

TITLE: Obstructive Sleep Apnea

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Introduction

Although it was almost two centuries ago that Charles Dickens described a hypersomnolent, overweight child in the *Posthumous Papers of the Pickwick Club*, leading Sir William Osler to use the term "pickwickian", the study of sleep and its disorders is still in its infancy. Although each human spends somewhere between a third and a half of his or her life asleep, little is known about why we need sleep.

In the last thirty years there has been a blossoming body of knowledge regarding the diagnosis and treatment of sleep disorders. The otolaryngologist is frequently involved in the management of these patients. A thorough understanding of sleep disorders and their treatment is key for the practicing otolaryngologist. One must realize, however, that since the field is relatively new there is little that is absolute, and what comprises standard of care today may change with the rapid evolution of the field.

Sleep

Sleep is a state with which we are all familiar but is poorly understood. Beginning with the advent of the EEG, investigators began studying the electrical activity of the brain during sleep. Also, it was recognized early on that there are two distinct sleep states: a peaceful, physiologically restive state (non-REM or NREM sleep), and a different state characterized by muscle atonia and autonomic activity (increased heart rate and blood pressure) and rapid eye movements (REM sleep). Using the EEG investigators have divided NREM sleep into 4 stages. Stage I is the lightest and stage IV is the deepest. Through the night a person cycles up and down through these stages, with REM sleep occurring intermittently between stage II and I. As the night goes on more time is spent in REM sleep. This is important to realize, as the obstructive sleep apnea syndrome is more likely to occur during REM sleep, and thus a partial night sleep study may miss clinically important sleep apnea if done in the first portion of the night.

Upper Airway Obstruction

The primary sleep disorders that are brought to the attention of the otolaryngologist are those referable to upper airway obstruction. In a child this most often is related to adenotonsillar hypertrophy. In adults patients may present for a variety of reasons, or they may be incidentally picked up while being seen for other reasons. Diagnosis and management will be discussed below.

The pathophysiology involves obstruction of the upper airway during sleep. This is often associated with snoring, although snoring may or may not be associated with airway obstruction. As the airway obstructs the patient becomes hypercapnic and hypoxemic. This leads to a sub-wakeful arousal which opens the airway. Thus, the obstruction is relieved but at the expense of sleep fragmentation. The patient therefore has non-restorative sleep.

Definitions

To understand the specific disorders one must understand some of the definitions. An *apnea* is defined as cessation of airflow for ten seconds which results in an arousal. If the chest wall continues to mechanically move during this time, then it is an *obstructive apnea*. If the chest wall does not attempt to ventilate, then it is presumably due to a neurologic etiology and is termed a *central apnea*. Sometimes there are characteristics of both an obstructive and a central apnea, and this is termed a *mixed apnea*. The number of apneas per hour is termed the *apnea index*.

A *hypopnea* is a less well-defined entity, but usually is considered a diminution in airflow which results in hypoxemia and results in an arousal. The number of hypopneas per hour is termed the *hypopnea index*.

Functionally, there is little difference between apneas and hypopneas, and the sum of these vents per hour is termed the *apnea-hypopnea index* (AHI). This is also referred to as the *respiratory disturbance index* (RDI). Occasionally a lab will also report the *arousal index*, which is the number of arousal per hour. This may be different than the RDI due to limb movement or other causes of arousal.

Generally the *obstructive sleep apnea syndrome* (OSAS) is considered to be an RDI > 5. Also described is the *obstructive sleep hypopnea syndrome* (OSHS) which is a hypopnea index of greater than 15, but as mentioned there is little clinical utility in differentiating this from OSAS. Severity of OSAS is also stratified by the RDI, with mild being considered 5-20, moderate 20-40, moderate-severe 40-60, and severe > 60.

As the field of sleep medicine progressed there arose an awareness of certain patients who report excessive daytime sleepiness but do not have OSAS (e.g. RDI < 5). By esophageal manometry some of these patients have been shown to have increased negative thoracic pressure during inspiration. Thus, their increased work of breathing is thought to be responsible for their symptoms. This syndrome has been termed the *upper airway resistance syndrome* (UARS). Together with OSAS these are jointly referred to as *sleep-disordered breathing* (SDB). Those patients who snore but have an RDI < 5 and who do not have increased intrathoracic pressure

upon inspiration simply have *primary snoring*. One hypothesis is that these disorders represent a spectrum of disease with primary snoring being the mildest, followed by UARS, and finally OSAS as the full manifestation of the disease.

Pathophysiology

Although incompletely understood, the pathophysiology of OSAS relates to airway collapse. This may occur at various levels, including the palate, the base of tongue, and the hypopharynx. Nasal obstruction appears to facilitate or exacerbate the syndrome although it does not appear to be primarily responsible. Unfavorable anatomy appears to be the most important cause. This can be due to a narrow palate, an elongated uvula, redundant tissue at the base of tongue, micro/retrognathia, a retrodisplaced hyoid, and so on. Adenotonsillar hypertrophy may be a cause as well, particularly in the pediatric population. Experimental evidence shows that in the pharynx the collapse occurs predominantly from the lateral walls, not merely from anteroposterior collapse as might seem likely in patients with elongated palates. This also may explain the way in which obesity increases the prevalence of OSAS, as the lateral pharyngeal fat pads may narrow the airway in the lateral dimension.

Although unfavorable anatomy is important etiologically, there also appears to be a physiologic defect in the pharyngeal dilators. There is also experimental evidence that longitudinal tension appears to be inversely related to airway collapse. Additionally, extrinsic factors such as sedating medications may exacerbate the physiologic defects.

Rationale for treating OSAS?

Untreated, OSAS has a rather impressive list of deleterious consequences. For example, the incidence of systemic hypertension and its attendant sequelae is much higher in these patients. It is thought that this is due to systemic catecholamine release. Better understood is the link to pulmonary hypertension and cor pulmonale. The lung shunts blood away from hypoxic areas, presumably as a evolutionary efficiency mechanism during times of regional hypoxia (i.e. mucus plugging). However, during chronic episodic total lung hypoxemia the entire pulmonary vasculature constricts, and ultimately right-heart failure ensues. Other abnormalities associated with OSAS include myocardial arrhythmias, coronary artery and cerebrovascular disease and polycythemia. Whether due to previously mentioned problems or due to an independent cause, patients with OSAS have been shown to have an increase in mortality. Lastly, the neurocognitive compromise associated with inadequate sleep has been linked to poor performance and increased rates of industrial and traffic accidents.

Evaluation

History

Frequently a patient's bed partner may give more important information than the patient. Loud snoring and observed apneas are key historical points. Epidemiologically OSAS occurs more commonly in obese people, in men, and with increasing age. However, absence of these factors clearly does not exclude the possibility of sleep apnea. One should try to get a feel for the amount of fatigue a person experiences, as well as questions about sleep hygiene, such as

amount of nighttime alcohol and caffeine. Persons with insomnia should be questioned about reading in bed or watching TV in bed, as these are thought to contribute to insomnia. Morning headaches and impotence may be a tip-off for OSAS. Lastly, the examiner should always be vigilant to the possibility of malignancy and investigate questions related to recent changes in symptoms, weight loss, and symptoms such as otalgia, dysphagia, dyspnea, and hoarseness.

A questionnaire is also frequently administered to the patient in order to ascertain the patient's subjective level of fatigue. One such tool commonly used is the Epworth Sleepiness Scale. In this scale a patient rates the chance of dozing off in eight different settings and a numerical score is tabulated. This may help identify patients in whom the subjective level of fatigue is discordant with the RDI, such as those patients with UARS.

Physical

Frequently the OSAS patient is obese. Neck size over seventeen inches has been shown to be a risk factor for OSAS. In someone with a neck over seventeen inches who snores the incidence of OSAS has been quoted at 30%. As part of the vital signs it is a good idea to calculate the body-mass index (BMI), which is obtained by dividing the weight in kilograms by the square of the height in meters. Additionally, one should check the blood pressure, as the prevalence of hypertension is very high in this population.

The rest of the physical exam should focus on sites of potential obstruction in the head and neck. In particular, one should look at the tonsils, the length and width of the soft palate, size of the tongue, and position of the jaw and hyoid. Nasal obstruction should be looked for, as well as dynamic nasal collapse. As always, a complete head and neck exam should be performed and the possibility of tumor excluded.

Muller's maneuver is designed to look for the site of collapse. With the flexible scope in position, the patient tries to inspire against a closed mouth and pinched nostrils. The inspiratory forces then serve to collapse the airway. By doing this one can get an idea of the relative collapsibility of the palate as compared to the base of tongue or the hypopharynx. Although not terribly sensitive or specific, it still can be a helpful diagnostic and decision-making aid and adds little effort to the physical exam.

Testing

The gold standard for assessing OSAS is the *polysomnogram*, or sleep study. Although not completely standardized, the typical polysomnogram (PSG) will have measurements including an electroencephalogram (EEG), electro-oculogram, submental and tibial electromyogram (EMG), nasal or oral airflow, respiratory movement or effort, oximetry, electrocardiogram (EKG), and sleeping position. Some may also include measurements of penile tumescence and multilevel esophageal manometry.

Some controversy exists about a split-night PSG. For economic and convenience reasons, frequently in the first half of the night a standard PSG is performed followed by PSG with CPAP titration during the second half (see *Treatment: Non-surgical*, below). Due to changes in the sleep architecture during the night it is possible that OSAS might not be picked up during the first half of the night. Thus, a negative first half of the study does not exclude OSAS

and a complete study should be done without CPAP titration. However, a positive first half study has been shown to be reliable for diagnosis of OSAS, and in studies looking at CPAP titration, no advantage was found in using the whole night's data over the first half of the night. Thus a positive study in the first half of the night may go on to CPAP titration in the second.

Although efforts are underway to investigate limited polysomnograms or versions that patients can perform at home, the gold standard at this time is still the full PSG at a sleep lab.

Cephalometric studies have been used to study potential OSAS patients. Using plain films of the head, measurements of various parameters are done and compared to normative data in an effort to predict the likelihood of OSAS in a particular patient. Although potentially useful as a screening tool, these studies are not routinely done at our institution.

The *multiple sleep latency test (MSLT)* is another tool to study sleep disorders. During a person's normal waking hours he or she takes four or five monitored naps separated by a few hours. The time to fall asleep, or sleep latency, is measured (abnormal being too quickly). This may reveal dysfunction in patients with otherwise normal PSGs. For example, this may reveal narcolepsy. Also, patients with UARS who have been treated with CPAP have been shown to have an improvement in their MSLT testing.

Lastly, any patient who is being considered for PSG should undergo *thyroid testing*, as thyroid dysfunction may cause similar symptoms and should be corrected prior to PSG testing. Also, many of the patients with OSAS will have cardiopulmonary dysfunction, and appropriate preoperative testing should be performed prior to any surgical intervention.

Treatment

Non-surgical

In obese patients one should consider *weight loss*. It is felt that there is a threshold level of weight for most individuals above which they experience symptoms and below which they do not. Also investigational tools such as computed tomography have demonstrated the increase in the airway with weight loss. Additionally, obese patients have many other medical benefits to gain from weight loss.

Sleep hygiene should be reviewed. Alcohol and sedating medicines may exacerbate OSAS. Patients with insomnia should be counseled to avoid nighttime caffeine and avoid activities such as reading or watching television in bed. Lastly, since OSAS is usually worse when supine, some patients may benefit from relatively simple measures such as sewing a tennis ball into the back of a T-shirt to promote sleeping on the side.

Continuous positive airway pressure (CPAP) remains a mainstay of treatment. Via facemask or nasal mask, positive airway pressure is delivered. This prevents pharyngeal collapse and has been shown to alleviate PSG abnormalities and symptoms in many patients. If delivered at two different pressure levels (for inspiration and expiration) it is referred to as BiPAP. CPAP titration can be done, as mentioned previously, during the second half of a split-night sleep study. This is done to find the lowest level of pressure that alleviates the apneas.

Despite its effectiveness, compliance is often an issue, as many cannot tolerate the mask or the high pressures necessary for certain individuals. Intolerance of CPAP is a common indication for surgery, and often the patient still requires CPAP after surgery but can tolerate it due to relief of nasal obstruction or the improved airway that requires lower pressures to achieve relief of OSAS.

Oral appliances are also effective in relieving OSAS. These work by mechanically moving the jaw or tongue forward and opening the airway. They have been shown to be effective, more so for mild or moderate OSAS. Although some find them difficult to use, in general compliance is superior to CPAP. At around 62% compliance though, overall efficacy is lower than for surgical procedures. No single appliance has been shown to be superior to another.

Surgical

When a patient opts for surgical therapy the otolaryngologist has a variety of considerations. Most would agree that prior to surgical intervention a PSG should be obtained. Some argue that medical therapy should be tried first with surgery reserved for medical failures. Many times, though, a combination of medical and surgical therapy is necessary for optimum results.

Anesthesia considerations are important in these patients. As many of them have cardiopulmonary issues, the appropriate pre-anesthesia testing is necessary. Also, many of these patients have the combination of a short, thick neck and some degree of retrognathia. This is a setup for an airway problem. Vigilant attention to intubation and extubation is necessary. Many anesthesiologists perform awake fiberoptic intubations and are very judicious in their use of medications which might compromise the airway. On the other end, it is recommended that extubation be done when relatively light. Of course, proper equipment for airway emergencies should be available. In this event, adjunctive measures such as the laryngeal mask anesthesia or trans-tracheal jet ventilation may buy the surgeon precious moments. As always, proper communication between the surgeon and anesthesiologist is important. Post-operative considerations include the possibility for post-obstructive pulmonary edema and the likelihood of post-operative hypertension. Depending on the individual patient, intensive care unit monitoring may be best for the first post-operative night.

Surgery to relieve nasal obstruction should be performed in adjunct to other sleep apnea surgery. Nasal surgery alone has a very low rate of efficacy for obstructive sleep apnea. Adenoidectomy can be performed when adenoid hypertrophy exists, but as with nasal surgery, tonsillectomy and adenoidectomy alone have a very low rate of efficacy in adults with sleep apnea.

If the obstruction appears to be at the palatal level, some palatal intervention is in order. The *uvulopalatopharyngoplasty* (UPPP) remains a mainstay of treatment. Described by Ikematsu in the 1950s for snoring, Fujita recognized in the 1980s that this was effective for the treatment of OSAS. In the procedure a portion of the soft palate, the uvula, and the tonsils are removed. The posterior pillars are sewn anterolaterally. Complications can include voice changes and oronasal regurgitation of food or liquids. Rarely, the devastating complication of

nasopharyngeal stenosis can occur.

Also described for the palate is the *laser-assisted uvulopalatoplasty* (LAUP). While this can be done in the office, it often requires multiple sessions. It may also be more appropriate for primary snoring.

Radiofrequency ablation can be used to create thermal lesions that have a similar effect to palatal surgery. These procedures must be done with the patient asleep because of associated pain.

For hypopharyngeal and base of tongue obstruction, first-line therapy at this institution is *genioglossal advancement and hyoid myotomy and suspension* (GAHM). In this procedure, a portion of the central mandible is advanced, and the inner table secured at the level of the outer table. This mechanically advances the tongue relative to the posterior pharynx. A lingual suspension procedure using a suture that anchors the base of tongue forward to the mandible may achieve similar results (though the long term results are not yet known). Usually along with the genioglossal advancement, a hyoid myotomy and suspension is performed. The neck is opened, and the hyoid bone is freed of its muscular attachments. It is then secured anteriorly by four sutures to the thyroid cartilage, moving the base of tongue forward. Also described for tongue base obstruction are procedures such as the lingual tonsillectomy, uvulopalatopharyngoglossoplasty, laser midline glossectomy, lingualplasty, and radiofrequency volumetric tissue reduction.

Worthy of mention is the Riley-Powell-Stanford surgical protocol. At their institution they have had success by performing UPPP and/or GAHM as first-line surgical therapy (phase I). This results in a 61% success rate. For patients with persistent disease, they then undergo *maxillomandibular osteotomy and advancement* (Phase II). This resulted in a 97% success rate in their patients who failed Phase I surgery. Success is defined as a 50% reduction in the RDI and an RDI of <20. Thus, maxillomandibular osteotomy and advancement may be a viable option for certain patients. At our institution, we involve the oral surgery service in the care of these patients.

Lastly, the definitive surgery for OSAS is *tracheotomy*, which results in upper airway bypass. This is usually reserved for morbidly obese patients. In these patients it is recommended by many to use the neck skin to line the tract, resulting in a more permanent tract. Tracheotomy in this patient population is technically difficult and is not free of complications.

Conclusion

Sleep medicine is an exciting, relatively new field that has emerged. The otolaryngologist has become a key figure in the diagnosis and management of sleep disorders due to his or her familiarity with the airway and the ability to intervene surgically. An understanding of the medical and surgical issues involved is necessary for the otolaryngologist to deal with this field which is rapidly evolving.

Bibliography

Anonymous. Cost justification for diagnosis and treatment of obstructive sleep apnea: position statement of the American Academy of Sleep Medicine. *Sleep* 23(8):1017-8, 2000 Dec.

Berger G, Finkelstein Y, Stein G, et al. Laser-assisted uvulopalatoplasty for snoring: medium- to long-term subjective and objective analysis. *Archives of Otolaryngology - Head & Neck Surgery* 127(4):412-7, 2001 Apr.

Carskadon MA, Dement WC. Normal human sleep: an overview. In: Kryer MH, Roth T, Dement WC, eds. *Principles and practice of sleep medicine*. Philadelphia: WB Saunders. 1994;16-25.

Chaudhary BA. Obstructive sleep apnea. *Resident and Staff Physician* 44(9) 21-34, 1998 Sep.

Coleman J. Overview of sleep disorders. *Otolaryngologic Clinics of North America* 32(2):187-93, 1999 Apr.

Coleman J. Sleep studies: current techniques and future trends. *Otolaryngologic Clinics of North America* 32(2):195-210, 1999 Apr.

Coleman J, Rathfoot C. Oropharyngeal surgery in the management of upper airway obstruction during sleep. *Otolaryngologic Clinics of North America* 32(2):263-76, 1999 Apr.

Goldberg AN, Schwab RJ. Identifying the patient with sleep apnea: upper airway assessment and physical examination. *Otolaryngologic Clinics of North America* 31(6):919-30, 1998 Dec.

He J, Kryger M, Zorick F, et al. Mortality and apnea index in obstructive sleep apnea. *Chest* 94:9-14, 1988.

Johnson JT. Uvulopalatopharyngoplasty. In Myers, EN (ed). *Operative Otolaryngology: Head and Neck Surgery*. Philadelphia: WB Saunders. 1997; 208-14.

Johnson JT, Braun TW. Preoperative, intraoperative, and postoperative management of patients with obstructive sleep apnea syndrome. *Otolaryngologic Clinics of North America* 31(6):1025-30, 1998 Dec.

Millman RP, Rosenberg CL, Kramer NR. Oral appliances in the treatment of snoring and sleep apnea. *Otolaryngologic Clinics of North America* 31(6):1039-48, 1998 Dec.

Picirillo JF, Thawley SE. Sleep-Disordered Breathing. In *Otolaryngology – Head and Neck Surgery*, 3rd ed. Cummings CW, et al (eds) Mosby:St Louis, 1999.

Redline S, Strohl KP. Recognition and consequences of obstructive sleep apnea hypopnea syndrome. *Otolaryngologic Clinics of North America* 32(2):303-31, 1999 Apr.

Sanders M, Black J, Constantino J, et al. Diagnosis of sleep disordered breathing by half-night polysomnography. *Am Rev Respir Dis* 144:1256-61, 1991.

Scharf S, Garshick E, Brown R, et al. A screening for subclinical sleep disordered breathing. *Sleep* 13:344-53, 1990.

Schwab RJ, Goldberg AN. Upper airway assessment: radiographic and other imaging techniques. *Otolaryngologic Clinics of North America* 31(6):931-68, 1998 Dec.

Schwartz AR, Eisele DW, Smith PL. Pharyngeal airway obstruction in obstructive sleep apnea. *Otolaryngologic Clinics of North America* 31(6):911-8, 1998 Dec.

Stroud R, Quinn FB. Obstructive sleep apnea syndrome. In *Dr Quinn's Online Textbook* available at www.utmb.edu/oto, 1998 Feb.

Troell RJ, Riley RW, Powell NB, Li K. Surgical management of the hypopharyngeal airway in sleep disordered breathing. *Otolaryngologic Clinics of North America* 31(6):979-1012, 1998 Dec.

Walker RP. Snoring and obstructive sleep apnea. In Bailey BJ, ed. *Head and Neck Surgery – Otolaryngology*. Philadelphia: Lippincott-Raven, 1998.

Yamashiro Y, Kreyger M. CPAP titration for sleep apnea using a split night protocol. *Chest* 107:62-6, 1995.

Friedman, Michael MD; Ibrahim, Hani MD; Joseph, Ninos J. **BS Staging of Obstructive Sleep Apnea/Hypopnea Syndrome: A Guide to Appropriate Treatment.** *Laryngoscope*. 114(3):454-459, March 2004.

Riley RW, Powell NB, Li KK, Guilleminault C. Surgical therapy for obstructive sleep apnea–hypopnea syndrome. In: Kryger MH, Roth T, Dement WC, eds. *Principles and Practice of Sleep Medicine*. Philadelphia, Pa: WB Saunders Co; 2000:913-928.

Cahali MB. Lateral pharyngoplasty: a new treatment for **obstructive sleep** apnea hypopnea syndrome. *Laryngoscope*. 113(11):1961-8, 2003 Nov.

Thatcher GW, Maisel RH. The long-term evaluation of **tracheostomy** in the management of severe **obstructive sleep** apnea. [Journal Article] *Laryngoscope*. 113(2):201-4, 2003 Feb.

Friedman M, Ibrahim H, Bass L. Clinical staging for sleep-disordered breathing. *Otolaryngol Head Neck Surg* 2002; 127: 13–21.

Walker-Engstrom ML, Tegelberg A, Wilhelmsson B, Ringqvist I. 4-year follow-up of treatment with dental appliance or **uvulopalatopharyngoplasty** in patients with **obstructive sleep** apnea: a randomized study. *Chest*. 121(3):739-46, 2002 Mar.

Woodson, Tucker “Obstructive Sleep Apnea Syndrome, Diagnosis and Treatment” SIPAC 1996

Please note:

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