We are IntechOpen, the world's leading publisher of Open Access books Built by scientists, for scientists



186,000

200M



Our authors are among the

TOP 1% most cited scientists





WEB OF SCIENCE

Selection of our books indexed in the Book Citation Index in Web of Science™ Core Collection (BKCI)

Interested in publishing with us? Contact book.department@intechopen.com

Numbers displayed above are based on latest data collected. For more information visit www.intechopen.com



Telegnathic Surgery for Obstructive Sleep Apnea

Dina Ameen Additional information is available at the end of the chapter http://dx.doi.org/10.5772/59276

1. Introduction

Obstructive sleep apnea (OSA) is a repetitive partial or complete upper airway collapse during sleep. It is defined as reparative episodes of hypopnea or apnea for at least 10 seconds in association with more than 2 % decrease in oxygen hemoglobin saturation. OSA along with snoring and upper airway resistant syndrome fall into a broad category of sleep related breathing disorder (SBD). The incidence of OSA is up to 9% of women and 24% of men aged 30–60y. Adverse consequences of OSA include: excessive daytime sleepiness (EDS), hypertension, ischemic heart disease, metabolic syndrome, stroke and death. There are many modalities for OSA treatment; conservative approach includes weight reduction, positioning devices, continuous airway pressure (CPAP), and oral appliances. Due to a large percentage of noncompliance with the conservative approach, surgical treatment is a valid option of OSA treatment.

Surgical options are tracheostomy, uvulopalatopharyngoplasty, palatal pillars, radiofrequency ablation of soft palate or tongue, anterior mandibular osteotomy, hypoid suspension, tongue reduction, tongue suspension and telegnathic surgery (maxillomandibular advancement). This chapter provides an overview in OSA surgical treatment.

2. Sleep stages

During normal sleep, the stages are:

1st **stage** is non-rapid eye movement (non-REM), lasting 70-90 minutes. The non-REM is the quiet sleep, which is consists of 4 stages.45-50% of total sleep is stage 2. Delta sleep is stages 3 and 4, which are a deeper sleep marked by the increasing appearance of high-amplitude slow waves. There is generalized slowness in all activities in non-REM sleep stage.



© 2015 The Author(s). Licensee InTech. This chapter is distributed under the terms of the Creative Commons Attribution License (http://creativecommons.org/licenses/by/3.0), which permits unrestricted use, distribution, and eproduction in any medium, provided the original work is properly cited.

2nd stage rapid eye movement (REM) stage will follow for 20 minutes. REM stage represents the deep sleep stage; it follows the non-REM stage with 20-25% of total sleep. During average night REM to non-REM ratio is 4:6 with intervals 60-90 minutes. Physiologic changes during REM are generalized muscle atonia except for ocular muscles, increase temperature, blood flow and oxygen use in the brain as well as increase in heart rate, blood pressure and respiration with dramatic fluctuations. Respiration is controlled by 2-control systems; metabolic and behavioral. Non-REM is predominantly controlled by the metabolic control system, which is influenced by hypoxia and hypercapnia. On the other hand during the REM sleep, behavioral control system is predominant. OSA usually occurs during stage 3, 4 and REM, which are the deep sleep stages, and that is because of blunt responses to hypoxia and hypercapnia along with the generalized muscle atonia; pharyngeal wall muscles may collapse [1] [2].

3. Anatomy

Upper airway obstructions can occur anywhere in the nasopharynx, oropharynx and hypopharynx. Nasopharyngeal obstruction examples are nasal septum deviation, nasal polyps and rhinosinusitis; they can cause mild OSA [3]. Most common sites of airway collapse occur in the hypopharynx [4]. It extends from the soft palate to the epiglottis; anteriorly it is formed by the base of the tongue and soft palate, while pharyngeal constrictor muscles form the posterior borders. Studies show that tongue volume and lateral walls of the pharynx are independent risks to OSA. There are many craniofacial abnormalities that cause OSA. Even a minimal change in maxillary or mandibular position can lead to upper airway collapse. OSA patients could have one or more of the following anatomical variations:

A retro-position of the mandible or the maxilla, micrognathia, long soft palate, increased thickness of the soft palate, macroglossia (large tongue) and differences in hyoid bone position [5].

The success of surgical treatment is depending on the recognition of the level of obstruction. There is a special surgical procedure for each site of obstruction.

4. Diagnosis

Many diagnostic tools can be used, yet physical examination is very important. A through physical examination of the nose, oral cavity, pharynx and neck should be done. Endoscopy gives clinicians visual assessment to the upper airway and may show possible sites of collapse. Endoscopic Mullar maneuver is a useful procedure for OSA[6]. The best results obtained by instructing the patient to lie down in supine position then inspire maximally then with closed nose and mouth, while placing the endoscope at the level of supraglottis, the examiner will be able to visualize the degree of pharyngeal collapse. Increase in negative pressure in the pharynx will demonstrate the point of collapse. Standardized performance and documentation is advocated to prevent any inter-investigator variability [7]. There are many classifications

for upper airway obstruction; Fujita's classification system described patterns of upper airway obstruction in OSA patients in 1985. Fujita classified airway obstruction into 3 types namely:

Type 1. Isolated palatal obstruction,

Type 2. Isolated retrolingual obstruction, and

Type 3. Palatal and retrolingual obstruction

Recently other modifications to Fujita classification were advocated by adding more details for the base of the tongue [8].

Polysomnography (PSG) is still the golden standard to establish OSA diagnosis. It can be used as a diagnostic tool as well as to assess therapeutic efficacy of a given treatment modality including weight loss, CPAP, oral appliances and MMA. It is usually done in a sleep clinic, as the patient should sleep at least for 4 hours, and the electroencephalogram (EEG), electrooculogram (EOG), electromyogram (EMG), and electrocardiogram (ECG) will be monitored [9].

There are many imaging studies proposed to evaluate the upper airway such as CT, MRI, dynamic scanning protocols e.g. ultrafast CT or MRI [7]. In oral and maxillofacial clinics cephalometric x-ray is still one of the most common x-rays used to diagnose and to evaluate treatment along with orthopantomogram (OPG). Both are a simple 2D image commonly used by oral and maxillofacial surgeon. It helps to detect posterior airway obstruction caused by skeletal disharmony. Examples of some important cephalometric measurements are:

- Sella nasion A point (SAN) 82 °
- Sella nasion B point (SAB) 80°
- Posterior airway space (PAS) 11mm
- Posterior nasal spine-palate (PNS-P) 35mm
- Mandibular plane-hyoid (MP-H) 15mm

OPG is used before any skeletal surgery to estimate the location of vital structures such as inferior alveolar nerve, mental foramen and apices of anterior teeth [10].

5. Risk factors and systemic complications

The incidence of OSA is different between men and women; most epidemiological studies reports male predominance with 5-8:1 ratio [11]. Male predominance is due to the sex related differences in upper airway anatomy and function, plus the differences in ventilator response to arousals from sleep [12]. But menopause women show a similar incidence to men because hormonal influences which play an important role in pathogenesis of OSA [13]. The other important risk factor is body mass index (BMI); the Wisconsin Sleep Cohort Study shows that one standard deviation difference in body mass index (BMI) was associated with a four-fold increase in disease prevalence [14] [15]. Partial or complete airway obstruction for more than

10 seconds will lead to decrease oxygen supply to vital organs such as the heart and brain which result in many sign and symptoms. Excessive daytime sleepiness, memory loss, impaired concentration, morning headache, decreased manual dexterity, libido and decrease sexual performance [16].

Systemic complications of OSA includes cardiovascular and neurocognitive disorders and death. Periods of apnea, prevent effective gaseous exchange at the alveoli which lead to hypoxia and hypercapnia. Apnea dependent hypoxia and hypercapnia increase sympathetic neural tone, which in turn cause vasoconstriction and increase in sympathetic nerve activity. Sympathetic nerve function rises progressively during apnea and is enhanced further by arousal. Increase in the sympathetic tone is the major cause of cardiovascular complications. OSA is associated with hypertension [16-18], arrhythmia, myocardial infarction [16, 19, 20], and congestive heart failure [16]. During the obstruction episodes there are marked changes in blood flow in cerebral arteries. Netzer et al reported 80% changes in cerebral blood flow in cerebral arteries [21]. During periods of apnea there is rapid increase in cerebral blood flow followed by rapid fall to below baseline levels after apnea periods. Fluctuation in cerebral blood flow along with many physiologic changes may lead to stroke [16]. Mortality rate of OSA can reach up to 30% in 15 years if left untreated [22]. Excessive daytime sleepiness could contribute to high rates of road traffic accidents. Studies show RTA among OSA patients is 1.3 to 7 times higher than the general population [23, 24].

6. Orthognathic surgery vs. telegnathic surgery

Maxillomandibular advancement is considered a telegnathic surgery, which involves maxillary and mandibular osteotomies to enlarge posterior airway space. Telegnathic surgery is derived from the Greek words *tele*, which means "over a distance," and *gnathis*, which relates to the jaws, whereas orthognathic surgery is derived from the Greek words, ortho, which means, "to straighten," and *gnathis meaning "jaw*". OSA patients are usually middle age, obese, mostly males with significant comorbid medical conditions; on the other hand patients with dentofacial abnormalities are young with no sex prevalence (male or female) and usually in a good health.

There are no major differences in the surgical techniques although the goals of therapy are different. In orthognathic surgery the goal is to correct the occlusion and improve esthetics while in telegnathic surgery the optimal goal is to relieve upper airway obstruction. Orthodontic treatment is a must for all patients with dentofacial deformities who are going for orthognathic surgery. In OSA patients accepting the existing bite can be used if the patient does not want to go through the lengthy orthodontic treatment. Surgical movement in the orthognathic surgery patient are dependent upon the esthetic requirement as well as occlusion correction, whereas in OSA patients a larger surgical movement of the maxilla and mandible should be done (up to 10mm) with the main concern being opening the posterior airway space [25, 26].

7. Classification

Respiratory disturbance index (RDI) represents the number of obstructive respiratory events per hour of sleep. An RDI of 5 is the upper limit of normal.

RDI= (apnea+hypopnea ÷ total sleep time) × 60 [27].

OSA is classified into mild, moderate and severe depending on the respiratory disturbance index (RDI) and oxyhemoglobin desaturation (SaO₂).

Mild OSA is when RDI 10-30 and SaO₂ >90%,

Moderate OSA is when RDI 30-50 and SaO₂ >85%, and

Severe OSA is when RDI >50 and $SaO_2 < 60\%$ [28].

8. Treatment options

8.1. Conservative treatment

After diagnosis of OSA, conservative treatment is indicated. It includes weight reduction, positional treatment, CPAP and oral appliances. Increase in BMI is a risk factor for OSA. Although high BMI is a risk factor for OSA, 30% of OSA patients are not obese. BMI is defined as the weight in kilograms divided by the height in meters squared (kg/m²). Overweight is considered when BMI of more than 25 kg/m² while obese is a BMI of more than 30 kg/m². Constriction of the hypopharynx and oropharynx are due to increase in neck circumference and increased fatty deposits in the peripharyngeal area [29]. In the Wisconsin Sleep Cohort Study, a 10% weight gain predicted a 32% increase in AHI, whereas a 10% weight loss predicted a 26% decrease in AHI [14].

Continuous positive airway pressure (CPAP) or nasal Continuous Positive Airway Pressures (nCPAP) are effective treatments for OSA. They are the 1st line treatment strategies when the patient is diagnosed with OSA. CPAP/ nCPAP work as a pneumatic splint to open the airway via tight fitting facemask or nasal mask and oxygen pump. There are many studies reporting the success of CPAP treatment. CPAP can stop and reverse all OSA complications. Treatments with CPAP result in decreased sympathetic tone, which will lead to decrease in blood pressure, AHI, oxygen desaturation and improve sleep efficiency, [30-32]. CPAP compliance however is only 65-80%, with 8% to 15% of patients stopping the treatment after the first night. This low compliance rate is due to many associated complications such as nasal dryness and congestion, sinus discomfort, massive epistaxis, skin rash and conjunctivitis from air leak. These complications plus the physical discomfort, noise and difficult transporting the unit lowers the CPAP tolerance [33,34].

OSA patients should be advised not to sleep in supine position; gravity is a factor that can cause upper airway collapse. Positional behavioral therapy is to educate the patient to alter

their sleep position by using a pillow or body belt; patients can alter their sleeping to a more lateral position that could open the airway and reduce collapsibility [35].

Mild to moderate OSA patients who are unable to tolerate CPAP can be treated with oral appliances. It simply prevents the mandible and associated muscles from going backward during sleep; some appliances actually advance the mandible from centric occlusion. Oral appliances should be adjusted on a periodic basis to prevent occlusal disturbances and temporomandibular joint dysfunction [36].

8.2. Surgical options

1969 Kuhlo et al was the first to recommend tracheostomy for OSA treatment [37]. Although tracheostomy is the most effective surgical procedure to treat OSA, it has morbidity and many adverse effects on the quality of life namely wound infection, stenosis and bleeding. Because of this many surgical techniques have developed to treat OSA. Based on the level of obstruction there are many surgical options; for example nasal surgeries such as septoplasty, turbinoplasty, polypectomy, adenectomy and tonsillectomy will address nasal obstruction. There are several palatal surgeries to address retropalatal obstruction for example: uvulopalatopharyngoplasty (UPPP), uvulopalatopharyngoplasty laser assisted (UPPP-LA), palatal pillar implants, radiofrequency ablation of the soft palate, and many others. Tongue operations like tongue suspension, radiofrequency ablation of the tongue, genial tubercle advancement with or without hyoid suspension are used for retrolingual obstruction. On the other hand maxillomandibular advancement with or without combined procedures address retropalatal and retrolingual levels of obstruction [26] [38].

The following section of this chapter will address some surgical techniques.

8.3. OSA treatment protocols

Successful OSA treatments depend on the recognition of the level of obstruction. Stanford University Sleep Disorders and Research Center proposed a protocol for OSA based on the site of obstruction.

Phase I protocol includes: UPPP, genioglossus advancement, and/or hyoid suspension then

Phase II protocol maxillomandibular advancement if the patient fails phase I treatment. MMA can be used to treat OSA as a primary or secondary procedure. MMA is considered primary when it is the 1st surgical procedure done to address multi-level of obstruction, when MMA done after phase one treatment it is called secondary MMA. Recently some surgeons prefer MMA advancement as the 1st surgical procedure especially if the patient has maxillary or mandibular deficiencies [26,39]. MMA is indicated if the diagnosis is confirmed severe OSA with RDI >50 and SaO_{2 <}60%, non-compliance or tolerance to CPAP, retroglossal obstruction, failure to respond to other surgical treatment such as UPPP and maxillomandibular hypoplasia [1].

8.4. Uvulopalatopharyngoplasty

Historically UPPP was an available option instead of tracheostomy until the recent expansion in surgical treatment of OSA. In 1981 Fujita et al introduced the concept of uvulopalatopharyngoplasty (UPPP) to enlarge retropalatal airway. UPPP involves partial excision of the uvula and redundant pharyngeal and palatal tissues, with primary closure of the anterior and posterior pillars under general anesthesia [40]. In 1991 O'Leary and Millman modify Fujita UPPP by excising the palatopharyngeus muscle [41]. Uvulopalatal flap is another modification published in 1996 by Powell et al [42]. UPPP complications range from velopharyngeal insufficiency, dysphagia (difficulty swallowing), voice changes, and death from general anesthesia [43]. With the advances in laser surgery uvulopalatopharyngoplasty–laser assisted (UPPP-LA) was developed using the same principle of scalpel UPPP [44]. Variable success rates reported in the literature is up to 70% and 78% respectively [45, 46]. Other studies show only 40 % success in eliminating snoring [47].

Today UPPP or UPPP-LA is rarely used as a single treatment modality; this is primarily due to the understanding of multilevel obstruction in most OSA patients. It is usually part of a staged protocol for OSA treatment [48]. UPPP can be performed with genioglossus advancement or with MMA. Hendler and Barry in 2001 published their data about 41 OSA patients; 33 of them treated with combined UPPP and modified mortised genioglossus advancement while the others had MMA combined procedures. All patients had pre-operative and post-operative polysomnography to evaluate treatment success. They reported comparable success rate of 86% in both groups concluding that UPPP/mortised genioglossus advancement is effective for the treatment of obstructive sleep apnea. Maxillomandibular advancement is effective for treating severe sleep apnea, and MMA can be done combined with UPPP/mortised genioglossus advancement in some cases as long as it is indicated in order to avoid multiple procedures [49].

8.5. Genioglossus advancement

Genioglossus muscle is a major pharyngeal dilator that plays an important role in OSA pathophysiology. In 1984 Riley et al. first reported advancing the genial tubercle with its genioglossus muscle attachment. The procedure was called inferior sagittal osteotomy [50]. If hyoid suspension to the inferior mandible is done at the same time it is called genioglossus advancement-hyoid myotomy [51]; the later was modified by suspending the hyoid to the thyroid cartilage. By advancing the genioglossus muscle the tension will increase at the tongue base thereby stabilize the hypopharyngeal airway [52]. In 1991 Riley et al modified the technique by limiting the osteotomy to a rectangular window and called it anterior mandibular osteotomy; this modification decreased anterior mandibular fracture [53]. In 2000, Lee and Woodson introduced a circular osteotomy of the genial tubercle [54]. All these modifications were done to address postoperative complications such as bone necrosis and anterior teeth pulp necrosis. Inferior sagittal osteotomy is indicated for patients with a deficient mandible in anteroposterior dimension, it involves genial tubercle advancement with the inferior border of the mandible while the occlusal relations ship is unchanged. On the other hand anterior mandibular osteotomy is indicated for patients with

normal mandibular anteroposterior dimension [52]; it requires careful assessment of the genial tubercle, based on a study done by Mintz et al [55] on 14 human skulls, the superior border of the genial tubercle is 6.45 mm inferior to the apices of central incisors with 35.4% of the genial tubercle were located less than 5mm inferiorly. After estimating genial tubercle location a rectangular window osteotomy is performed leaving the inferior border of the mandible intact then advancing the segment to stabilize the hypopharyngeal airway. It will require a 90 degree rotation of the osteotomized segment and placing the lingual cortical plate anterior to the buccal/labial cortical plate [52]. Trephine osteotomy approach is another technique using the same concept but with trephine burr in an attempt to decrease postoperative complications (anterior teeth roots amputation) [54]. Foltán and René [56] published a follow-up of 31 patients who had genioglossus advancement by the modified genioplasty with hyoid myotomy. They reported 74% success rate showed by significant dropping in RDI and oxygen desaturation index. Genioglossus advancement with or without hyoid suspension is a valid technique to treat OSA; it could be performed alone or as an adjunct to UPPP [57]. Another technique to address retrolingual obstruction is tongue base surgeries. Tongue suspension is a revisable minimally invasive surgery performed via submental incision. By introducing a large suture into the base of the tongue and suspending the tongue to the mandibular lingual surface. Omur et al reported high success (81.81%) of tongue base surgery with UPPP. They conclude that tongue base suspension combined with UPPP has been shown to reduce RDI better than UPPP alone [58].

8.6. Maxillomandibular advancement

It is well recognized that patients with maxillomandibular deficiencies will ultimately develop OSA; from this observation MMA is advocated for OSA treatment even in patients with normal skeletal proportion [59]. MMA will increase the posterior airway dimension by physically expanding the skeletal structure. The forward movement of the maxillomandibular complex improves the tension and collapsibility of the velopharyngeal and suprahyoid muscles [60] [61]. Since the majority of OSA patients are middle age with a saggy and droopy soft tissue; forwarded movement of the mandibulomaxillary complex will not only bypass the obstruction; it will also provide facial rejuvenation by augmenting soft tissue support [62]. MMA has many advantages such as decrease number of surgeries needed by utilizing one surgery to bypass several sites of obstructions (by performing Le Fort I advancement; it will open the nasal valve and consequently improve air flow, tighten the soft palate and pharyngeal muscles at the same time while mandibular advancement will tighten genioglossus and suprahyoid muscles), avoid the need for tracheostomy in the postoperative period and ultimately decrease medical costs by decreasing hospital stay. If the patient will undergo simultaneous MMA and soft tissue procedure such as UPPP or tongue reduction surgeries; temporary tracheostomy may be indicated to ensure patent airway. Other indications for temporary tracheostomy are difficult airway, RDI >60 and Sao2 <60, morbid obesity and significant craniofacial abnormality [1] [59]. MMA is considered one of the most successful treatment modalities for OSA after tracheostomy and CPAP [63] [64]. MMA success rate is very high comparing to other surgical treatment. Riley et al [65] reported the largest MMA series with success rate of 98%. In 2004 Dattilo and Drooger [66] reported 93% success rate in 14 of 15 cases, whereas Hochban et al in 1997 reported 97% success rate in 37 of 38 cases [67].

MMA as a surgical techniques per se is the same as classical orthognathic surgery it involve maxillary Le Fort I advancement and mandibular advancement simultaneously. The amount of advancement is usually 10mm - the maximum amount of possible advancement. There are some differences that should be considered with MMA e.g. vascular supply, bone healing and the need of adjunctive surgical procedures. Most of MMA candidate patients had unsuccessful UPPP; palatal scar may cause difficulty in advancing the maxilla or compromise its blood supply. Patients treated with UPPP may have or be at risk for velopharyngeal insufficiency (VPI). Advancing the maxilla may theoretically cause VPI or worsen existing VPI. During MMA surgery, based on cephalometrics and model surgery, the mandible is advanced first; this is because the amount of advancement is arbitrary and without any considerations of the maxillary incisors esthetic position or functional occlusion. [1,59,68]

Holty and Guilleminault published a meta-analysis of 53 reports describing 627 OSA patients with maxillomandibular advancement for the treatment of obstructive sleep apnea; they concluded that major and minor complication rates for MMA were 1.0% and 3.1%, respectively with cardiac complications as the most major complications. Facial paresthesia is the most common complication after MMA with 86% of cases resolved by 12 months after surgery. No postoperative deaths were reported. Most subjects reported satisfaction after MMA with improvements in quality of life measures [69]. Patients with poor response to MMA often have had UPPP. The possible cause is failure of the airway to stretch laterally in the retropalatal area caused by soft palate scarring from the previous surgery, making the tissues of the lateral pharyngeal walls stiffer and thus less responsive to advancement [1,59].

9. Distraction osteogenesis and OSA

Recently, many surgeons suggest distraction osteogenesis for treating OSA. Distraction osteogenesis has many advantages over the traditional MMA surgical technique; better soft tissue adaptation, elimination of the need of a bone graft, less soft tissue dissection and better stability. On the other hand, lengthy treatment and the need of postoperative orthodontic treatment are the disadvantages of this kind of treatment [70] [71].

10. Summary

OSA surgical treatment success is primarily dependent on careful diagnosis and recognition of levels of obstruction. Many surgical protocols are there in the literature. MMA along with tracheostomy are the most successful surgical procedures to treat OSA.

	Orthognathic surgery	Telegnathic surgery
Sex	Male or female	Male
Age	Young	Middle age or older
Health	Usually healthy	Many medical comorbidities
Surgical goal	Correct the occlusion and improve esthetics	Relive upper airway obstruction.
Type of movement	Depend on the dentofacial deformity	Maxillomandibular advancement
Amount of movement	Depend of the esthetic position of maxillary central incisors and facial esthetic	Up to 10mm advancement
Orthodontic treatment	Must	Might accept the existing bite

 Table 1. Differences between Orthognathic and Telegnathic surgery

	RDI=(apnea+hypopnea ÷ total sleep time) × 60	SaO ₂
Mild	10-30	>90%
Moderate	30-50	>85%
Severe	>50	<60%

Table 2. OSA classification

Conservative / medical treatment	Surgical treatment
Weight reduction	Tracheostomy
CPAP, nCPAP	Nasal surgeries
Positional device	Palatal surgeries
Oral appliances	Tongue surgeries
	Skeletal surgeries
Table 3. OSA Treatment	

Author details

Dina Ameen^{*}

Address all correspondence to: dr.dinaameen@gmail.com

Clinical Fellow at the University of Alabama at Birmingham, USA

References

- [1] Waite P D. Obstructive sleep apnea A review of the pathophysiology and surgical management. Oral Surgery Oral Medicine Oral Pathology ; 85(4).
- [2] Boudewyns AN, Van de Heyning. Site of upper airway obstruction in obstructive apnoea and influence of sleep stage. Eur Respir J. 1997; 10(11): p. 2566–72.
- [3] Georgalas C. The role of the nose in snoring and obstructive sleep apnoea: an update. Eur Arch Otorhinolaryngol. 2011; 268(9): p. 1365-73.
- [4] Schwab RJ, Gefter WB, Hoffman EA, et al. Dynamic upper airway imaging during awake respiration in normal subjects and patients with sleep disordered breathing. Am Rev Respir Dis. 1993; 148(5): p. 1385–400.
- [5] Chebbo A, Tfaili A, Ghamande S. Anatomy and Physiology of Obstructive Sleep Apnea. Sleep Med Clin. 2013; 8: p. 425–431.
- [6] Hsu PP, Tan AK, Tan BY, et al. Uvulopalatopharyngoplasty outcome assessment with quantitative computer-assisted videoendoscopic airway analysis. Acta Otolaryngol. 2007;(127): p. 65-70.
- [7] Boris A Stuck, Joachim T Maurer. Airway evaluation in obstructive sleep apnea. Sleep Medicine Reviews. 2008;(12): p. 411-436.
- [8] Kent E. Moore, and Ceib Phillips. A Practical Method for Describing Patterns of Tongue-Base Narrowing (Modification of Fujita) in Awake Adult Patients With Obstructive Sleep Apnea. J Oral Maxillofac Surg. 2002;(60): p. 252-260.
- [9] Mansoor Madani, Marcella Frank, Ryan Lloyd, Dessislava I. Dimitrova, Farideh Madani. Polysomnography Versus Home Sleep Study: Overview and Clinical Application. Atlas Oral Maxillofacial Surg Clin N Am. 2007; 15: p. 101-109.
- [10] Nuntigar Sonsuwan, Sirithorn Suchachaisri, Ladda Chaloeykitti. The relationships between cephalometric parameters and severity of obstructive sleep apnea. Auris Nasus Larynx. 2011; 38: p. 83-87.
- [11] Tishler, Larkin, Schluchter, Redline. Incidence of sleep disordered breathing in an urban adult population: the relative importance of risk factors in the development of sleep-disordered breathing. AJMA. 2003;(289): p. 2230-2237.
- [12] Strohl KP, Redline S. Recognition of obstructive sleep apnea. Am J Respir Crit Care Med. 1996;(154): p. 279-289.
- [13] Bixler EO, Vgontzas AN, Lin HM, et al. Prevalence of sleep-disordered breathing in women: effects of gender. Am J Respir Crit Care Med. 2001;(163): p. 608-613.
- [14] Young T, Palta M, Dempsey J, et al. The occurrence of sleep-disordered breathing among middle-aged adults. N Engl J Med. 1993;(328): p. 1230-1235.

- [15] Peppard PE, Young T, Palta M, et al. Longitudinal study of moderate weight change and sleep-disordered breathing. JAMA. 2000;(284): p. 3015-3021.
- [16] Jacek Wolf, Joanna Lewicka, Krzysztof Narkiewicz. Obstructive sleep apnea: An update on mechanisms and cardiovascular consequences. Nutrition, Metabolism & Cardiovascular Diseases. 2007; 17: p. 233-240.
- [17] Bixler EO, Vgontzas AN, Lin HM, Ten Have T, Leiby BE, Vela-Bueno A, et al. Association of hypertension and sleep-disordered breathing. Arch of Intern Med. 2000; (160): p. 2289-95.
- [18] Nieto FJ, Young TB, Bonnie KL, Shahar E, Samet JM, Redline S, et al. Association of sleep-disordered breathing, sleep apnea, and hypertension in a large communitybased study. JAMA. 2000;(283): p. 1829-36.
- [19] Peled N, Abinader EG, Pillar G, Sharif D, Lavie P. Nocturnal ischemic events in patients with obstructive sleep apnea syndrome and ischemic heart disease effects of continuous positive air pressure treatment. J Am Coll Cardiol. 1999;(34): p. 1744-9.
- [20] Sahar E, Whitney CW, Redline S, Lee ET, Newman AB, Nieto FJ, et al. Sleep disordered breathing and cardiovascular disease. Cross-sectional results of the sleep heart study. AM J Respir Crit Care Med. 2001;(163): p. 19-25.
- [21] Netzer N, Werner P, Jochums I, Lehmann M, Strohl KP. Blood flow of the middle cerebral artery with sleep-disordered breathing:correlation with obstructive hypopneas. Stroke 1998;29:7-93.
- [22] Young T, Finn L, Peppard PE, et al. Sleep disordered breathing and mortality: eighteen-year follow-up of the Wisconsin sleep cohort. Sleep. 2008;: p. 1071-8.
- [23] Findley LJ, Unverzagt ME, Suratt PM. Automobile accidents involving patients with obstructive sleep apnea. Am Rev Respir Dis. 1998;: p. 138-337.
- [24] Vidya Krishnan, Susheel P. Patil. Obstructive Sleep Apnea and Transportation: Medicolegal Issues. Sleep Med Clin. 2013; 8: p. 591–605.
- [25] Lars Andersson Karl-Erik Kahnberg, M. Anthony (Tony) Pogrel.Oral and Maxillofacial Surgery : Blackwell Publishing Ltd ; 2010.
- [26] Bagheri SC. Current therapy of Oral & Maxillofacial Surgery: Saunders ; 2012.
- [27] John G. Park, Kannan Ramar, and Eric J. Olson. Updates on Definition, Consequences, and Management of Obstructive Sleep Apnea. Mayo Clin Proc. 2011 jun: p. 549-555.
- [28] Institute for Clinical Systems Improvement web site. Healthcare guideline: Diagnosis and treatment of obstructive sleep apnea in adults. [Online].; 2008.
- [29] Hoffstein V, Mateika S. Differences in abdominal and neck circumferences in patients with and without obstructive sleep apnea. Eur Respir J. 1992;(5): p. 377-381.

- [30] Giles TL. Continuous positive airways pressure for obstructive sleep apnea in adults. 2006 oct: p. CD005308.
- [31] Pepperell JC, Ramdassingh-Dow S, Crosthwaite N, Mullins R, Jenkinson C, Stradling JR, et al. Ambulatory blood pressure after therapeutic and subtherapeutic nasal continuous positive airway pressure for obstructive sleep apnoea: a randomised parallel trial. Lancet. 2002;(359): p. 204-10.
- [32] Bonsignore MR, Parati G, Insalaco G, Marrone O, Castiglioni P, Romano S, et al. Continuous positive airway pressure treatment improves baroreflex control of heart rate during sleep in severe obstructive sleep apnea syndrome. Am J Respir Crit Care Med. 2002;(166): p. 279-86.
- [33] Waldhorn RE, TW Herrick, MC Nguyen, AE O'Donnell, J Sordero, Potolicchio SJ. Long term compliance with nasal continuous positive airway pressure therapy of obstructive sleep apnea. Chest. 1990;(97): p. 33-38.
- [34] Wells RD, Freedland KE, Carney RM, Duntley SP, Stepanski EJ. Adherence, reports of benefits, and depression among patients treated with continuous positive airway pressure. Psychosomatic Medicine. 2007; 65(5): p. 449-454.
- [35] Raphael C. Heinzer, Cyril Pellaton, Vincianne Rey, Andrea. Rossetti, Gianpaolo Lecciso, José Haba-Rubio, Mehdi Tafti, Gilles Lavigne. Positional therapy for obstructive sleep apnea: An objective measurement of patients' usage and efficacy at home. Sleep Medicine. 2012;(13): p. 425-428.
- [36] Tatsuya Fukuda, Satoru Tsuiki, Mina Kobayashi, Hideaki Nakayama, Yuichi Inoue. Selection of response criteria affects the success rate of oral appliance treatment for obstructive sleep apnea. Sleep Medicine. 2014;(15): p. 367–370.
- [37] Kuhlo W, Doll E, Frank MD. Erfolgreiche Behandlungeines Pickwick-Syndroms durch eine Dauertrachealkanüle. Dtsch Med Wochenschr. 1969;(94): p. 1286-90.
- [38] Macario Camacho et al. Surgical Treatment of Obstructive Sleep Apnea. Sleep Med Clin. 2013;(8): p. 495-503.
- [39] Hillel D. Ephros, Mansoor Madani, Brett M. Geller, Robert J. DeFalco. Developing a Protocol for the Surgical Management of Snoring and Obstructive Sleep Apnea. Atlas Oral Maxillofacial Surg Clin N Am. 2007;(15): p. 89–100.
- [40] Fujita S, Conway W, Zorick F, et al. Surgical correction of anatomic abnormalities of obstructive sleep apnea syndrome: uvulopalatopharyngoplasty. Otolaryngol Head Neck Surg. 1981;(89): p. 923 – 34.
- [41] O'Leary MJ, Millman RP. Technical modification of uvulopalatopharyngoplasty: the role of the palatopharyngeus. Laryngoscope. 1991;(101): p. 1332 5.
- [42] Powell N, Riley R, Guilleminault C, Troell R. A reversible uvulopalatal flap for snoring and sleep apnea syndrome. Sleep. 1996;(19): p. 593 – 9.

- [43] Kezirian EJ. Incidence of serious complicaions after uvulopalatopharyngoplasty. Laryngoscope. 2004; 114(3): p. 450-453.
- [44] Madani M. Laser assisted uvulopalatopharyngoplasty (LA-UPPP) for the treatment of snoring and mild to moderate obstructive sleep apnea. Atlas Oral Maxillofac Surg Clin North Am. 2007; 15(2): p. 129-137.
- [45] Madani M. Complications of laser-assisted uvulopalatopharyngoplasty (LA-UPPP) and radiofrequency treatments of snoring and chronic nasal congestion: a 10-year review of 5,600 patients. J Oral Maxillofac Surg. 2004; 62(11): p. 1351-1362.
- [46] Li HY, Wang PC, Lee LA, Chen NH, Fang TJ. Prediction of uvulopalatopharyngoplasty outcome: anatomy-based staging system versus severity-based staging system. Sleep. 2006; 29(12): p. 1537-1541.
- [47] Lyngkaran T, Kanaglaingam J, Rajeswaran R, Georgalas C, Kotecha B. Long-term out- comes of laser-assisted uvulopalatoplasty in 168 patients with snoring. J Laryngol Otol. 2006; 120(11): p. 932-938.
- [48] Walenczak I, Siekiewicz A, Rogowski M, Swietek M. The evaluation of the effectiveness of uvulopalatopharyngoplasy in the treatment of selected patients with mild to moderate obstructive sleep apnea—one year of postoperative follow up. Pol Merkur Lekarski. 2007; 22(132): p. 529-531.
- [49] Hendler, Barry H. A protocol for uvulopalatopharyngoplasty, mortised genioplasty, and maxillomandibular advancement in patients with obstructive sleep apnea: An analysis of 40 cases. Journal of oral and maxillofacial surgery. 2001; 59(8): p. 892.
- [50] Powell NB, Riley RW, Guilleminault C. Maxillofacial surgical techniques for hypopharyngeal obstruction in obstructive sleep apnea. Operative techniques. Otolaryngol Head Neck Surg. 1991;(2): p. 112 – 9.
- [51] Riley RN. Powell NB, Guilleminault C. Inferior sagittal osteotomy of the mandible with hyoid suspension: a new procedure for obstructive sleep apnea. Otolaryngol Head Neck Surg. 1986;(94): p. 589.
- [52] N. Ray Lee, Mansoor Madani. Genioglossus Muscle Advancement Techniques for Obstructive Sleep Apnea. Atlas Oral Maxillofacial Surg Clin N Am. 2007; 15: p. 179-192.
- [53] Riley RW, Powell NB, Guillaminault C. Obstructive sleep apnea and the hyoid: a revised surgical procedure. Otolaryngol Head Neck Surg. 1994;(111): p. 717-21.
- [54] Lee NR, Woodson T. Genioglossus muscle advancement via a trephine osteotomy approach. Operative techniques. Otolaryngol Head Neck Surg. 2000;(11): p. 50-4.
- [55] Mintz SM, Ettinger AC, Geist JR, et al. Anatomic relationship of the genial tubercles to the dentition as determined by cross-sectional tomography. J Oral Maxillofac Surg. 1995;(53): p. 1324.

- [56] Foltán, René. Genioglossus advancement and hyoid myotomy in treating obstructive sleep apnoea syndrome–A follow-up study. Journal of craniomaxillofacial surgery. 2007;(35): p. 246.
- [57] YIN, S. Genioglossus advancement and hyoid suspension plus uvulopalatopharyng-oplasty for severe OSAHS. Otolaryngology head and neck surgery. 2007; 136(4): p. 626.
- [58] Mehmet Omur, Dilaver Ozturan, Feyzi Elez, Celal Unver, Sabri Derman. Tongue Base Suspension Combined With UPPP in Severe OSA Patients. Otolaryngology– Head and Neck Surgery. 2005;(133): p. -218223.
- [59] Louis T. George, H. Dexter Barber, Brian M. Smith. Maxillomandibular Advancement Surgery: An Alternative Treatment Option for Obstructive Sleep Apnea. Atlas Oral Maxillofacial Surg Clin N Am. 2007;(15): p. 163–177.
- [60] Fairburn SC, Waite PD, Vilos G, et al. Three-dimensional changes in upper airways of patients with obstructive sleep apnea following maxillomandibular advancement. J Oral Maxillofac Surg. 2007;(65): p. 6-12.
- [61] Li KK, Guilleminault C, Riley RW, Powell NB. Obstructive sleep apnea and maxillomandibular advancement: an assessment of airway changes using radiographic and nasopharyngoscopic examinations. J Oral Maxillofac Surg. 2002;(60): p. 526–30.
- [62] Li KK, Riley RW, Powell NB, et al. Patient's perception of the facial appearance after maxillomandibular advancement for obstructive sleep apnea syndrome. J Oral Maxillofac Surg. 2001;(59): p. 377–80.
- [63] Waite PD, Shettar SM. Maxillomandibular advancement surgery: a cure for obstructive sleep apnea syndrome. Oral Maxillofac Surg Clin North Am. 1995;(7): p. 327–36.
- [64] Li KK. Surgical management of obstructive sleep apnea. Clin Chest Med. 2003;(24): p. 365–70.
- [65] Riley RW, Powell NB, Guilleminalt C. Obstructive sleep apnea syndrome: A review of 306 consecutively treated surgical patients. Otolaryngol Head Neck Surg. 1993; (108): p. 117.
- [66] Dattilo DJ, Drooger SA. Outcome assessment of patients under- going maxillofacial procedures for the treatment of sleep apnea: Comparison of subjective and objective results. J Oral Maxillofac Surg. 200;(62): p. 164.
- [67] Hochban W, Conradt R, Bradenburg U, et al. Plast Reconstr Surg. Surgical maxillofacial treatment of obstructive sleep apnea. 1997;(99): p. 619.
- [68] Scott B. Boyd. Management of Obstructive Sleep Apnea by Maxillomandibular Advancement. Oral Maxillofacial Surg Clin N Am. 2009;(21): p. 447–457.

- [69] Jon-Erik C. Holty, Christian Guilleminault. Holty CG. Maxillomandibular advancement for the treatment of obstructive sleep apnea: A systematic review and metaanalysis. Sleep Medicine Reviews. 2010;(14): p. 287–297.
- [70] Kasey K. Li, Nelson B. Powell, Robert W. Riley, Christian Guilleminault. Distraction Osteogenesis in Adult Obstructive Sleep Apnea Surgery: A Preliminary Report. J Oral Maxillofac Surg. 2002;(60): p. 6-10.
- [71] Stevan H. Thompson, Michael Quinn, Joseph I. Helman, Dale A. Baur. Maxillomandibular Distraction Osteogenesis Advancement for the Treatment of Obstructive Sleep Apnea. J Oral Maxillofac Surg. 2007;(65): p. 1427-1429.

