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Review Article

# DIAGNOSIS AND MANAGEMENT OF OBSTRUCTIVE SLEEP APNEA

Ankit Mehta[1]*, Padma Ariga[2]

[1]Post Graduate, Department of Prosthodontics, Saveetha Dental College, Poonamallee, Chennai, India

[2]Professor, Department of Prosthodontics, Saveetha Dental College, Poonamallee, Chennai, India

*Corresponding Author Email: mehtaankiit06@gmail.com

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**ABSTRACT**

Sleep is defined as a naturally recurring state characterized by altered consciousness, relatively inhibited sensory activity and inhibition of nearly all voluntary muscles. Poor sleep quality results in excessive daytime sleepiness, impaired health status, depressive symptoms and poor quality of life. Upper airway sleep disorders (UASD) are becoming commonly recognized by medical and dental communities. Snoring is a mild form of these disorders. Obstructive Sleep Apnea (OSA) is a severe yet under-diagnosed sleep related breathing disorder. It is characterized by repetitive complete or partial occlusion of the upper airway, resulting in apnea, hypopnea, oxygen desaturation and sleep fragmentation. OSA can lead to a series of systemic, dental and psychological problems if left untreated. Efforts have been made to pin-point the symptoms to effectively evaluate this disorder and to efficiently treat it. This review gives a brief description on the evaluation criteria, associated systemic complications and series of invasive and non invasive treatment options for management and improvement of individuals affected with OSA.

**Keywords:** Obstructive sleep apnea, Upper airway sleep disorder, Sleep disorders and Apnea

## INTRODUCTION

Sleep disorder breathing (SDB) describes a group of disorders characterized by abnormalities of respiratory pattern during sleep. This includes simple snoring, Upper Airway Resistance Syndrome (UARS) and Obstructive Sleep Apnea (OSA). Simple snoring is a common complaint affecting 30 % of the adults and is a sign of airway obstruction. UARS is characterized by resistance to breathing during sleep with day-time sleepiness and excessive fatigue as primary symptoms. OSA is characterized by repetitive upper airway collapse/ narrowing during sleep which results due to inadequate motor tone of tongue/ airway dilator muscles, micrognathia, retrognathia, macroglossia and soft tissue hyperplasia[1]. Studies have shown a strong association of OSA with cardiovascular disease, hypertension, stroke, arrhythmias and insulin resistance. Patients with OSA have 30 % more risk of heart attack than those unaffected[3]. Repetitive apnea exposes the cardiovascular system to a cascade of intermittent hypoxia, exaggerated negative intra-thoracic pressure, which in turn surges the sympathetic nervous system activity (SNA) and blood pressure and frequent awakening, all of which may have adverse cardiovascular consequences[4].

### Epidemiology

Prevalence rate of OSA from the Wisconsin sleep cohort study in patients aged 30-60 years was 9-24 % for males and 4-9 % for women[3]. Prevalence of OSA is similar in Caucasians and Asians, indicating that OSA is not only common in developing but also in developed countries. However, the disease prevalence is higher in the subgroups with overweight or obese subjects and elderly people. Inter-ethnic studies suggest that African-American ethnicity may be at significant risk for developing OSA. The increased prevalence of OSA among American, Indian and Hispanic adults, and increased severity among Pacific Islanders and Maoris, were mainly explained by the increased obesity indices[2]. The primary risk factor for OSA includes the male gender, those over the age of 40, overweight individuals, and BMI in particular is the strongest risk factor. Twenty six percent of patients with BMI greater than 30 % and 33 % of those with BMI greater than 40 % are at a moderate risk. Neck circumference of 15.7 (40 cm) may have a greater sensitivity and specificity than BMI in predicting OSA, regardless of the person's sex. Incidence of OSA among male and female are 3:1 prior to menopause. There is at least three fold increases in the risk of OSA among post menopausal women compared to premenopausal women.

### Pathophysiology

Patients with OSA generally have narrow pharynx which is contributed by two major factors- (i) craniofacial structural abnormalities and (ii) larger soft tissue mass or abnormal tissue deposits which can also increase extra-luminal tissue pressure and lowers the threshold for airway collapse. Additional anatomical factors include the sleeping posture which strongly influences the collapsibility of airway by the gravitational force which causes retropulsion of tongue and soft palate while lying supine, thus generating increased positive pressure and narrowing the airway[3]. OSA contributes to the development or progression of Heart Failure (HF), although a possibility does exist that HF might contribute

to causation of OSA. This could be due to the flow of accumulated fluid in the leg while upright during day and flows into the neck when recumbent during sleep there by, causing distention of the neck vein and/ or oedema of the peri-pharyngeal soft tissue which increases the peri-pharyngeal tissue pressure, predisposing to pharyngeal obstruction[4].

**Evaluation**

Sleep apnea is usually recognised as a problem by family members who witness the apneic episodes or by a primary care doctor by the individual's risk factors and symptoms. Clinical symptoms can include excessive daytime sleepiness (EDS) that usually begins during quiet activities, daytime fatigue, feeling tired despite a full night's sleep, morning headaches, personality and mood changes, dry or sore throat, gastroesophageal reflux and sexual dysfunction. Diagnosis of OSA is based on the evaluation of clinical symptoms and risk factors with screening questionnaires, as well as a physical examination. Epsworth Sleepiness Scale (ESS) is a questionnaire used to help determine how frequently the patient is likely to doze off in 8 frequently encountered situations. ESS is a subjective self assessment measure and may be inaccurate for a number of reasons. Therefore, if a patient has multiple risk factors for sleep apnea, the individual should be sent for further evaluations if there is a suspicion of sleep apnea despite a low ESS. Berlin questionnaire is another effective screening tool which addresses snoring behaviour, EDS/ fatigue and history of obesity or hypertension with sensitivity regards to high risk patient being 86 %[4]. STOP BANG questionnaire term refers to mnemonic that represents 8 most common risk factors: Snoring, Tiredness, Observed apneas, elevated blood Pressure, BMI, Age, Neck circumference and Gender. The patient receives a point for each positive risk factor. A score of 3 or more represents a higher likelihood of OSA[5]. Physical examination includes obesity, enlarged neck circumference, and/ or structural cranio-facial bony abnormalities, hypothyroidism and acromegaly. Mallampati score is used routinely by sleep physicians to evaluate the risk of OSA. The classification provides a score between 1 and 4 based on the anatomic appearance of the airway seen when an individual opens his mouth[6]. Polysomnography (PSG) is a formal sleep evaluation test to quantify the amount of time spent in various stages of sleep during the night and to clinically document the relevant events such as cardiopulmonary abnormalities and/ or changes in sleep stages. PSG includes a limited multi–channel recording of an individual's electroencephalography (EEG) to assess the sleep architecture, sleep stages and arousals. In addition, surface electromyography of the chin and all the four limbs along with electro-oculogram, airflow, pulse oximetry, respiratory effort, electrocardiographic (ECG) tracing, body position and snoring are accumulated. Portable polysomnographic device gives an advantage to perform the study overnight at individual's home and is cost effective. The main limitation lies in the fact that it does not have an EEG component, therefore its might show greater inability to assess the individual's sleep architecture and arousals patterns[7,8].

**Diagnosis**

Apnea is defined as a more than 90 % reduction in tidal volume lasting for 10 seconds. Hypopnea is a reduction in tidal volume of 50 % to 90 %, lasting for 10 seconds accompanied by a 3 % decrease in oxyhemoglobin saturation ($SaO_2$) or terminated by arousal from sleep. OSA is generally defined as the presence of more than 5 episodes of apnea or hypopnea per hour of sleep (i.e. Apnea Hypopnea Index [AHI]) and when accompanied by either hyper somnolence or at least 2 episodes of choking or gasping during sleep, recurrent awakening, un refreshing sleep, daytime fatigue, impaired concentration or memory, is called as an OSA syndrome[4]. AHI is an index used to assess the severity of sleep apnea based on the total number of apnea and hypopnea occurring per hour of sleep. Severity of the disease can be stratified using AHI. An AHI score of 5-15 is classified as mild, 15-30 is considered moderate and greater than 30 is considered severe[9]. OSA is usually confirmed by overnight polysomnography or portable cardio respiratory monitor devices used to study sleep architecture, cardiac rhythm, $SaO_2$, airflow and thoraco-abdominal movements.

**Systemic Complications**

OSA has been widely recognized as an important risk factor for hypertension, cardiovascular disease (CVD), stroke and death. The underlying pathogenesis is particularly intermittent hypoxia which triggers the sympathetic nerve activity, systemic inflammation and oxidative stress, causing endothelial cell dysfunction, systemic inflammation, metabolic dysregulation and coagulopathy. The absolute key factor however remains unknown[9].

**Hypertension**

Cross sectional analyses suggests OSA as an identifiable and independent cause of hypertension and frequent blood pressure monitoring is recommended for such individuals. Oxygen desaturation during apnea/hypopnea increases sympathetic activity secondary to micro arousals at night, nocturnal fluctuations in catecholamines are some of the prominent factors in the causation of hypertension[10-12]. Dependent and independent vasodilatation due to endothelial dysfunction may also result in raised peripheral vascular resistance and consequently, hypertension[7].

**Coronary artery disease**

Circulatory physiology is greatly affected by change in sleep pattern, OSA related hypoxia, hypercapnia, blood pressure surges due to sympathetic over activation and the acute imbalance of vasoactive hormones. These changes not only provoke acute coronary syndromes but also, their persistence ultimately leads to chronic consequences such as heart failure[13].

**Cerebrovascular diseases**

Sleep disordered breathing is highly prevalent amongst stroke patients and is regarded as a poor prognostic marker due to the associated sub-total/total paralysis of facial and pharyngeal muscles[14,15]. Nevertheless, further data is needed to determine whether OSA is related to

cerebrovascular morbidity independent of other vascular risk factors.

**Heart failure**

Patients with heart failure per se have a high prevalence of OSA. This might be attributed to oedema of neck soft tissues which makes the pharyngeal tissue prone to collapse, thereby leading to further tightening of the airway[16,17].

**Cardiac arrhythmias**

Abnormal cardiac pattern of sudden deaths amongst OSA could possibly be explained due to higher incidences of life threatening ventricular arrhythmias amongst patients with sleep disorders[18]. OSA not only increases the prevalence of atrial fibrillation (AF) but also predicts pre-discharge AF after surgery and recurrent post ablation[16,19-21].

**Chronic kidney disease (CKD)**

High prevalence of OSA and its influence on cardiovascular events have been reported among individuals with end-stage renal disease[22,23]. Prevalence and severity of OSA among non dialysis CKD patients is considerably higher than those in the general population, in which 1 of 5 adults suffered mild OSA and significant increase in the risk of OSA was associated in individuals with decreased GFR[24]. Narrowing of pharyngeal cross section by 12 % less than that in the normal individuals is one of the factors associated with decreased GFR[6].

**Management**

**Continues positive air pressure (CPAP)**

The most common and effective treatment for OSA is continuous positive airway pressure (CPAP), which decreases no of apnea and hypopnea episodes and reduces the amount of oxygen desaturation over night[26]. It consists of a mask containing a tube which connects to the oxygen device and fits over the nose, supplying a steady stream of air and pressure to prevent the tissues from collapsing during sleep[27]. Randomized clinical trials have demonstrated improvement in many health outcomes like subjective sleepiness, quality of life (QOL), blood pressure and long term treatment which may reduce the incidence of cardiovascular events, at least in patients with severe OSA[28-31]. Recent meta-analysis conclusively states that CPAP reduces subjective and objective daytime sleepiness when compared with pill placebo or placebo CPAP[29,32]. Health outcome in patients with moderate to severe OSA were similar after treatment with CPAP and Mandible Advancement Devices (MAD). However, it is recommended that MAD treatment should be considered in patients with mild to moderate OSA or in individuals who have failed or refuse CPAP treatment[33].

**Dental Devices**

American Academy of Sleep Medicine recommends dental device for patients with mild to moderate obstructive sleep apnea who are not appropriate candidates for CPAP or who have not been helped by it. Oral devices are of two basic configurations, the tongue retaining device (TRD) and the MAD which generally works by directly or indirectly preventing the tongue from approaching the posterior wall of the pharynx and hence maintaining the airway space[27,34]. Although the two appliances have similar treatment effects of reducing AHI; the higher response rate, over all acceptance and compliance of MAD suggests that it is a superior treatment for OSA in the clinical set up[35,36]. Although, dental devices significantly reduce apnea in patients sleeping on their back or stomach, it does not work well in individuals lying on their side[27].

**Surgical Procedures**

The aim of OSA surgery is to eliminate the airway collapse and to reduce airway resistance during sleep without causing impairment to the normal function of the upper airway and associated structures. General indications for surgery include moderate to severe OSA, excessive daytime sleepiness (even when the AHI is ≤ 20/h), OSA with co-morbid conditions (e.g. arrhythmias, hypertension), with anatomic airway abnormalities and failure of medical OSA management[37]. Relative contraindications to surgery include morbid obesity (except for bariatric surgery and tracheotomy), severe or unstable cardiopulmonary disease, active alcohol/ illicit drug abuse, older age, unstable psychological problems, or unrealistic expectations from surgical therapy[38]. Various surgical procedures are now available to increase the posterior airspace and to successfully treat OSA in CPAP intolerant patients. However, treatment outcome is considered successful when an AHI less than 20 and a reduction in AHI of 50 % or more is attained after surgery[47].

**Tracheotomy**

Kuhlo *et al* in 1969 effectively treated OSA by means of a tracheotomy by by-passing the upper airway[42]. Tracheotomy is effective at preventing arrhythmias, reducing pulmonary artery pressure and improving hypertension and diabetes in patients with OSA along with reported resolution of nocturnal symptoms and daytime sleepiness[41-43]. Unfortunately, tracheotomy has several problems including patient dissatisfaction (e.g. psychosocial aspects), post-operative complications (e.g. wound infection, tissue necrosis, bleeding), recurrent bronchitis, granulation tissue and stomal stenosis[44-47]. Permanent tracheotomy is used in highly selected cases with severe OSA who are intolerant of CPAP unlike temporary tracheotomy which is used before other OSA procedures (e.g. uvulopalatopharyngoplasty, bariatric surgery) to protect the airway, particularly in morbidly obese subjects[48].

**Uvulopalatopharynoplasty (UPPP)**

In 1980 Fujita and Conway *et al* reported UPPP as a technique to enlarge the oropharyngeal airway lumen by excising redundant tissues from the soft palate, tonsillar pillars and uvula[49,50]. It reduces the risk of nasopharyngeal incompetence and is associated with less postoperative pain. It is however contraindicated in patients with excessively long or bulky soft palate[51,52]. UPPP lowers the positive airway pressure requirements, thus improving CPAP compliance in selected patients[53]. Early postoperative complications include wound dehiscence, haemorrhage, infection and transient velopharyngeal

incompetence (nasal regurgitation and hypernasal speech)[54]. Late postoperative complications include pharyngeal discomfort (dryness, tightness), postnasal secretions, dysphasia, inability to initiate swallowing, odynophagia, nasopharyngeal stenosis, taste and speech disturbances, tongue numbness and rarely permanent velopharyngeal incompetence[55,56].

**Laser assisted palatoplasty (LAUP)**

Laser assisted palatoplasty is a procedure similar to uvulopalatopharyngoplasty, but it uses a $CO_2$ laser to shape the soft palate and is an effective surgical technique for snoring. However, it has limited OSA efficacy[57]. Two randomized trials of LAUP found no significant change in the AHI and daytime sleepiness (measured by the Epworth sleepiness scale) between surgery and control groups[58]. LAUP is not approved by the American Academy of Sleep Medicine to treat OSA[59]. In addition, common complication includes early postoperative pharyngeal oedema, with up to 59 % complaining of persistent side effects after LAUP[60,61].

**Tonsillectomy**

Tonsillectomy is one of the most common surgical procedures in and is considered as a first-line therapy for children's with OSA[62,63]. Complete resection of the tonsils with adenoidectomy is preferred over partial intra capsular tonsillectomy (tonsillotomy) as it reduces postoperative morbidity (pain), but postoperative objective measures of efficacy (AHI reduction) are lacking[65-67]. Tonsillectomy improves quality of life parameters in children with OSA, with improvements in the behaviour scores and sleep disturbances[68-71].

**Radiofrequency Ablation (RFA)**

RFA can be considered as a treatment in patients with mild to moderate obstructive sleep apnea who cannot tolerate or who are unwilling to adhere to positive airway pressure therapy, or in whom oral appliance have been considered and found ineffective or undesirable[72]. Somnoplasty involves directed radiofrequency energy to ablate and reduce soft tissues of the palate thereby, decreases snoring by scar-induced stabilization of the soft palate[73-75]. A randomized placebo-controlled trial in patients with mild OSA found no statistically significant improvement in the AHI or symptoms after somnoplasty[76].

**Pillar Palatal Implants**

This procedure involves inserting matchstick size rigid polyester implants via a hollow needle delivery tool into the soft palate which improves snoring by stiffening the soft palate, but their effect on OSA is less clear and the long-term benefits on OSA are unknown[77-81].

**Biatric surgery**

Surgically induced weight loss was first performed in 1967 and is safe, results in marked and sustained weight loss, and is associated with improved mortality compared to conventional weight-loss strategies[82-84]. Surgically induced weight loss improves the AHI. However, more than 50 % of bariatric recipients with preoperative OSA have a residual disease despite weight loss[85].

## CONCLUSION

OSA is a common complication which affects the population worldwide. The prevalence and consequence of OSA is likely to increase in years to come. However, its diagnosis and treatment is still not a common approach especially in developing countries. Medical communities face many hurdles regarding the development of adequate early screening and appropriate treatment. It is well known fact that effects of OSA is far reaching, and early detection/treatment will be beneficial to individual patients, as well as cost effective public health measure to reduce morbidity and mortality. Physicians of all specialities should screen for the presence of sleep disturbance and consider referral to a sleep specialist when indicated.

## REFERENCES


1. John G Park, Kannan Ramar and Eric J Olson. Updates on Definition, consequences and management of Obstructive Sleep Apnea. Concise review for clinicians 2001; 86(6): 549-555.
2. Jamie CM Lam, SK Sharma and Bing Lam. Obstructive sleep apnea: Definition, Epidemiology and natural history. Indian J Med Res 2010; 165-170.
3. Matthew L Ho and Brass SD. Obstructive Sleep Apnea. Neurology International 2011; 3: e15.
4. Netzer NC, Stoohs RA, Netzer CM, Clark K, Strohl KP. Using the Berlin Questionnaire to identify patients at risk for the sleep apnea syndrome. Ann Intern Med 1999; 131: 485-91. http://dx.doi.org/10.7326/0003-4819-131-7-199910050-00002
5. Chung F, R Subramayam, P Liao, E Sasaki, C Sharpiro and Y Sun. STOP questionnaire: A tool to screen patients for obstructive sleep apnea. Anaesthesiology 2008; 108: 812-21. http://dx.doi.org/10.1097/ALN.0b013e31816d91b5
6. Nuckton T, Glidden DV, Browner WS, Claman DM. Physical examination: Mallampati score as an independent predictor of obstructive sleep apnea. Sleep 2006; 29: 903-8.
7. Series F, Marc I, Corenia T, La Forge J. Utility of nocturnal home oximetry for case finding in patients with suspected sleep apnea hyopnea syndrome. Ann Intern Med 1993; 119: 449-53. http://dx.doi.org/10.7326/0003-4819-119-6-199309150-00001
8. Mc Nicholas WT, Levy P. Portable monitoring in sleep apnea: the way forward. Eur Resp J 2011; 37: 749-51. http://dx.doi.org/10.1183/09031936.00180410
9. Mehmood Butt, Girish Dwivedi, Omer Khair, Gregory YH Lip. Obstructive sleep apnea and cardiovascular disease. International Journal of Cardiology 2009.
10. Fletch EC, Miller J, Schaaf JW, Fletcher JG. Urinary catecholamine's before and after tracheotomy in patients with obstructive sleep apnea and hypertension. Sleep 1987; 14: 35-44.
11. Baruzzi A, Riva R, Cirignotta F, Zucconi M, Capepeli M, Lugaresi E. Atrail natriuretic peptide and catecholamine's in obstructive sleep apnea syndrome. Sleep 1991; 103: 722-7.
12. Carlson JT, Hedner J, Elam M, Ejnell H, Sellgren J, Wallin BG. Augmented resting sympathetic activity in awake patients with obstructive sleep apnea. Chest 1993; 103: 1763-8. http://dx.doi.org/10.1378/chest.103.6.1763
13. Hanly P, Sasson Z, Zuberi N, Lunn K. ST segment depression during sleep in obstructive sleep apnea. Am J Cardiol 1993; 71: 1341-5. http://dx.doi.org/10.1016/0002-9149(93)90552-N
14. Good DC, Henkle JQ, Gelber D, Welsh J, Verhulst S. Sleep disorder breathing and poor functional outcome after stroke. Stoke 1996; 27: 252-9.
15. Sahlin C, Sandberg O, Gustafson Y *et al*. Obstructive sleep apnea is a risk factor for death in patients with stroke – a 10 year follows up. Arch Intern Med 2008; 168: 297 -301. http://dx.doi.org/10.1001/archinternmed.2007.70
16. Javaheri S, Parker TJ, Liming JD *et al*. Sleep apnea in 81 ambulatory male patients with stable heart failure- types and their prevalence's, consequence and presentations. Circulation 1998; 97: 2154-9. http://dx.doi.org/10.1161/01.CIR.97.21.2154
17. Shepard Jr JW, Pevernagie DA, Stanson AW, Daniels BK, Sheedy PF. Effects of changes in central venous pressure on upper airway size in patients with obstructive sleep apnea. Am J Respir Crit Care Med 1996; 153: 250-4. http://dx.doi.org/10.1164/ajrccm.153.1.8542124

18. Harbison H, O Reilly P, Mc Nicholas WT. Cardiac rhythm disturbances in the obstructive sleep apnea syndrome effects of nasal continuous positive airway pressure therapy. Chest 2000; 118: 591-5. http://dx.doi.org/10.1378/chest.118.3.591
19. Sin DD, Fitzgerald F, Parker JD, Newton G, Floras JS, Bradley TD. Risk factor for central and obstructive sleep apnea in 450 men and women with congestive cardiac failure. Am J Respire Crit Care Med 1999; 160: 1101-6. http://dx.doi.org/10.1164/ajrccm.160.4.9903020
20. Moee T, Gullsby S, Rabben T. Sleep disordered breathing: a novel predictor of atrail fibrillation after coronary artery bypass surgery. Coron Artery Dis 1996; 7: 475-8. http://dx.doi.org/10.1097/00019501-199606000-00011
21. Jongnarangsin K, Chugh A, Good E *et al*. Body mass index, obstructive sleep apnea and outcomes of catheter ablation of atrial fibrillation. J Cardiovascular Electophysol 2008; 19: 668-72. http://dx.doi.org/10.1111/j.1540-8167.2008.01118.x
22. Beecroft JM, Pierratos A, Hanly PJ. Clinical presentation of obstructive sleep apnea in patients with end stage renal disease. J Clin Sleep Med 2009; 22: 1190-1197.
23. Markou N, Kanakaki M, Myrianthefs P, Hadjiyanakos D, Vlassopoulos D, Damianos A, Siamopoulos K, Vasiliou M. Konstantopoulos S. Sleep-disordered breathing in nondialyzed patients with chronic renal failure. Lung 2006; 184: 43-49. http://dx.doi.org/10.1007/s00408-005-2563-2
24. Young T, Peppard PE, Gottlieb DJ. Epidemiology of obstructive sleep apnea: A population health perspective. Am J Respir Crit Care Med 2002; 165: 1217-1239. http://dx.doi.org/10.1164/rccm.2109080
25. Beecroft JM, Pierratos CT Chan, PA Mc Farlane, PJ Hanly. Pharyngeal narrowing in end stage renal disease: implication for obstructive sleep apnea. Eur Respire 2007; 30: 965-971. http://dx.doi.org/10.1183/09031936.00161906
26. Lianne M. A Effect of Continuous Positive Airway Pressure on Fatigue and Sleepiness in Patients with Obstructive Sleep Apnea: Data from a Randomized Controlled Trial. Sleep 2011; 34: 122-6.
27. Dr Sitaram Wagle, Dr Preeti Kalai, Dr Harendra Shahi, Dr Kamal Kishore Agarwal, Dr Pankaj Jain. Sleep apnoea and its management: A Review. International journal of Enhanced Research in Medicine and Dental Care 2014; 1(3).
28. Mc Daid C, Duree KH, Griffin SC, Weatherly HL, Stradling JR, Davies RJ, Sculpher MJ, Westwood ME. A systematic review of continuous positive airway pressure for obstructive sleep apnea hypopnea syndrome. Sleep Med Rev 2009; 13: 427-436. http://dx.doi.org/10.1016/j.smrv.2009.02.004
29. Giles TL, Lasserson TJ, Smith BH, White J, Wright J, Cates CJ. Continuous positive airway pressure for obstructive sleep apnoea in adults. Cochrane Database Syst Rev 2006; 3: CD001106.
30. Bazzano LA, Khan Z, Reynolds K, He J. Effect of nocturnal nasal continuous positive airway pressure on blood pressure in obstructive sleep apnea. Hypertension 2007; 50: 417-423. http://dx.doi.org/10.1161/HYPERTENSIONAHA.106.085175
31. Antonopoulos CN, Sergentanis TN, Daskalopoulou SS, Petridou ET. Nasal continuous positive airway pressure treatment for obstructive sleep apnea, road traffic accidents and driving simulator performance: a meta-analysis 2011; 15: 301-310.
32. Patel SR, White DP, Malhotra A, Stanchina ML, Ayas NT. Continuous positive airway pressure therapy for treating a diverse population with obstructive sleep apnea: results of a meta-analysis. Arch Intern Med 2003; 163: 565-71. http://dx.doi.org/10.1001/archinte.163.5.565
33. Phillips CL, Grunstein RR, Darendeliler MA, Mihailidou AS, Srinivasan VK, Yee BJ, Marks GB, Cistulli PA. Health outcomes of continuous positive airway pressure versus oral appliance treatment for obstructive sleep apnea. American Journal of Respiratory and Critical Care Medicine 2013; 187(8): 879-887. http://dx.doi.org/10.1164/rccm.201212-2223OC
34. Lt Col B Jayan, Brig BNBM Prasad, Col RK Dhiman. Role of Oral Appliance in the Management of Sleep Disorders. Medical Journal Armed Forces India 2009; 65(2).
35. Deane SA, Cistulli PA, Ng AT, Zeng B, Petocz P, Darendeliler MA. Comparison of Mandibular Advancement Splint and Tongue Stabilizing Device in Obstructive Sleep Apnea: A Randomized Controlled Trial. Sleep 2009; 32(5): 648 -653.
36. Jon Erik Holty, Guilleminault C. Maxillomandibular advancement for the treatment of obstructive sleep apnea: A systematic review and meta-analysis. Sleep Medicine Reviews 2012; 14: 287–297. http://dx.doi.org/10.1016/j.smrv.2009.11.003
37. Troell RJ, Riley RW, Powell NB, Li K. Surgical management of the hypopharyngeal airway in sleep disordered breathing. Otolaryngology Clin North Am 1998; 31(6): 979–1012. http://dx.doi.org/10.1016/S0030-6665(05)70102-X
38. Powell NB, Riley RW, Guilleminault C, Murcia GN. Obstructive sleep apnea, continuous positive airway pressure and surgery. Arch Otolaryngology Head Neck Surg 1998; 99(4): 362–9.
39. Li KK. Surgical management of obstructive sleep apnea. Clin Chest Med 2003; 24(2): 365–70. http://dx.doi.org/10.1016/S0272-5231(03)00016-9
40. Guilleminault C, Simmons FB, Motta J, Cummiskey J, Rosekind M, Schroeder JS, Dement WC. Obstructive sleep apnea syndrome and tracheostomy. Long-term follow-up experience. Arch Intern Med 1981; 141(8): 985–8. http://dx.doi.org/10.1001/archinte.141.8.985
41. Lugarese E, Coccagna G, Mantovani M, Brignani E. Effects of tracheostomy in two cases of hypersomnia with periodic breathing. J Neurol Neurosurg Psychiatr 1973; 36(1): 15–26. http://dx.doi.org/10.1136/jnnp.36.1.15
42. Weitzman ED, Pollack CP, Borowiecki B. Hypersomnia-sleep apnea due to micrognathia. Reversal by tracheoplasty, Arch Neurol 1978; 35(6): 392–5. http://dx.doi.org/10.1001/archneur.1978.00500300066013
43. Bhimaraj A, Haviliqi N, Ramanchandran S. Rapid reduction of antihypertensive medications and insulin requirements after tracheostomy in a patient with severe obstructive sleep apnea syndrome. J Clin Sleep Med 2007; 3(3): 297–9.
44. Conway WA, Victor CD, Maqulliquan DJ Jr, Fujito S, Zorick FJ. Adverse effects of tracheostomy for sleep apnea. The Journal of the American Medical Association 1981; 246(4): 347–50. http://dx.doi.org/10.1001/jama.246.4.347
45. Thatcher GW, Maisel RH. The long-term evaluation of tracheostomy in the management of severe obstructive sleep apnea. Laryngoscope 2003; 113(2): 201–4. http://dx.doi.org/10.1097/00005537-200302000-00001
46. Fedok FG, Strauss M, Houck JR, Cadieux RJ, Kales A. Further clinical experience with the silicone tracheal cannula in obstructive sleep apnea. Otolaryngol Head Neck Surg 1987; 97(3): 313–8.
47. Gross ND, Cohen JI, Andersen PE, Wax MK. Defatting tracheotomy in morbidly obese patients. Laryngoscope 2002; 112(11): 1940–4. http://dx.doi.org/10.1097/00005537-200211000-00006
48. Campanini A, De Vito A, Frassineti S, Vicini C. Temporary tracheotomy in the surgical treatment of obstructive sleep apnea syndrome: personal experience Acta Otorhinolaryngol Ital 2003; 23(6): 474–8.
49. Fujita S, Conway N, Zorick F, Roth T. Surgical correction of anatomic abnormalities in obstructive sleep apnea syndrome: uvulopalatopharyngoplasty. Otolaryngol Head Neck Surg 1981; 89(6): 923–34.
50. Conway W. Uvulo-palato-pharyngoplasty in treatment of upper airway sleep apnea. Am Rev Respir Dis 1980; 121(Suppl): 121.
51. Li HY, Li KK, Cheng NH, Wang CJ, Wang PC, Liao YF. Three-dimensional computed tomography and polysomnography findings after extended uvulopalatal flap surgery for obstructive sleep apnea. Am J Otolaryngol 2005; 26(1): 7–11. http://dx.doi.org/10.1016/j.amjoto.2004.06.006
52. Li HY, Chen NH, Shu YH, Wang PC. Changes in quality of life and respiratory disturbance after extended uvulopalatal flap surgery in patients with obstructive sleep apnea. Arch Otolaryngol Head Neck Surg 2004; 130(2): 195–200. http://dx.doi.org/10.1001/archotol.130.2.195
53. Chandrashekariah R, Shaman Z, Auckley D. Impact of upper airway surgery on CPAP compliance in difficult-to-manage obstructive sleep apnea. Arch Otolaryngol Head Neck Surg 2008; 134(9): 926–30. http://dx.doi.org/10.1001/archotol.134.9.926
54. Fairbanks DN. Uvulopalatopharyngoplasty complications and avoidance strategies. Otolaryngol Head Neck Surg 1990; 102(3): 239–45.
55. Jaghagen EL, Berggren D, Dahlgvist A, Isberg A. Prediction and risk of dysphagia after uvulopalatopharyngoplasty and uvulopalatoplasty. Acta Otolaryngol 2004; 124(10): 1197 203. http://dx.doi.org/10.1080/00016480410017954
56. Lysdahl M, Haraldsson PO. Uvulopalatopharyngoplasty versus laser uvulopalatoplasty: prospective long-term follow-up of self-reported symptoms. Acta Otolaryngol 2002; 122(7): 7527. http://dx.doi.org/10.1080/003655402/000028035
57. Verse T, Pisig W. Meta-analysis of laser-assisted uvulopalatopharyngoplasty. What is clinically relevant up to now;

Laryngorhinootologie 2000; 79(5): 273–84. http://dx.doi.org/10.1055/s-2000-8805
58. Franklin KA, Anttila H, Axelsson S, Gislanon T, Massita P, Myhre KI, Rehnqvist N. Effects and side-effects of surgery for snoring and obstructive sleep apnea–a systematic review 2009; 32(1): 27–36.
59. Morgenthaler TI, Aurona RN, Brown T, Zak R, Alessi C, Boehlecke B, Chesson AL Jr, Friedman L, Kapur V, Maganti R, Owens J, Pancer J, Swick TJ. Practice parameters for the use of autotitrating continuous positive airway pressure devices for titrating pressures and treating adult patients with obstructive sleep apnea syndrome. An American Academy of Sleep Medicine report. Sleep 2008; 25(2): 143–7.
60. Walker RP, Gopalsami C. Laser-assisted uvulopalatoplasty: postoperative complications. Laryngoscope 1996; 106(7): 834–8. http://dx.doi.org/10.1097/00005537-199607000-00009
61. Terris DJ, Clerk AA, Norbash AM, Troell RJ. Characterization of postoperative oedema following laser-assisted uvulopalatoplasty using MRI and polysomnography: implications for the outpatient treatment of obstructive sleep apnea syndrome. Laryngoscope 1996; 106(2 Pt 1): 124–8. http://dx.doi.org/10.1097/00005537-199602000-00002
62. Bluestone CD. Current indications for tonsillectomy and adenoidectomy. Ann Otol Rhinol Laryngol Suppl 1992; 155: 58–64.
63. Rosenfeld RM, Green RP, Green RP. Tonsillectomy and adenoidectomy: changing trends. Ann Otol Rhinol Laryngol 1990; 99(3 Pt 1): 187–91.
64. Waters KA, Cheng AT. Adenotonsillectomy in the context of obstructive sleep apnoea. Paediatr Respir Rev 2009; 10(1): 25–31. http://dx.doi.org/10.1016/j.prrv.2008.10.002
65. Krishna P, La Page MJ, Hughes LF, Lin SY. Current practice patterns in tonsillectomy and perioperative care. Int J Pediatr Otorhinolaryngol 2004; 68(6): 779–84. http://dx.doi.org/10.1016/j.ijporl.2004.01.010
66. Celenk F, Bayazit YA, Yilmaz M, Kemaloglu YK, Uygur K, Ceylan A, Korkuyu E. Tonsillar re growth following partial tonsillectomy with radiofrequency. Int J Pediatr Otorhinolaryngol 2008; 72(1): 19–22. http://dx.doi.org/10.1016/j.ijporl.2007.09.007
67. Vlastos IM, Parpounas K, Economides J, Helmis G, Koudoumnakis E, Houlakis M. Tonsillectomy versus tonsillotomy performed with scissors in children with tonsillar hypertrophy. Int J Pediatr Otorhinolaryngol 2008; 72(6): 857–63. http://dx.doi.org/10.1016/j.ijporl.2008.02.015
68. Ye J, Liu H, Zhang G, *et al*. Postoperative respiratory complications of adenotonsillectomy for obstructive sleep apnea syndrome in older children: prevalence, risk factors, and impact on clinical outcome. J Otolaryngol Head Neck Surg 2009; 38(1): 49–58.
69. Lima Junior JM, Silva VC, Freitas MR. Long term results in the life quality of children with obstructive sleep disorders submitted to adenoidectomy/adenotonsillectomy. Braz J Otorhinolaryngol 2008; 74(5): 718–24.
70. Goldstein NA, Fatima M, Campbell TF, *et al*. Child behaviour and quality of life before and after tonsillectomy and adenoidectomy. Arch Otolaryngol Head Neck Surg 2002; 128(7): 770–5. http://dx.doi.org/10.1001/archotol.128.7.770
71. Mitchell RB, Kelly J. Behavioural changes in children with mild sleep-disordered breathing or obstructive sleep apnea after adenotonsillectomy. Laryngoscope 2007; 117(9): 1685–8. http://dx.doi.org/10.1097/MLG.0b013e318093edd7
72. R Nisha Aurora, Casey KR, Kristo D, Auerbach S, Bista SR, Chowdhuri S, Karippot A, Lamm C, Ramar K. Practice Parameters for the surgical modifications of the upper airway for obstructive sleep apnea in adults. Sleep 2010; 33(10): 1408-1413.
73. Li KK, Powell NB, Riley RW, *et al*. Radiofrequency volumetric tissue reduction for treatment of turbinate hypertrophy: a pilot study. Otolaryngol Head Neck Surg 1998; 119(6): 569–73. http://dx.doi.org/10.1016/S0194-5998(98)70013-0
74. Li KK, Powell NB, Riley RW, *et al*. Radiofrequency volumetric reduction of the palate: an extended follow-up study. Otolaryngol Head Neck Surg 2000; 122(3): 410–4. http://dx.doi.org/10.1067/mhn.2000.102183
75. Coleman SC, Smith TL. Midline radiofrequency tissue reduction of the palate for bothersome snoring and sleep-disordered breathing: a clinical trial. Otolaryngol Head Neck Surg 2000; 122(3): 387–94. http://dx.doi.org/10.1067/mhn.2000.102120
76. Powell NB, Riley RW, Troell RJ, *et al*. Radiofrequency volumetric tissue reduction of the palate in subjects with sleep-disordered breathing. Chest 1998; 113(5): 1163–74. http://dx.doi.org/10.1378/chest.113.5.1163
77. Friedman M, Vidhyasagar R, Bliznikas D, Joseph NJ. Patient selection and efficacy of pillar implant technique for treatment of snoring and obstructive sleep apnea/hypopnea syndrome. Otolaryngol Head Neck Surg 2006; 134(2): 187–96. http://dx.doi.org/10.1016/j.otohns.2005.10.032
78. Friedman M, Schalch P, Lin HC, Kakodkar KA, Joseph NJ, Mazloom N. Palatal implants for the treatment of snoring and obstructive sleep apnea/hypopnea syndrome. Otolaryngol Head Neck Surg 2008; 138(2): 209–16. http://dx.doi.org/10.1016/j.otohns.2007.10.026
79. Nordgard S, Stene BK, Skjøstad KW. Soft palate implants for the treatment of mild to moderate obstructive sleep apnea. Otolaryngol Head Neck Surg 2006; 134(4): 565–70. http://dx.doi.org/10.1016/j.otohns.2005.11.034
80. Nordgard S, Stene BK, Skjostad KW, Bugten V, Wormdal K, Hansen NV, Nilsen AH, Midtlyng TH. Palatal implants for the treatment of snoring: long-term results. Otolaryngol Head Neck Surg 2006; 134(4): 558–64. http://dx.doi.org/10.1016/j.otohns.2005.09.033
81. Maurer JT, Hein G, Verse T, Hörmann K, Stuck BA. Long-term results of palatal implants for primary snoring. Otolaryngol Head Neck Surg 2005; 133(4): 573–8. http://dx.doi.org/10.1016/j.otohns.2005.07.027
82. Flum DR. Perioperative safety in the longitudinal assessment of bariatric surgery. N Engl J Med 2009; 361(5): 445–54. http://dx.doi.org/10.1056/NEJMoa0901836
83. Sjostrom L, Narbro K, Sjostrom CD, Karason K, Larsson B, Wedel H, Lystig T, Sullivan M, Bouchard C, Carlsson B, Bengtsson C, Dahlgren S, Gummeson A, Jacobson P, Karlsson J, Lindross AK, Lonroth H, Naslund I, Olbers T, Stenlof K, Torgerson J, Agren G, Carlsson LM. Effects of bariatric surgery on mortality in Swedish obese subjects. N Engl J Med 2007; 357(8): 741–52. http://dx.doi.org/10.1056/NEJMoa066254
84. Buchwald H, Avidor Y, Braunwald E, Jensen MD, Pories W, Fahrbach K, Schoelles K. Bariatric surgery: a systematic review and meta-analysis. The Journal of the American Medical Association 2004; 292(14): 1724–37. http://dx.doi.org/10.1001/jama.292.14.1724
85. Jon Erik C Holty, Barrett G Levesque, Jennifer Schneider Chafen, Vaughan Tuohy, John R Kapoor, Ingram Olkin, Dena M Bravata. Obstructive sleep apnea is prevalent and persistent among patients undergoing bariatric surgery: a systematic review 2010; 138(5).

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