligate nose breather, and with increased demands, mouth breathing was born. This trend of mouth breathing, downward migration of the tongue base and descent of the hyoid, is associated with retrognathic changes in mandibular posture. The increase in mouth breathing is associated with less time spent with tongue to the palate, and therefore, with narrowing of the maxilla and an increased facial height. This downward and backward rotation of the maxilla and mandible is a powerful predictor of SDB as well as TMJD and malocclusion. A variety of researchers, clinicians, and anthropologists have identified an underdeveloped maxilla as being the root cause of malocclusion and naso-oropharyngeal constriction. Early identification of mouth breathing is therefore recommended as early as the first year of life.

Although the primary function of the genioglossus muscle is to protect the patency of the upper airway, an improper oral resting posture of the tongue will have a negative influence on the development of the oral cavity and the airway. The anatomy of the upper airway in turn guides the growth and development of the nasomaxillary complex, mandible, temporomandibular joint, and ultimately, the occlusion of the teeth; thus, malocclusion and facial dysmorphism may be the result of compensation for a narrowed airway (Fig. 1).

**Genioglossus Muscle Stabilizing the Airway**

There are several etiologic factors that have been linked in varying degrees to the development of SDB in children, which have implications for the potential utility of OMT as a therapeutic intervention; these implications include feeding methods, oral habits, craniofacial abnormalities, hypertrophic tonsils and adenoids, chronic mouth breathing sleep position, and restricted frenum. For example, bottle-feeding has been shown to be a major contributing factor to an anterior open bite in the primary dentition, whereas overuse of spouted (“sippy”) cups may also contribute to a low tongue-rest posture, thereby leading to a narrow high palate. Oral habits such as the habitual use of a thumb or pacifier may also lead to a low tongue rest posture and OMD. It has been noted that the frequency, intensity, and duration of oral habits and mouth-soothing devices may lead to OMDs. When the thumb or another object is in the mouth often and/or for a prolonged period of time, as a self-soothing strategy for example, it applies pressure against the palate, and the tongue may develop a low rest posture. Also, incorrect pressure exerted on the jaws may lead to airway problems and a TMJD. Other oral habits such as finger-sucking, nail biting, lip licking, and tongue sucking may develop in infancy and persist into adulthood, leading to malocclusion.

Moeller et al
Mouth breathing or an open mouth at rest may be one cause of OMDs. If the mouth is open, the tongue usually rests down and forward. This position may cause an abnormal growth pattern, which may lead to a forward head and neck posture, malocclusion, and SDB. Mouth breathing also involves lack of lip closure, which is necessary for jaw stability and to create the intraoral negative pressure necessary to hold the tongue in place. Moreover, in mouth breathing there is a lack of tongue-to-palate contact, necessary to create the “suction-cup” effect that holds the tongue in place and prevents it from falling into the pharynx.

Hypertrophic tonsils and adenoids may also lead to OMD and SDB. If the palatine tonsils are hypertrophic, the tongue is prevented from swallowing properly, forcing the tongue to come forward during the swallow and to rest forward and down. However, removing the tonsils and adenoids does not always change the breathing pattern from oral to nasal, especially in the long-term. A myofunctional therapist may be needed to assist the child in retraining the function of the tongue, in breathing, chewing, and swallowing, and to eliminate maladaptive oral habits. Finally, restricted lingual or labial frenula may cause an OMD; if the tongue is not able to create a vacuum seal on the palate, then a high and narrow palate may result, which is considered to be a risk factor for OSA (Fig. 2).

Several studies support an empiric basis for myofunctional therapy in the treatment of SDB in adults. In an often-referenced study, Guimarães and colleagues reported not only reduced symptoms of sleep apnea but also objective evidence of decreased disease severity. The study reports that the apnea/hypopnea index (AHI) was reduced by 39% in those patients, after 3 months of myofunctional therapy. More recently, a series of studies on the application of myofunctional therapy of SDB in children from Stanford University showed that the addition of myofunctional therapy to adenotonsillectomy or palatal expansion reduced the risk of reoccurrence of SDB. A retrospective investigation by Guillermual and colleagues evaluated the application of myofunctional therapy along with adenotonsillectomy and orthodontic treatment. In patients who received myofunctional therapy, the AHI and the oxygen desaturation were normalized, whereas most subjects who did not receive myofunctional therapy experienced a relapse in both the AHI and the mean minimum oxygen saturation. The authors conclude that the absence of myofascial (myofunctional) treatment is associated with an increased risk of SDB recurrence.

Although studies that show a specific effect of myofunctional therapy on children’s sleep is relatively small, research supporting that OMT indeed normalizes the basic orofacial functions involved in SDB is more robust. For example, Izu and colleagues found that oral breathers were more likely to have snoring and OSAs and suffer from adenotonsillitis and otological symptoms. Cunha and colleagues found that breathing abnormalities in children not only alter sleep but affect chewing and food intake. Normalizing orofacial functions in children also requires time. Marson and colleagues demonstrated the effectiveness of an OMT program to normalize nasal breathing with peak results at 12 weeks, whereas Gallo and Campiotto,
using a similar protocol, found nasal breathing was normalized after about 10 sessions.

**CLINICAL ASSESSMENT**

Every health professional who works with patients with sleep disorders has different tools available for assessment, based on their needs, scope of practice, and preferences. Myofunctional therapists, as a multidisciplinary group of professionals, use various tools and practices, which often overlap but retain some individual characteristics depending on the background of the therapist. Moreover, myofunctional therapists are trained to identify other underlying orofacial dysfunctions that are affected or are a contributing factor in sleep disorders.

As part of the standard evaluation, the orofacial myofunctional therapist takes a thorough medical and developmental history, with an emphasis on SDB risk factors. Important components of the assessment include identification of oral habits that interfere with a proper oral rest posture, recognition of the incorrect rest position of the tongue, determination of incorrect swallow, labial and lingual frenum restriction and inadequate lip seal, and evaluation of functional head and neck posture (after age 3–4 years) (Figs. 3–14).
Fig. 5. Tongue rest position.

Fig. 6. Over-developed mentalis muscle.

Fig. 7. Tense peri-oral muscles.

Fig. 8. Open lips at rest: may be flacid, swollen or cracked.

Fig. 9. High narrow palate.

Fig. 10. Forward head posture.
Treatment

Treatment consists of habit elimination and behavior modification, jaw stabilization exercises, repatterning the oral facial muscles and changing their function for optimal nasal breathing, oral rest position, chewing, and swallowing. There are 4 basic components to the treatment:

1. Restoring Proper Rest Oral Posture

The first step is to educate the patient about problematic oral habits they may have and how to modify or eliminate the behavior, in terms of reduced frequency, duration, and the intensity of the habit. Myofunctional therapists use a variety of supportive techniques to allow the patient to first understand the damage being done and then to solicit a commitment to change, even in young children. Then, the patient is supported with rewards and positive reinforcement from both the family and the therapist. Therapists then will introduce diaphragmatic breathing and create a lip seal (in the absence of airway blockages or allergies), so that the lips are closed during the night. Therapy then continues with training the blade of the tongue to go to the “spot,” which is located posterior to the first rugae or ridge posterior to the maxillary central incisors on the palate. This therapy will also help to substitute the thumb with the tongue if necessary.

2. Repatterning of Facial Muscles

Next, the therapist will work with a sequential set of exercises to activate and then repattern the oral facial muscles. Therapists work with the muscles of mastication. Therapists work with the muscles of mastication. Therapists work with the muscles of mastication. Therapists work with the muscles of mastication. Therapists work with the muscles of mastication. Therapists work with the muscles of mastication.
aid the proper position of the genioglossus at night. Then, additional training addresses the orbicularis oris as well as the intrinsic and extrinsic tongue muscles, the buccinators, and the perioral muscles.

3. Teaching Proper Chewing and Swallowing

Next, proper chewing and swallowing is gradually introduced. Proper oral posture is reinforced even during sleep, with subconscious auto-suggestion and biofeedback. Success is evaluated using the Mallampati score, the grade of tongue scalloping, relaxation, or activation of the perioral muscles, as well as attaining a lip seal and palatal tongue rest position during both the day and the night.

4. Functional posture training

Myofunctional therapists are trained to promote a functional head position during sleep, to avoid the jaw being in close proximity to the chest because this position may contribute to SDB. Also, OMTs instruct patients to hold an upright head and neck posture, especially during the swallowing process.

If myofunctional therapists suspect that the “tongue-tie” (or lip-tie) is contributing to a child’s SDB, they will evaluate both the labial and the lingual frenae, usually after a few weeks of exercises to ensure that full range of motion of the tongue and lips is possible. If the restriction remains, the patient is referred to a physician or dentist who is comfortable doing the surgery. After the release, the patient must immediately do exercises to assure proper function of the tongue. Otherwise, more revisions may be required.

The key to successful treatment is to establish a rapport with the pediatric patient and the caregiver and to motivate and monitor the outcome on a weekly basis for several months and then gradually reduce the frequency of appointments to once a month. The therapist must also enlist the assistance of the parent or caregiver to become the “therapist” at home to assure a successful result.

Because myofunctional therapy relies on active patient participation, OMTs use several techniques that are based on the 10 principles of neurolasticity. Neurolasticity means the ability of the brain to change, following physiologic or pathologic input, generating an adaptive response. These principles include the following.

**Use it or lose it**

In general, because muscle function requires energy, if the muscles are not properly used, the brain stops or reduces nourishing those muscles and hypotonia may follow. Two studies indicated that loss of prolonged sensory input translates to a reduction of the somatocortical representation, such as in children with a habitual open mouth during the day and at night.

**Use it and improve it**

Myofunctional therapy revolves around the principle of improving a function through repetition, metacognition, and awareness. For example, the tongue is repositioned and trained to contact the palate comfortably, thus providing the natural negative pressure (suction) that keeps the tongue, and especially the genioglossus, in the proper position during sleep.

**Plasticity is experience specific**

This principle suggests that the success of some therapy protocols for sleep disorders relies on targeting the very muscles that are hypofunctioning at night, such as the soft palate, tongue, and pharyngeal walls.

**Repetition matters**

“Practice” improves performance by creating, maintaining, and expanding new neural areas corresponding to the new behavior. In myofunctional therapy repetition is paramount so that a new behavior, such as the tongue position or lips closure, is rehearsed every day and every evening until the new habit is formed.

**Intensity matters**

Ideally, patients should practice neuromuscular exercises every day; otherwise, the intensity of the neuromuscular change does not generalize to the night hours.

**Time matters**

According to Fisher and Sullivan, the training modality that is most effective is protracted and continuous, as opposed to brief and intermittent. Therefore, patients may need to be kept in therapy or follow-up mode for a prolonged period of time (usually 1 year, but 2 years is better for habituation).

**Salience matters**

The need to motivate the patient by increasing the saliency or importance of therapy is a central element, because the higher the motivation and understanding of the reason some exercises need to be performed daily, the more likely the patient will perform the exercises prescribed.

**Age matters**

Children are in the best condition to transform sensory-motor inputs into correct functions and
make them a life-long habit. In children, not only is neuroplasticity at its best but also muscles and soft tissues drive the development of bones through principles of the functional matrix and epigenetic influences.17,26

**Transference**

The transference principle supports the co-occurrence of multiple functions when an overlapping one has been established.27 When the patient breathes well through the nose, other functions can now easily take place even if they were hampered before, such as tongue repositioning or lip seal.

**Interference**

When a patient learns a new behavior (such as nasal breathing), the old behavior (such as oral breathing) has the ability to interfere neurologically with the establishment of the new one. It is only by continuous repetition of the new behavior and suppression of the old one that plasticity occurs.

Because a transition from daily myofunctional retraining to nocturnal activities, when the brain is not directly engaged, requires a good degree of patience and perseverance, building motivation relies on the skills of the therapist. Motivation assists the development of habituation, which is a function of time (now, later, or in the future). In therapy, feedback must be used constantly, be it visual, auditory, or tactile. Therapy implies self-talk, but a visual reminder or touch stimulus may be needed as well.

Myofunctional therapy alone may be successful in treating mild-to-moderate SDB, but in many children with SDB, the best results are achieved with a combination of patient myofunctional retraining and other therapeutic options, such as adenotonsillectomy, oral appliances, or positive airway pressure (Fig. 15). Although more research is needed to document the effectiveness of OMT in the treatment as well as the prevention of SDB in the pediatric population, the potential benefits of including a myofunctional therapist in a team approach should not be underestimated.

**REFERENCES**

10. Romero CC, Scavone-Junior H, Garib DG, et al. Breastfeeding and non-nutritive sucking patterns related to the prevalence of anterior open bite in...
YOU NOW NEED TO VISIT YOUTUBE TO WATCH THE BRIEF “TONGUE TIE AND SURGERY” VIDEO BY SARAH HORNSBY, RDH, AT https://youtu.be/3TozTfIenWEI.

UPON COMPLETION OF THE “TONGUE TIE AND SURGERY” VIDEO, YOU WILL NEED TO CONTINUE WITH THE ADDITIONAL REQUIREMENTS FOR UNIT 6, WHICH INCLUDE ANOTHER READING, AS WELL AS COMPLETING THE UNIT 6 TEST.
# Management of Infants Presenting with Ankyloglossia

**Date:** June 2018

<table>
<thead>
<tr>
<th>Received ....................... 06/24/18</th>
<th>Scientific Review .............. 07/27/18</th>
<th><strong>Biological Support</strong></th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>Approval</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Provisional Approval</td>
</tr>
<tr>
<td></td>
<td></td>
<td>No Opinion</td>
</tr>
<tr>
<td></td>
<td></td>
<td>No Approval</td>
</tr>
</tbody>
</table>

**Explanation of IAOMT position:** The IAOMT is particularly focused on the impact of oral health on the overall health and development of individuals. The research below discusses the impact of ankyloglossia on the infant and mother. The IAOMT neither promotes nor discourages individual practitioner adoption of the procedures described but does encourage members to evaluate and treat patients with the best care and judgment. This scientific review is provided to help members utilize available research on this subject.

**Name of SR:** Management of infants presenting with ankyloglossia.

**Alternative name(s) of SR:** Efficacy of laser frenectomy on breastfeeding infants with ankyloglossia

**What is this SR related to?**

<table>
<thead>
<tr>
<th>Medicine</th>
<th>Yes</th>
</tr>
</thead>
<tbody>
<tr>
<td>Dentistry</td>
<td>Yes</td>
</tr>
</tbody>
</table>

**This SR is a:** Procedure

**Do you have a vested financial interest in any part of this SR?** No

**Purpose of the SR:** Health care providers and oral medicine physicians have an obligation to assess, treat and/or refer nursing dyads who are having issues during breastfeeding. Our role includes identifying functional issues and anatomical anomalies during the office visit for either mom or baby and to offer education and support by either referral or creating an appropriate integrative team of providers for the dyad. This SR serves to provide concise and research based solutions for a nursing mother and child when presenting with ankyloglossia (tongue tied).

**SR History:** Breastfeeding the neonate is considered to be the optimal form of nutrition and is often met with maternal and infant distress due to ankyloglossia, a short, tight lingual frenum or tongue tie. Successful management of this condition requires the physician/dentist to understand the risks and benefits of the various treatment modalities normally prescribed for the treatment of this condition.

**Briefly describe the SR:** This review characterizes the sequelae of ankyloglossia on breastfeeding and other functions as well as treatment options and desired outcomes. Specifically, it will address the following:

1) Ankyloglossia and its prevalence
2) How breastfeeding is beneficial in terms of
   a. Mother and baby Bonding
   b. Nutrition
   c. Immunological protection
   d. Decreased risk of Auto-immune condition
   e. Decreased risk of malocclusion
3) Assessment tools and diagnosis necessary for Ankyloglossia
4) What are the symptoms and how they range from poor nutrition in the baby and soreness of the
Specifically, by outline if appropriate, describe the SR:

Breastfeeding (BF) the neonate is considered to be the optimal form of nutrition and is often met with maternal and infant distress due to ankyloglossia, a short, tight lingual frenum or tongue tie ("CADTH Rapid Response Reports," 2016; Francis, Krishnaswami, & McPheeters, 2015; Manipon, 2016). A procedure referred to as a frenectomy or frenulectomy may be considered to split or release the tight sublingual frenulum using scissors, scalpel or laser to allow for ideal movement and function ("CADTH Rapid Response Reports," 2016). The etiology of tight frena have been attributed to a genetic condition with a disruption of normalized apoptosis (Veyssiere et al., 2015).

The prevalence of ankyloglossia ranges from 4 to 10 % ("CADTH Rapid Response Reports," 2016; Segal, Stephenson, Dawes, & Feldman, 2007; Veyssiere et al., 2015). The prevalence is low and often times undiagnosed (Tsaousoglou, Topouzelis, Vouros, & Seulean, 2016). Interestingly, the frenectomy procedure has increased 89% from 2004 to 2013 in Canada and 834% in the USA between 1997 and 2012 (Joseph et al., 2016; Walsh, Links, Boss, & Tunkel, 2017).

Optimal breastfeeding provides the preferred nutrition for the infant as well as a sense of well-being for the mother and infant. The infant is dependent upon the mother for non-nutritional bonding as well as nutritional growth and development; therefore, the mother must have appropriate nutrition and behavioral health (Fanaro, 2002; Lau, 2018). The benefits for breastfeeding are extensive, including immunological protection from illnesses and disease through human breast milk (Heinig, 2001). Human milk is responsible assisting in the maturation of the infants immunological system, gut mucosa, intestinal flora and has been shown to reduce exposure to antigens consumed (Andreas, Kampmann, & Mehring Le-Doare, 2015; Jackson & Nazar, 2006; Oddy, 2002). Early breastfeeding termination may be a marker for chronic diseases later in life, such as auto-immune conditions (Jackson & Nazar, 2006). Breastfed babies have potentially enhanced cognition, reduction in the risk of diabetes, obesity, celiac disease, asthma, rhinitis, hay fever, pneumonia, hypertension and allergies (Andreas et al., 2015; Huang et al., 2017; Schack-Nielsen & Michaelsen, 2007; Turck, 2007). Alternatively, bottle fed babies are at increased risk of malocclusion, mouth breathing and dental caries (Brahm & Valdes, 2017). Breastfeeding was shown to provide safeguard against Class 2 malocclusions in both primary and permanent teeth, as well as posterior cross bites (Borrie, 2018).

Breastfeeding benefits for the mother include enhanced bonding between mom and baby, contraction of the uterus, reduction in the risk of breast and ovarian cancers, lactation amenorrhea, economical savings and convenience (Andreas et al., 2015). The World Health Organization recommends breastfeeding babies for at least 2 years but most mothers stop prematurely due to societal pressures, stigmas and other physical barriers (Brockway & Venturato, 2016; Dowling & Brown, 2013).

A healthy latch requires a wide-mouth, flanged lip that creates a seal at the breast, suction created by lowering the tongue and a suck-breathe-swallow pattern (Wiessinger & Miller, 1995). Both breastfed and
bottle fed infants demonstrates a suck-swallow-breathe reflex during the milk extraction process (Sakalidis & Geddes, 2016). Evaluation of peristaltic movement was conducted using submental ultrasound as well as intra-oral vacuum (Geddes, Kent, Mitoulas, & Hartmann, 2008; Geddes et al., 2012).

The body of the tongue is comprised of the genioglossus muscle and the attachment to the floor of the mouth is referred to as the lingual frenulum (Ferrés-Amat, Pastor-Vera, Mareque-Bueno, Prats-Armengol, & Ferrés-Padró, 2016). Short lingual frenula are comprised of type I collagen primarily as well as a high concentration of muscle fibers in the histological mucosal frenum (Martinelli, 2014). Ankyloglossia is the result of a short lingual frenulum or genioglossus and results in limited mobility, such as elevation and protrusion (Ferres-Amat, 2016 Jan).

A variety of assessment tools for classifying lingual and / or labial frenula exist including the Hazelbaker Assessment Tool for Lingual Frenulum Function (HATLFF), Coryllos grading system, Briston Tongue Assessment Tool (BTAT), Infant Breastfeeding Assessment Tool and Kotlow’s free-tongue measurement (Buryk, Bloom, & Shope, 2011; Ferrés-Amat, Pastor-Vera, Rodríguez-Alessi, Mareque-Bueno, & Ferrés-Padró, 2017; Power & Murphy, 2015; Walsh & Tunkel, 2017; Yoon et al., 2017). The two most commonly used are the HATLFF and BTAT (Ferrés-Amat et al., 2017). The HATLFF has been shown to be highly reliable means to assess tongue-tied infants (Amir, James, & Donath, 2006). It is imperative to evaluate and classify lingual restrictions, but also to assess the maxillary labial frenum (Crippa, Paglia, Ferrante, Ottonello, & Angiero, 2016). Diagnosis of a restricted maxillary frenulum is characterized by blanching at the alveolar ridge when the lip is elevated or flanged and considered abnormal if it is abnormally wide or when “no apparent zone of the attached gingiva along the midline or the interdental papilla shifts when the frenum is extended” (Devishee, Gujjiari, & Shubhashini, 2012).

Symptoms for the baby can include poor weight gain, weight loss, dehydration, difficulty latching and early termination of breastfeeding (Manipon, 2016). The two most important symptoms are latch/ suck and nipple pain (Ito, 2014; Wong, Patel, Cohen, & Levi, 2017). When an infant has an anatomical alteration which precludes normalized breastfeeding function, they will compensate to nourish their bodies, which leads to symptoms. A thorough investigation into symptoms experienced by both the infant and mother must be explored before arriving at a diagnosis of ankyloglossia. If the infant is unable to create an appropriate seal due to a tethered lip or vacuum due to a tethered tongue, then the baby compensates, often by increasing work load by “chomping” or attempting to suck more aggressively. This creates quick fatigue and significant pain for the mother. Higher vacuum pressure often results in pain (H. McClellan et al., 2008) and is a cause for early cessation (H. L. McClellan et al., 2012). Without the necessary elements for function, the baby will not extract enough milk, become quickly fatigued and will not gain or possibly loose weight. This is obviously dangerous, can happen quickly and result in failure to thrive (Post, Rupert, & Schulpen, 2010).

In addition to symptoms that the baby may experience, the mother may often experience nipple pain, damaged nipples, mastitis, clogged ducts, poor milk supply and emotional distress. The most common reason for premature breastfeeding cessation is due to maternal pain ("CADTH Rapid Response Reports," 2016; Segal et al., 2007). Extended feeding times are associated with lower milk supply (Geddes et al., 2012). Higher vacuum levels are associated with increased maternal pain and may result in early cessation of breastfeeding (Geddes et al., 2010; H. L. McClellan, Kent, Hepworth, Hartmann, & Geddes, 2015; Perrella, Lai, & Geddes, 2015).

A tethered tongue limits the ability to normally function during breastfeeding in addition to other activities. This results in a range of symptoms including but not limited to difficulty feeding, swallowing, speech, difficulty cleansing the oral cavity of food and debris, gingival recession, open mouth breathing and
crowding of dentition. Patients with ankyloglossia develop symptoms or characteristics that are the direct result of a tethered tongue creating forces on the cranial complex. Measurements of the maxillary and mandibular intercuspal canine distance shows shorter distances in addition to the maxillary molar intercuspal distance, resulting malocclusion (Meenakshi & Jagannathan, 2014; Srinivasan & Chitharanjan, 2013). The severity of ankyloglossia corresponds to the extent of malocclusion, including narrow maxilla, mandibular incisor spacing, open mouth and forward head posture (Vaz & Bai, 2015). Tongue-tie is also a marker for breathing disordered sleep, speech and swallowing dysfunction, sleep breathing disorder, attention issues due to lack of restful sleep and social issues such as eating an ice cream or kissing (Chinnadurai et al., 2015; Reddy, Marudhappan, Devi, & Narang, 2014; Sane et al., 2014; Suter & Bornstein, 2009; Yoon et al., 2017). The general consensus is that the tethered tongue directly effects craniofacial growth and development (Pompeia, Ilinsky, Ortolani, & Faltin, 2017). Lateral cephalometric radiographs conducted in 7-11 year old children with tongue tie showed a more superior and posterior position of the hyoid bone (Ardekani, Tabatabaee, Halvani, Tabatabaee, & Yasaee, 2016). If a child in primary dentition has dental caries on the maxillary incisors, careful evaluation must be given to the maxillary labial frenum as food and debris can accumulate and cleansing the facial surfaces may be inhibited by the maxillary labial frenulum (Kotlow, 2010).

A frenectomy should be considered when there are symptoms presenting with the baby and/or mom, such as difficulty breastfeeding or maternal nipple pain ("CADTH Rapid Response Reports," 2016). The International Board Certified Lactation Consultant (IBCLC) serves an essential role in the integrative team for the nursing dyad; therefore lactation assessment support is encouraged to address the following issues unless the following they can be ruled out and/or addressed by the appropriate provider: insufficient glandular tissue, previous radiation, prior breast surgery, hormonal anomalies, medications that inhibit milk production, inappropriate frequency of feeding the infant, improper latch, mother separation, supplemental feeding, neonatal insufficient oral muscular strength, inadequate weight gain of the infant, not enough soiled diapers, nipple injury, engorgement, plugged ducts, breast mastitis, anomalies of the skin, torticollis, birth disorders, Raynaud’s phenomenon, breast abscess, galactocele, yeast infection, bloody nipple discharge and excess of milk supply. (Giugliani, 2004; Riskin et al., 2014; Spencer, 2017). The IBCLC’s evaluation includes a thorough evaluation of breastfeeding mechanics and anatomical structures. Evaluation of the latch, positioning and attachment may be improved, which may avoid the need for an invasive procedure (Douglas, 2016; H. McClellan et al., 2008; Royal College of Midwives. Learning; Wattis, Kam, & Douglas, 2017). The IBCLC offers support both before and after the procedure, unlike the provider who performs the release, which is considered primarily procedural (Berg, 1990; Henry & Hayman, 2014).

An integrative team should be constructed for the patient prior to the release, including a lactation consultant and a bodyworker, such as a myofunctional therapist, oral mycologist, cranial sacral therapist or chiropractor. Due to advances in technology, both lactation consultants and bodyworkers are able to work with patients remotely. Studies show that stimulation of suck with myofunctional therapy prior to and after the procedure result in improved surgical outcomes and reduced the risk of scar formation (Ferrés-Amat, Pastor-Vera, Rodriguez-Alessi, Mareque-Bueno, & Ferrés-Padró, 2016). Excellent outcomes have been reported with myofunctional therapy beginning one week prior to the release in patients presenting with delayed speech and tongue tie (Ferres-Amat, 2016 Jan). Outcomes were beneficial after the release when myofunctional therapy and lactation support were included in the comprehensive care plan (Ferrés-Amat, Pastor-Vera, Rodriguez-Alessi, et al., 2016).

For older children who are developing speech, a speech and language pathologist (SLP) referral and assessment and therapy is recommended prior to and after the procedure to ensure accurate diagnosis and appropriate support (Veyssiere et al., 2015). The speech therapist offers support to evaluate alterations in
speech as well as rehabilitation of oral structures, tongue exercises and speech therapy before and after the procedure (Khan, Sharma, & Sharma, 2017).

Overall, the frenectomy procedure is considered to be safe, low risk with no major complications reported (Brookes & Bowley, 2014; Ito, 2014). Most common risks include bleeding, pain and fussiness. Reattachment leading to a subsequent release occurs at a rate of 0.003-13% (Brookes & Bowley, 2014) and is more common with posterior tongue tie. In patients with Pierre-Robin Sequence, frenectomies are contraindicated due to the risk of airway obstruction (Genther, Skinner, Bailey, Capone, & Byrne, 2015). Although very rare, a mucocele of the minor salivary glands, subacute massive edema of the submandibular region, infection of sublingual hematoma and hypovolemic shock have been reported (Isaiah & Pereira, 2013; Santos, Filho, Piva, & Khalil Karam, 2012; Sirinoğlu, Certel, & Akgün, 2013; Tracy, Gomez, Overton, & McClain, 2017).

A major risk factor that should be thoughtfully considered in the neonate is bleeding. Vitamin K Deficiency Bleeding (VKDB), or hemorrhagic disease of the newborn, has been reported in two Nigerian frenectomy cases as well as two frenectomy cases by researchers at the University of North Carolina. The infants experiences hypovolemic shock and underwent cardiopulmonary resuscitation for the US babies, blood transfusions, IV fluids and antibiotics were given to the Nigerian babies (Opara, Gabriel-Job, & Opara, 2012; Tracy et al., 2017).

Vitamin K dependent clotting factors include FII, FVII, FIX and FX, which are diminished and inactive in the newborn without the presence of Vitamin K (Chalmers, 2004). Definitive diagnosis is dependent on the analysis of these factors and is remedied by Vitamin K injection. VKDB is characterized by early VKDB which occurs during the first 24 hours of life, classic VKDB which happens 24 hours to 7 days of life and late VKDB, occurring 2 to 24 weeks after birth (Sutor, von Kries, Cornelissen, McNinch, & Andrew, 1999; Zipursky, 1999). Early VKDB is associated with absorption of medicines that interfere with Vitamin K metabolism (Chalmers, 2004). Breastfed babies are at higher risk, as much as 90% with males at twice the risk, of classical and late VKDB due to less Vitamin K in mother’s milk, miniscule amounts cross the placenta and malabsorption (Chalmers, 2004) (Sutor, Dagres, & Niederhoff, 1995; Zipursky, 1999). Other predisposing conditions include patients with cholestasis and cystic fibrosis. Infants who have not received the intramuscular form of Vitamin K are 81 times higher risk of forming VKDB (McNinch & Tripp, 1991). Mothers taking anti-convulsants (seizure), tuberculosis medicines or anti-coagulants, result in bleeding in the newborn and are contraindicated (Zipursky, 1999). It should be noted that while Hemophilia A (FVIII) and B (FIX) are the most common inherited bleeding disorders, approximately 30% are due to a novel mutation and present with no family history (Chalmers, 2004).

EMLA is a topical anesthetic that is often used for numbing the tissues of infants prior to frenectomy release. EMLA 5% is comprised of lidocaine and prilocaine and is safe when used on neonates below the age of 3 months (Brisman, Ljung, Otterbom, Larsson, & Andrèsson, 1998; Essink-Tjebbes, Hekster, Liem, & van Dongen, 1999). Topical benzocaine is contraindicated in children under the age of 2 due to the risk of methemaglobinemia (Dahshan & Donovan, 2006; FDA, 05/23/2018; Ip, Patel, Chi, Shah-Manek, & Lau, 2018; So & Farrington, 2008). Methemoglobinemia may result in agitation, hypoxemia, cyanosis, lethargy and vomiting (Chung, Batra, Itzkevitch, Boruchov, & Baldauf, 2010; Dahshan & Donovan, 2006). Post-operatively, pain management is important and parents may choose homeopathy, other non drug based remedies or Tylenol.

Literature reviews revealed that the efficacy of frenectomies are beneficial for breastfeeding infants with ankyloglossia and mothers report improvement in symptoms (Francis et al., 2015; Ito, 2014; Riskin et al., 2014). Patients show improved outcomes when performed at an early age as opposed to waiting (Riskin et
Objective improvements in weight gain, breastfeeding function, longer breastfeeding times and reductions in maternal pain have been reported as well (Brookes & Bowley, 2014). Not only do maternal symptoms improve post frenectomy, so does suck-breathe-swallow pattern in infants (R. L. Martinelli, Marchesan, Gusmão, Honorio, & Berretin-Felix, 2015). Significant improvements in breastfeeding was noted immediately and continued one month after the procedure and was noted more in anterior as opposed to posterior tongue tie (Ghaheri, Cole, Fausel, Chuop, & Mace, 2017). Many other studies showed enhanced breastfeeding, decreased pain, improved latch after a frenectomy was performed (Ghaheri et al., 2017; J, 2016; Muldoon, Gallagher, McGuinness, & Smith, 2017; Pransky, Lago, & Hong, 2015; Sharma & Jayaraj, 2015). While the long-term effects of frenectomy of breastfeeding are beneficial, the procedure does not always eliminated symptoms (Dollberg, Marom, & Botzer, 2014).

Studies have shown that breastfeeding is not improved or partially improved when only the tongue is released when the infant has a concomitant lip tie (Wiessinger & Miller, 1995). The maxillary labial frenum should be evaluated at the initial oral evaluation for restriction. This site is often not evaluated, diagnosed and consequently, left untreated and plays a critical role to proper function (Crippa et al., 2016).

A review of the literature revealed an increased number of articles on tongue tie and recent publication of systematic reviews and randomized controlled trials. However, the author suggests more research on tongue tie and treatment outcomes to evaluate efficacy (Bin-Nun, Kasirer, & Mimouni, 2017). Health care providers should encourage initiation, duration normalized breastfeeding and provide support to nursing dyads as well as assist in the removal of obstacles when possible (Brockway & Venturato, 2016; Sriraman & Kellams, 2016).

Manufacturer(s), distributor, or publisher: N/A

Scientific Literature:


Copyright (c) 2016 Canadian Agency for Drugs and Technologies in Health.


Legal Aspects of this SR:

Please write five (5) multiple choice questions related to your SR (for Accreditation test). Only ask questions related to the content of your SR:

1. Symptoms of ankyloglossia in breastfeeding babies and mothers may include a. Pain with latch, b. Infant weight loss, c. Difficulty latching, d. All the above. **D.**
2. Uncontrolled bleeding is a risk factor a. When a breastfeeding mother is taking anti-tuberculosis medicines, b. When a breastfeeding mother is taking seizure medicines, c. The infant has not had a Vitamin K injection, d. All the above. **D.**
3. Ankyloglossia may lead to a. malocclusion, b. narrow arch, c. wide arch, d. both a and b **D.**
4. The integrative team for the nursing dyad may include a. DDS or MD, b. IBCLC (International Board Certified Lactation Consultant), c. Bodyworker, d. All the above. **D.**
5. A healthy latch is comprised of a. Wide latch, b. Lip flange, c. Tongue vacuum, d. All the above. **D.**
6. The World Health Organization recommends breastfeeding at least until the age of a. 6 months, b. 1 year c. 2 years. **C.**

Applicant Name: Crystal “Robyn” Abramczyk, DDS, MS, FAGD, NMD, IBDM, AIAOMT  
Office Phone: 972-772-7645  
Office FAX: 469-402-2003  
Home Phone: 214-901-4942  
Home FAX:  
e-mail: robynseaman@yahoo.com  
IAOMT Member #: 1719  
IAOMT Chapter: USA

The IAOMT and Mankind Thanks You!
SUPPLEMENTARY RESOURCES FOR
IAOMT BIOLOGICAL DENTAL HYGIENE CERTIFICATION
Unit 6: Sleep-Disordered Breathing, Myofunctional Therapy, and Ankyloglossia

You are required to read this two-page document. However, you do not need to click on any of the links in this document because you are NOT required to read the articles in their entirety or visit any of the organization websites listed below. These materials are being provided to you as additional resources about the topics presented in this unit.

SELECTED RESEARCH ARTICLES:

- “Results of this study indicated that the prevalence of SDB [sleep-disordered breathing] was high and highly associated with malocclusion; anterior open bite and posterior crossbite are risk factors for SDB.”
- [Click here to read the entire article](#).

- “This article is a review of research findings regarding the many behavior problems associated with disordered breathing in addition to their significance when compared to symptom free controls. Also discussed are those cephalometric and dental measures that can be helpful in assessing whether a child has a serious problem.”
- [Click here to read the entire article](#).

- “We propose the use of tongue range of motion ratio as an initial screening tool to assess for restrictions in tongue mobility. ‘Functional’ ankyloglossia can thus be defined and treatment effects followed objectively by using the proposed grading scale: grade 1: tongue range of motion ratio is >80%, grade 2 50–80%, grade 3 < 50%, grade 4 < 25%.”
- [Click here to read the entire article](#).

- “After excluding the influence of repeated apnea and hypoxia, simple snoring was still significantly associated with MetS [metabolic syndrome], especially in women. Furthermore, the associations were more obvious for hypertension among males and for abdominal obesity and hyper-TG among females. In addition to OSA, simple snoring also should be valued.”
- [Click here to read the entire article](#).
ORGANIZATIONS WITH ADDITIONAL RESOURCES:

**Academy of Orofacial and Myofunctional Therapy (AOMT)**
The mission of the AOMT is to offer the best foundation for health professionals to learn Orofacial Myofunctional Therapy and bring its benefits to those who need it.

**American Academy for Oral Systemic Health (AAOSH)**
AAOSH is an organization of healthcare leaders and health professionals dedicated to expanding awareness of the relationship between oral health and whole body health.

**American Academy of Physiological Medicine & Dentistry (AAPMD)**
AAPMD is dedicated to providing its professional membership tools and ability to recognize the role and importance of optimal airway physiology and sleep in the areas of health, development, performance, and function.

**American Speech-Language-Hearing Association (ASHA)**
ASHA is the national professional, scientific, and credentialing association for audiologists; speech-language pathologists; speech, language, and hearing scientists; audiology and speech-language pathology support personnel; and students.

**The Foundation for Airway Health (FAH)**
FAH champions the recognition, diagnosis and treatment of airway related disorders through collaboration, awareness, research, and education.

**International Association of Orofacial Myology (IAOM)**
The IAOM is the oldest and largest orofacial myology certification organization, and consists of partnership of professionals, educators, and colleagues.

**International Affiliation of Tongue and Lip Tie Professionals (IATP)**
IATP is a multi-disciplinary group of healthcare professionals who advocate for research, education, and integrated clinical practice to improve the lives of all people affected by oral restrictions.
YOU NOW NEED TO TAKE THE UNIT 6 TEST AT https://www.cvent.com/d/mhq1j5.

IT IS AN OPEN BOOK TEST AND CONSISTS OF 15 QUESTIONS. YOUR SCORE WILL BE AUTOMATICALLY CALCULATED AND SENT TO YOU VIA EMAIL.

UPON COMPLETION OF THE UNIT 6 TEST, YOU WILL NEED TO CONTINUE WITH THE REST OF THE CERTIFICATION REQUIREMENTS.

ACCESS THE MATERIALS FOR YOUR NEXT UNIT BY USING THE LINK TO THE IAOMT COURSE PDFs AT www.iaomt.org/bdhc-materials/.