

Obstructive sleep apnea - a review

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Abstract

Sleep related problems affect large group of people of all ages around world. One of the most common disorders is Obstructive Sleep Apnea (OSA) characterized by repetitive, complete or partial collapse of upper airway during sleep. Due to complexity of disease and associated medical conditions, a broad spectrum of clinicians play important roles in treatment of OSA. Evaluation of OSA is best performed using a multidisciplinary team approach.

This article aims to review the exhaustive current literature for relevant information regarding terminology, pathophysiology, prevalence and treatment including conservative and surgical.

Keywords : Obstructive sleep apnea, Epworth sleepiness scale, Continuous Positive Airway Pressure (CPAP) Therapy, Polysomnography, Osteotomy, Palatal implants

Introduction

Obstructive Sleep Apnea (OSA) was first reported in the medical literature in 1965, although William Osler recognized the stigmata in the early 1900s and named the trait "Pickwickian Syndrome" after a character in a Charles Dickens novel.(1) It is defined as a presence of five or more apneas/hypopneas per hour of sleep with daytime symptoms. It is a common sleep-related breathing disorder of major public health importance(2)

In adults, apnea is defined as cessation of airflow for greater than 10 seconds. Hypopnea is defined as 50% or greater decrease in airflow, often accompanied by hypoxemia or arousal. OSA is also characterized by cessation of respiration during sleep, secondary to obstruction of upper airway(3).

OSA is characterized by recurrent episodes of partial or complete upper airway collapse at the end of expiratory phase during sleep. The collapse is due to reduction in or complete cessation of

airflow despite ongoing inspiratory efforts. The events are caused by multiple factors like high intra-luminal pressure, obesity, decreased dilator and increased constrictor activity, low tracheal traction, etc (4).

Review of Literature

Prevalence

It is estimated that the prevalence of OSA in general is 1-6% (5). Community based studies has shown the male to female ratio for OSA is in the range of 2:1 or 3:1 (6). In an epidemiological study by Young et. al., it was estimated that 2% of adult women and 4% of adult men fulfilled diagnostic criteria of OSA i.e. apnea-hypopnea index (AHI) ≥ 5 and daytime hypersomnolence (7).

Differences in gender specific OSA prevalence is not uniformly the same across all ages. These differences were not observed in the pediatric and adolescent age groups. Difference in the middle age is attributed to sex hormones which play an important role in the natural history of OSA. (8) Menopause is a risk factor for sleep-disordered breathing while prevalence of OSA in elderly is similar in men and women (9). Finally the influence of other risk factors like alcohol consumption and smoking has been associated with higher prevalence of OSA in men (10).

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However, the male predominance reported for OSA in epidemiological studies is more marked in the clinical setting, with estimates male/female ratio as high as 8-10:1 (11).

Pathophysiology

The upper airway is composed of bony structures and soft tissues and it can be divided into 4 sections – nasopharynx (from nasal turbinates to hard palate), velopharynx (from hard palate to tip of uvula), oropharynx (from tip of uvula to

tip of epiglottis) and hypopharynx (from tip of epiglottis to level of vocal cords) (12). The most common site of upper airway collapse in OSA is velopharynx. The collapse usually extends to other sites; however, it can also begin at other locations within the upper airway (13).

Upper airway anatomy is an important consideration in understanding the pathophysiology of OSA given the relationship between its structure and function. (12)

Table 1 : Factors predisposing to collapse of the upper airway and the development of OSA

- Restriction in size of bony compartment
 - Mandibular hypoplasia
 - Maxillary hypoplasia
- Increase in soft tissue volume
 - Deposition of fat around upper airway (e.g., in obesity)
 - Macroglossia
 - Enlargement of soft palate -Thickening of lateral pharyngeal walls
 - Adenotonsillar enlargement
 - Pharyngeal inflammation and edema
- Increase in pharyngeal compliance
- Decrease in pharyngeal dilator muscle activity
 - Impairment of mechanoreceptor sensitivity
 - Impairment of upper airway neuromuscular reflexes
 - Impairment of strength & endurance of pharyngeal dilator muscles
- Decrease in lung volume
- Instability of ventilatory control » Increase in surface tension
 - Hormonal factors
 - Presence of testosterone (e.g., male gender or testosterone replacement)
 - Absence of progesterone (e.g., menopause)
- Endocrine disorders (eg, hypothyroidism or acromegaly)

Table 2 : Risk Factors

Unmodifiable	Potentially modifiable	Associated conditions (examples)
Increasing age	Obesity	Marfan's syndrome
Male gender	Neck or visceral fat distribution	Hypothyroidism
Ethnicity	Upper airway soft tissue abnormalities	Down's syndrome
Menopause	Craniofacial abnormalities	Acromegaly
Genetics	Alcohol consumption	

Symptoms and Signs

SYMPTOMS

- Snoring
- Witnessed Apneas
- Excessive daytime sleepiness
- Nocturnal choking
- Unrefreshed sleep
- Poor sleep quality
- Insomnia

SIGNS

- Obesity
- Increased neck circumference
- Increased waist circumference
- Retrognathia
- Maxillary constriction
- Increased overjet
- Increased overbite

SYMPTOMS

- Morning headaches
- Impaired concentration
- Impaired memory
- Nocturia
- Impotence
- Anxiety and depression
- Esophageal reflux

SIGNS

- Tonsillar hypertrophy
- Macroglossia
- Oropharyngeal narrowing
- Soft palate edema and erythema
- Nasal obstruction
- Hypertension

Evaluation and Diagnosis

Epworth Sleepiness Scale (ESS)

The ESS developed by Murray Johns at the Epworth Sleep Center, Richmond, Victoria, Australia is an excellent measure of the patient's general level of daytime sleepiness. Patients simply score their likelihood of falling asleep in 8 different situations. (14)

Name: _____	Date _____
Your age (Yrs): _____	Your Sex (Male=M, Female=F): _____
How likely are you to doze off or fall asleep in the following situations, in contrast to feeling just tired? This refers to your usual way of life in recent times. Even if you have not done some of these things recently, try to work out how they would have affected you. Use the following scale to choose the most appropriate number for each situation:	
0 = would never doze	
1 = slight chance of dozing	
2 = moderate chance of dozing	
3 = high chance of dozing	
<u>Situation</u>	<u>Chance of dozing (0-3)</u>
Sitting and reading	_____
Watching TV	_____
Sitting inactive in a public place (e.g., a theatre or a meeting)	_____
As a passenger in a car for an hour without a break	_____
Lying down to rest in the afternoon when circumstances permit	_____
Sitting and talking to someone	_____
Sitting quietly after a lunch without alcohol	_____
In a car, while stopped for a few minutes in traffic	_____

Fig. 1 : Questionnaire for Epworth Sleepiness Scale (14).

ESS - A score of greater than 10 is consistent with excessive daytime sleepiness and a score of greater than 16 is indicative of a high level of daytime sleepiness (14).

There is evidence that a questionnaire based scale as simple and brief as ESS can give valid measurements of sleep propensity in adults. That it can distinguish patients who simply snore from those with even mild OSA is evidence for the sensitivity of the ESS. (15)

Apnea-Hypopnea Index (AHI)

The primary measure of sleep-disordered breathing is the AHI, which is the number of apneas and hypopneas per hour of sleep. OSA is classified as mild (AHI 5-15), moderate (AHI 15-30) severe (AHI > 30) (16).

Polysomnography (PSG)

The nocturnal polysomnography has been the objective gold standard for modern diagnosis of OSA (17).

PSG evaluates sleep-disordered breathing, sleep architecture, and oxygen desaturation. A typical 8-hour nocturnal laboratory PSG involves measurement of

physiologic parameters including electroencephalogram, electrooculography, chin movements and leg movements via electromyography. Electrocardiography, heart rate, respiratory effort, chest wall movement, abdominal wall movement, airflow, and oxygen saturations are also monitored. As these physiologic parameters are scored, a sleep technologist documents body position. The recordings and scoring data are interpreted by a physician to diagnose OSA (17).

Treatment Protocol

Conservative

Conservative approaches involving weight loss, smoking cessation and alcohol moderation are encouraged and useful in selected patients with OSA. Weight loss has shown to be beneficial in reducing the severity of OSA (18). Major weight loss such as may occur following bariatric surgery can result in resolution of OSA. Positional therapy (e.g. using a backpack that prevents the subject from sleeping in the supine position) is most beneficial when OSA predominantly occurs supine in position. However, studies suggest only a partial response to such therapy (19).

A large range of pharmacologic approaches to treating OSA have been explored over many years, but these have been shown to be minimally or not at all effective (20).

Non-Surgical

1. Continuous Positive Airway Pressure (CPAP) –

CPAP therapy for OSA was developed in the early 1980s and subsequently became the initial line of treatment for symptomatic OSA. It still continues to be used as a conservative therapy. (21)

Sleep induces collapse and reduces the diameter of the floppy-toned pharyngeal musculature, as a result of negative transmural pressure. CPAP counteracts this change by pneumatically splinting the upper airway via the application of a positive pressure across the airway walls. This prevents the narrowing (hypopnea) or complete collapse (apnea) of the breathing conduit during sleep. (22)

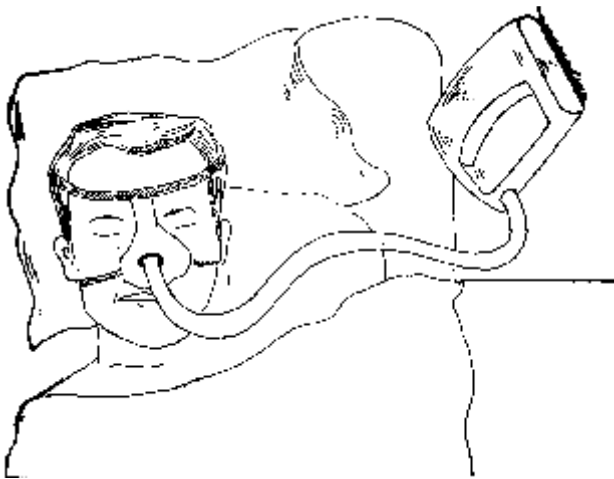


Fig. 2: A diagrammatic representation of a patient receiving CPAP therapy.

Table 3: Relative and absolute contraindications to the use of continuous positive airway pressure (CPAP) (23)

Clinical situation	Rationale for avoiding CPAP
Tracheo-oesophageal fistula	Ineffective because of loss of pressure to the gut
Upper airway abnormalities (cleft palate, choanal atresia)	Technically difficult/ impossible, often traumatic
Congenital diaphragmatic hernia	Leads to intestinal distension and clinical deterioration
Absent/poor respiratory effort	Ineffective carbon dioxide removal
Extreme prematurity	Lack of reliable evidence in the most preterm infants

1. Oral Appliances

Oral Appliance (OA) therapy for snoring, obstructive sleep apnea or for both is simple and cost-effective. It may be indicated in patients who are unable to tolerate CPAP or poor surgical risks.

Their efficacy is variable and act by a) increase in airway space by stabilizing the mandible in an anterior position, b) advancement of the tongue or soft palate and c) possibly a change in genioglossus muscle activity. The appliances should be used during sleep for life and must be comfortable for the patient (24).

Oral Appliances therapy falls into two main categories: those which hold the tongue forward (24) (Tongue retaining device - TRD) (12) and the ones which reposition the mandible forward (Mandible repositioning appliances - MRA) during sleep (24). MRA's are the most evaluated type of oral appliances (12).

Table 4: Food and Drug Administration (FDA) Approved Oral Appliances for the Treatment of Obstructive Sleep Apnea (25)

Appliances	Manufacturer
Adjustable PM Positioner	Jonathan Parker, DDS
Triation (EMA-T)	
Elastic Mandibular Advancement	Frantz Design, Inc.
Elastomeric Sleep Appliance	Village Park Orthodontics
Herbst	Orthodontics, SUNY at Buffalo
Equalizer Airway Device	Sleep Renewal Inc.
NAPA	Great Lakes Orthodontics Ltd.
Klearway	Great Lakes Orthodontics Ltd.
OSAP	Snorefree, Inc.
PM Positioner	Jonathan A Parker, DDS
Sleep-In Bone Screw System	.Influence Inc.
Silencer	Silent Knights Ventures, Inc
SNOAR Open Airway Appliance	Kent J Toone, DDS
Thornton Airway Appliance	W. Keith Thornton, DDS

Surgical

A. Non Invasive

1. Injection Snoreplasty -

Soft Palate sclerotherapy (Injection Snoreplasty) is a popular technique as a primary treatment of palatal snoring because of its comparative advantages over other anti-snoring procedures. It is very simple during a routine office visit, minimally painful, is highly effective and is very inexpensive. After topical anesthesia, midline soft palate is

injected submucosally with a small amount of sodium tetradecyl sulfate-a well described, safe sclerotherapy agent. Controlled fibrosis eliminates or significantly diminishes palatal flutter snoring (26).

2. Palatal Implants -

Relatively new procedure, the Pillar palatal implant system, consisting of a delivery system and an implant, is designed to reduce airway collapse and obstruction at the level of the soft palate by placement of 3 woven implants. The implants are flexible enough to allow full soft palate

function but stiff enough to provide structural support. In addition, the porosity of the implant surface allows tissue ingrowth to anchor the implant, and the surface texture encourages formation of a fibrotic capsule that extends and connects the 3 implants, thereby further stiffening the soft palate. The implant is a segment of braided polyethylene terephthalate 18 mm long and 2 mm in diameter. Polyethylene terephthalate has been widely used in human implants and stimulates a fibrotic response (27).

A. More Invasive

Table 5: Range of surgical techniques for OSA in adults (28)

Surgical site	E.g. of surgical techniques available	Advantages	Potential difficulties
Nasal	Septoplasty	Adjunct for better tolerance of CPAP and lower pressures	Septal perforation can adversely affect future CPAP use.
	Septorhinoplasty		
	Turbinate reduction		
	Endoscopic sinus surgery	Improve nasal airway	Requires expert assessment of nasal symptoms/examination to identify pathology
Oropharyngeal surgery	Tonsillectomy	Prevent retropalatal restriction	Pain
	Uvulo-Palatopharyngoplasty	Combined with other procedures in multi-level approach	May affect future CPAP tolerance
	Laser assisted Uvulo-palatoplasty		Absence of long-term data in OSA.
	Radiofrequency thermo-therapy (Soft palate)		
Hypopharyng-eal surgery	Radiofrequency thermotherapy (Tongue base)	Combined with other procedures in multilevel approach	Absence of long-term data in OSA.
	Hyoid suspension	Directly deals with anatomical abnormality	Morbidity associated includes dysphagia, odynophagia, dysphonia and aspiration. Robotic approach is resource intensive and restricted to specialised centres. May require 'covering' tracheostomy in post-op period
	Midline glossectomy		
	Epiglottic wedge resection		
Maxillofacial	Maxillo-mandibular advancement	Highly effective	Highly Invasive Need for prolonged fluid diet. Velopharyngeal incompetence.
Tracheal	Tracheostomy	Bypasses obstructive segment Highly efficient	Invasive Technically difficult to perform in obese individuals
Bariatric surgery	Roux-en-Y gastric bypass	Objective improvement demonstrated-decrease CPAP requirements	Maintenance in weight loss required for benefits
	Vertical banded gastroplasty	Further health benefits	May not be deemed curative

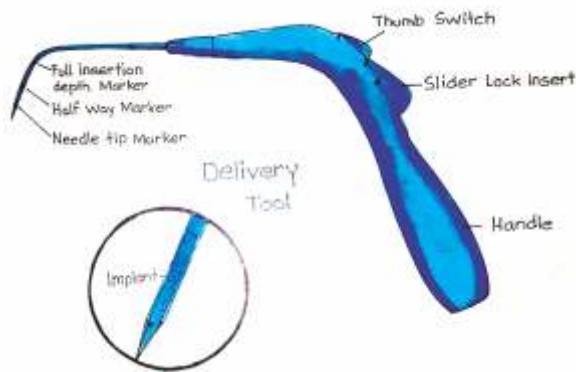


Fig. 3 : Delivery Tool for the placement of palatal implants.

Colin Sullivan, in 1981 showed CPAP as the gold standard in the treatment of OSA. There are only minimal side effects with this mode of treatment. However, despite its high efficacy, patients frequently cannot tolerate its usage every night for life and thus long-term acceptance has been found to be low. (29)

MRDs for OSA patients is considered as another form of noninvasive therapy. A review of the literature showed that these MRDs are more acceptable than CPAP and have reasonable success rates when used in mild-to-moderate OSA. However, the long compliance rates are still not good and there are complications associated with long term usage of the MRDs, such as temporo-mandibular joint problems and changes in the occlusion. (30)

When the non-surgical therapies for OSA fail or are unacceptable to the patients, surgical options are considered. The first surgical treatment for OSA was tracheotomy in 1969 by Kuhol. Previously, in 1964, Ikematsu started treating snoring with a soft palate procedure known as uvulopalatopharyngoplasty (UPPP). Following that, Fujita published results on UPPP in OSA. However, Sher's review in 1996 showed the success rate to be close to 40%. Since then, a number of procedures were developed to treat OSA. They are all designed to improve the

posterior airway from the nasal aperture to the larynx. These procedures are shown in Table 5. (31)

Most of the soft-tissue procedures only augment one part of the posterior airway and thus were limited in their

success rate when used individually. Derived from Moore's concept, two principles of therapy were developed. The first principle states that the entire upper airway is affected, especially in moderate and severe OSA. The second principle states that the more severe the disorder, the more aggressive the surgical therapy has to be to achieve success. Modern surgical reasoning suggests that severe OSA affects the entire airway and that multilevel procedures are necessary to achieve good results. Riley et al found the success rate for UPPP, genioglossus advancement (GGA), and hyoid suspension (HS) to be 61% and Friedman, et al achieved a 41% success for UPPP. (31)

Hard tissue surgical procedures have shown better success rates but are more tedious and may have higher morbidity. Maxillomandibular advancement

(MMA) which is modeled after conventional orthognathic surgery has achieved remarkable success rates of 97-100%. Therefore, it is important to examine the patients carefully before deciding on the most appropriate surgical procedures. (32)

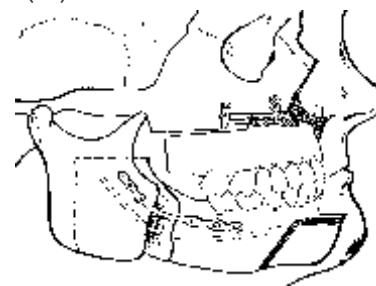


Fig. 4 : A diagrammatic representation showing typical osteotomy cuts with fixation by mini plates after Maxillo-mandibular advancement.

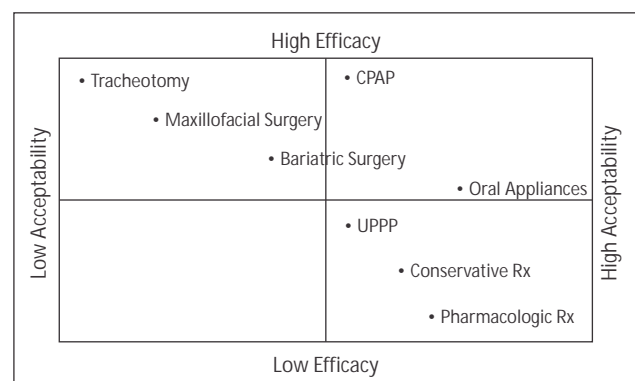


Fig. 4 Positioning of various OSA treatment classes in a matrix comparing efficacy and acceptability of treatments. (33)

Conclusion

Obstructive Sleep Apnea is a relatively common condition that predisposes the patient to physical harm, significant social discord and poor quality of life. Conservative and surgical techniques are components of multimodal algorithm of OSA which focuses on modifying skeletal or soft tissue anomaly. It is

important to understand intended goal of procedure, some procedures have been shown to independently improve the quality of life as well as acceptance of therapy. Emphasis on modifying the treatment procedure is key factor to achieve best and long term effects. Team effort and collaborated approach gives the optimal result for patient.

References

1. Bagheri SC, Bell RB, Khan HA; Current Therapy in Oral and Maxillofacial Surgery. Elsevier; 2011.
2. Malhotra A, White DP. Obstructive Sleep Apnea. *Lancet* 2002; 360:237-45.
3. Lurie A. Obstructive sleep apnea in adults: epidemiology, clinical presentation and treatment options. *Adv Cardiol* 2011;46:1-42.
4. Schafer, T., 2006. *Physiology of Breathing During Sleep*. Karger, Basel, pp. 21–28.
5. Marin J, Gascon J, Carrizo S, Gispert J. Prevalence of sleep apnoea syndrome in the Spanish adult population. *Int J Epidemiol* 1997;26:381–6.
6. Young T. Analytic epidemiology studies of sleep-disordered breathing: what explains the gender difference in sleep disordered breathing? *Sleep* 1993;16:S1–2.
7. Young T, Palta M, Dempsey J, Skatrud J, Weber S, Badr S. The occurrence of sleep-disordered-breathing among middle aged adults. *N Engl J Med* 1993;328:1230–5.
8. Sanchez-Armengol A, Fuentes-Pradera A, Capote-Gil F, et al. Sleep-related breathing disorders in adolescents aged 12 to 16 years. *Clinical and polygraphic findings*. *Chest* 2001;119:1393–400.
9. Ancoli-Israel S, Kripke D, Klaber M. Morbidity, mortality and sleep-disordered breathing in a community dwelling elderly. *Sleep* 1996;19:277–82.
10. Young T, Peppard PE, Gottlieb DJ. Epidemiology of obstructive sleep apnea. A population health perspective. *Am J Respir Crit Care Med* 2002;165:277–82.
11. Redline S, Kump K, Tishler PV, Browner I, Ferrette V. Gender differences in sleep-disordered breathing in a community based sample. *Am J Crit Care Med* 1994;149:722–6.
12. Lavigne GJ, Cistulli PA, Smith MT. *Sleep Medicine for Dentists: A Practical Overview*. Canada: Quintessence Publishing Co, Inc; 2009.
13. Ryan CM, Bradley TD. Pathogenesis of obstructive sleep apnea. *J Appl Physiol* 99:2440, 2005.
14. Johns MW. Reliability and factor analysis of the of the Epworth Sleepiness Scale; *Sleep* 1992;15(4):376-81.
15. MW, Johns. A new method for measuring daytime symptoms: The Epworth Sleepiness Scale. *Sleep*, 14(6):540-545.
16. Sleep-related breathing disorders in adults: recommendations for syndrome definition and measurement techniques in clinical research. The report of an American Academy of Sleep Medicine Task Force. *Sleep* 1999;22(5):667-89.
17. Iber C, Ancoli-Israel S, Chesson A, et al. The AASM manual for the scoring of sleep and associated events: rules, terminology and technical specifications. Westchester (IL): American Academy of Sleep Medicine; 2007.
18. Veasey SC, Guilleminault C, Strohl KP, Sanders MH, Ballard RD, Magalang UJ. Medical therapy for obstructive sleep apnea. A review by the Medical Therapy For Obstructive Sleep Apnea Task Force of the Standards of Practice Committee of the American Academy of Sleep Medicine. *Sleep* 2006;29:1036-44.
19. Jokić R, Klimaszewski A, Crossley M, Sridhar G, Fitzpatrick MF. Positional treatment vs continuous positive airway pressure in patients with positional obstructive sleep apnea syndrome. *Chest* 1999;115:771-781.
20. Buchanan PR, Grunstein RR. Neuropharmacology of obstructive sleep apnea. In: Pandi-Perumal SR, Monti JM (eds). *Clinical Pharmacology of Sleep*. Basel, Switzerland: Birkhauser; 2006:21-41.
21. Sullivan CE, Issa FG, Berthon-Jones M, Eves L. Reversal of obstructive sleep apnea by continuous positive airway pressure applied through the nares. *Lancet* 1981;1(8225):862-865.
22. Remmers JE, deGroot WJ, Sauerland EK, Anch AM. Pathogenesis of upper airway occlusion during sleep. *J Appl Physiol* 1978;44:931-938.
23. Cheema IU, Ahluwalia JS. The rational use of nasal continuous positive airway. *Current Paediatrics* (2003) 13, 190-195.
24. Ferguson KA, Cartwright R, Rogers R, et al. Oral appliances for snoring and obstructive sleep apnea: a review. *Sleep* 2006;29(2):244-62.
25. Miloro M, Ghali GE, Larsen PE, Waite PD, Petersons Principles of Oral and Maxillofacial Surgery. 2nd edition. Ontario: BC Decker Inc; 2004.
26. Brietzke SE, Mair EA. Operative techniques in Otolaryngology. *Head and Neck Surgery*. Vol. 13, Issue 3, Sept. 2002, 185-87.
27. Nordgard S, Hein G, Stene BK. One-year results: Palatal implants for the treatment, *Otolaryngology–Head and Neck Surgery* (2007) 136, 818-822.
28. Kotecha BT, Hall AC. Role of surgery in adult obstructive sleep apnoea. *Sleep Medicine Reviews*, Volume 18, Issue 5, October 2014, 405–413.
29. Kribbs NB, Pack AI, Kline LR. Objective measurement of patterns of nasal CPAP use by patients with obstructive sleep apnea. *Am Rev Respir Dis* 147:887-895, 1993.
30. V, Hoffstein. Review of oral appliances for treatment of sleep-disordered breathing. *Sleep Breath* 11:1-22, 2007.
31. Lye KW, Deatherage JR. Surgical Procedures for the Treatment of Obstructive Sleep Apnea. *Semin Orthod* 2009;15:94-98.
32. Prinsell, JR. Maxillomandibular advancement surgery in a site-specific treatment approach for obstructive sleep apnea syndrome. *J Oral Maxillofac Surg* 47:1256-1261, 1989.
33. Cistulli PA, Grunstein RR. Medical devices for the diagnosis and management of obstructive sleep apnea. *Expert Rev Med Devices* 2005;2:749-763.