OBSTRUCTIVE SLEEP APNEA; A MINI-REVIEW

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ABSTRACT

Obstructive apnea syndrome (OSA) could be a doubtless serious disorder assaultive numerous individuals round the world. Many of those people square measure unknown, and albeit diagnosed usually exhibit a poor compliance with the employment of continuous positive airway pressure at nights, an awfully effective non-surgical treatment. A variety of surgical procedures are projected to manage and treat OSA. This article throws insights into assessing the sites of obstruction and variety of surgical procedures designed to deal with OSA. The scope of this text is to produce info to dentists that allows them to spot the patients United Nations agency have OSAS and to guide these patients in creating enlightened decisions regarding treatment options.

Keywords: Apnea, obstructive sleep apnea, sleep, snoring.

INTRODUCTION

Obstructive sleep apnea (OSA) is a deliberate sleep disorder. This condition is characterized by a temporary and repetitious cessation of breathing resulting from repeated upper airway closure during a person's sleep which subsidize to cardiovascular complications, decreased work productivity and are often associated with arousals, sleep fragmentation, intermittent hypoxia, and hypercapnia including excessive daytime sleepiness, growth failure, school failure, behavioral problems or cor pulmonale, automobile accidents, or even sudden death. Cessation of airflow leads to disruption of gas exchange and thus causes sympathetically driven recurrent arousal from sleep. OSAS has long been recognized as a major contributor to morbidity and mortality in developed countries. According to a survey conducted on a semi urban Indian population, it was found that around 6.2% among total sample were diagnosed with high-risk OSAS and 33.5% of the obese population were at high risk of OSAS[1,2].

The prevalence of OSAS based on the Wisconsin cohort study performed in the USA involving middle-aged adults 30–60 years of age, is 2% in females and 4% in males[3]. The long-term effects of sleep fragmentation and intermittent hypoxia on health are numerous leading to systemic hypertension, impaired glucose metabolism and cardiovascular disease, as well as societal effects such as increased car accidents[1].

OSA is classified by several different components. Apneas, hypopneas and blood oxygen saturation (SaO2) dips are the main constituents of this disorder. An apneic episode is a complete cessation of airflow lasting 10 sec. or more, while a hypopnea is a reduction in respiratory airflow combined with a SaO2 decrease of >4%. Additional factors, which the sleep specialist feels are clinically significant, may also be accounted for and included when diagnosing OSA.

Signs and symptoms of obstructive sleep apnea include[4]:

- Excessive daytime sleepiness
- Loud snoring
- Observed episodes of breathing cessation during sleep
- Abrupt awakenings accompanied by gasping or choking
- Awakening with a dry mouth or sore throat
- Morning headache
- Difficulty concentrating during the day
- Experiencing mood changes, such as depression or irritability
- High blood pressure
- Nighttime sweating
- Decreased libido

PATHOPHYSIOLOGY

Factors leading to obstructive sleep apnea include

1. A compromised often completely closed extra thoracic upper airway (obstructive event).

2. Marked reduction or cessation of brain stem respiratory center motor output (central event)

3. A combination of obstruction and central events.

These events produce intermittent hypoxemias leading to arousals from sleep, sleep fragmentation and further causing over compensatory responses of autonomic nervous system. The common sites of obstruction are retropalatal, retropharyngeal and hyypopharyngeal areas. Bony abnormalities like reduced mandibular body length, inferiorly positioned hyoid bone, retroposition of maxilla, 10 increased vertical length of the upper airway can lead to OSA. An enlarged soft palate and tongue would reduce the anteroposterior diameter where as enlarged pharyngeal walls reduces the lateral plane[5].

OSA is caused by repetitive bouts of upper airway obstruction during sleep as a result of the narrowing of respiratory passages. The most common site of obstruction is the nasopharynx[6]. It is important to differentiate OSA from the less common central sleep apnea, which is caused by an imbalance in the brain's respiratory control centers during sleep. While the pathogenesis of OSA is thought to be multifactorial, anatomic defects are thought to play a major role[6,7].

As the patient falls asleep, muscles of the nasopharynx begin to relax and the surrounding tissue collapses, causing compromise of the airway. As oxygen levels in the body start to drop and carbon dioxide levels rise, the patient is aroused from sleep; this causes an increase in sympathetic tone and subsequent contraction of nasopharyngeal tissue, which allows alleviation of the obstruction. Upon the patient's falling back to sleep, however, the airway is again subjected to narrowing until the patient is aroused from sleep once again. The cycle continues throughout the night, causing decreased time spent in rapid eye movement sleep and an overall decrease in quality of sleep. Because of the gravity-dependent factors discussed above, most obstructive symptoms happen in the supine position[8-12].

In healthy subjects, the phasic activity of some dilator muscles has been found to decline during rapid eye movement sleep and the pharyngeal cross-sectional area has been found to be smaller during sleep than during wakefulness . Indeed, reflex mechanisms from both chemoreceptors and mechanoreceptors which control the activity of pharyngeal dilator muscles are reduced during sleep [13-16].

RISK FACTORS

Obesity, male sex, age, heritable factors, craniofacial anatomic predisposition are the risk factors. Approximately 60-70% of OSA are obese. The occurrence of OSA in non obese exhibits different characteristics. In Indian children Adenoids and tonsillar enlargement (72%) were the causes for snoring, whereas disproportionate upper airway anatomy at multiple levels like long thick soft palate (62%), long and edematous uvula (62.8%), webbing 19 of tonsillar pillars (40%) were the causes. Incidence of OSA increases with age from 2% at age 30 for females to 28% at 60 years and from 4% at age 30 to 12 67% at 60 years of age for males. Elderly also have high snoring and high excessive daytime sleepiness 20 (EDS), with 27% being habitual snorers. The 21 prevalence of obesity is increasing in Asians. The increase in prevalence from 2.3% to 19.6% occurred over a period of a decade up to 1998 and rate further 2 increased when a lower cutoff value of 23- 25kg/M was given for Asians. The national family health survey, India (NFHS) report shows that 12.1% of men and 14.8% of women in India are overweight or 22 obese. According to a study in industrial workers in a city in India 30.9% of men and 32.8% of women were obese[17-20].

COMPLICATIONS[21]

Obstructive sleep disorder is taken into account a heavy medical condition. Complications may include:

- **Daytime fatigue and sleepiness.** The continual awakenings related to preventative sleep disorder build traditional, restorative sleep not possible. People with preventative sleep disorder usually expertise severe daytime somnolence, fatigue and irritability. They may have issue concentrating and realize themselves falling asleep at work, whereas look TV or maybe once driving.
  Children and teenagers with preventative sleep disorder might do poorly in class and unremarkably have attention or behavior issues.
- **Cardiovascular problems.** Sudden drops in blood gas levels that occur throughout preventative sleep disorder increase force per unit area and strain the circulatory system. Many people with preventative sleep disorder develop high force per unit area (hypertension), which may increase the danger of cardiopathy.
  The additional severe the preventative sleep disorder, the larger the danger of arteria malady, heart attack, heart failure and stroke. Men with preventative sleep disorder seem to be in danger of coronary failure, whereas ladies with preventative sleep disorder do not.

Obstructive sleep disorder will increase the danger of abnormal heart rhythms (arrhythmias). These abnormal rhythms can lower blood oxygen levels. If there is underlying cardiopathy, these continual multiple episodes of low blood gas may lead to overtime from a internal organ event.

- **Complications with medications and surgery.** Obstructive sleep disorder is also a priority with sure medications and anaesthesia. These medications, like sedatives, narcotic analgesics and general anesthetics, relax your higher airway and should worsen your preventative sleep disorder.
  If you've got preventative sleep disorder, you will expertise worse respiration issues once surgical procedure, particularly once being insensible and lying on your back. People with preventative sleep disorder could also be additional susceptible to complications once surgery.
  Before you've got surgery, tell your doctor if you've got preventative sleep disorder or symptoms associated with preventative sleep disorder. If you've got preventative sleep disorder symptoms, your doctor might take a look at you for preventative sleep disorder before surgery.
- **Eye problems**. Some analysis has found a association between preventative sleep disorder and sure eye conditions, like eye disease. Eye complications can usually be treated.
- **Sleep-deprived partners**. Loud snoring will keep those around you from obtaining sensible rest and eventually disrupt your relationships. Some partners might even favor to sleep in another space. Many bed partners of individuals WHO snore ar sleep underprivileged likewise.

People with preventative sleep disorder may additionally complain of memory issues, morning headaches, mood swings or feelings of depression, and a necessity to urinate often in the dark (nocturia).

TESTS FOR DETERMINATION

Currently, polysomnography, which requires a sleepover in a laboratory, is the optimum test for diagnosing sleep apnea. It involves the evaluation of sleep staging, airflow and ventilatory effort, arterial oxygen saturation, electrocardiogram, body position, and periodic limb movements. Polysomnography is not always readily available. Alternative methods to consider are evaluation using pulse oximetry and portable (home) monitoring of cardiopulmonary channels[22].

- **Polysomnography.** During this sleep study, you are connected to instrumentation that monitors your heart, lung and brain activity, breathing patterns, arm and leg movements, and blood oxygen levels while you sleep. You may have a full-night study, during which you are monitored all night, or a split-night sleep study.
  In a split-night sleep study, you will be monitored throughout the primary 1/2 the night. If you are diagnosed with preventative sleep disorder, employees might wake you and provides you continuous positive airway pressure for the half of the night. This take a look at will facilitate your doctor diagnose preventative sleep disorder and alter positive airway pressure medical care, if acceptable.
  This sleep study may also facilitate rule out different sleep disorders, such as periodic limb movements of sleep or narcolepsy, which also can cause excessive daytime sleepiness, but require different treatment[23].
- **Home sleep apnea testing**. Under sure circumstances, your doctor may provide you with an at-home version of polysomnography to diagnose obstructive sleep apnea. This test usually involves measurement of airflow, breathing patterns and blood oxygen levels, and possibly limb movements and snoring intensity[23].

HEALTH RELATED QUALITY OF LIFE IN OSA

Obstructive sleep apnea has a tremendous effect on a person's quality of life. Instrumental activities of daily living are often interrupted or at least made more difficult as a result of this disorder. The consequences of OSA may include memory deficits, decreased alertness, irritability, depression, headaches, or a decrease in the ability to concentrate. These effects can result in an impairment of work efficiency, relationship and social problems, and increased automobile accidents[24-26].

MANAGEMENT

OSAS can be managed nonsurgically or surgically. The treatment modality should focus on the potential contributing factors identified by the history, physical examination, and upper airway imaging. The severity of the patient's condition must also be taken into consideration in developing a treatment plan. Since obesity is a risk factor for OSAS, a weight loss program can reduce sleep apnea[27].

NON-SURGICAL METHOD

Continuous positive airway pressure

Continuous positive airway pressure(CPAP) is the gold standard treatment option for moderate-to-severe cases of OSA and a better option for mild sleep apnea. The first treatment of sleep apnea was introduced in 1981; CPAP provides a steady stream of pressurized air to patients through a mask that they wear[28].

Oral appliances

Oral or dental appliances may be the choice for patients with mild-to-moderate sleep apnea. However, they are not a remedy for all patients. This airflow maintains the airway open, preventing pauses in breathing and restoring normal oxygen levels. Novel CPAP models are small, light, and virtually silent. A variety of devices that move the tongue forward or move the mandible to an anterior and forward position to improve patency of the airway have been marketed[29].

Oral appliances are preferred to people with mild-to-moderate OSA who either prefer it than CPAP or they are not able to successfully comply with CPAP therapy. Oral appliances agree very like sports' mouth guards, and that they aid in maintaining Associate in Nursing open and patent airway by placement or helpful the submaxilla, tongue, tongue, or uvula. Some are specifically designed for snoring and others are planned to treat both snoring and sleep apnea[30]

Surgical treatment method

Tracheotomy is the primary treatment modality for OSA and a temporary measure during other surgical procedures where airway cannot be maintained through oro-nasal routes. Thatcher and Maisel state that tracheostomy leads to quick reduction in sequelae of OSA and results in only a very few complications[30].

The initial methods used were surgical and upper airway reconstructive or bypass procedures. Bypass procedure comprises nasal procedures which include septoplasty, functional rhinoplasty, nasal valve surgery, nasal polypectomy, and endoscopic procedures.

Oropharyngeal and nasopharyngeal procedures include uvulopalatopharyngoplasty, palatal advancement pharyngoplasty, tonsillectomy, and excision of tori mandibularis.

Hypopharyngeal procedures include tongue reduction, partial glossectomy, lingual tonsillectomy, and mandibular advancement. Laryngeal procedures include epiglottoplasty and hyoid suspension. Global airway procedure consists of maxillo-mandibular advancement bariatric surgery. Uvulopalatopharyngoplasty is the most widely performed surgery for OSA[31].

CONCLUSION

OSAS is a condition that results in significant morbidity and mortality. Hence, dentists must be aware of the signs and symptoms of OSAS such that they can confirm the diagnosis, and treatment can be initiated at the appropriate time. In patients with anatomic abnormalities of the maxilla and mandible resulting in a narrow pharyngeal airway, orthognathic surgery proves to be an excellent choice of treatment.

References

1. Singh A, Prasad R, Garg R, Kant S, Hosmane GB, Dubey A, et al. A study to estimate prevalence and risk factors of obstructive sleep apnoea syndrome in a semi-urban Indian population. Monaldi Arch Chest Dis 2017;87:773.
2. R.C. Stansbury, P.J. StrolloClinical manifestations of sleep apnea J Thorac Dis, 7 (2015), pp. E298-E310.
3. Young T, Palta M, Dempsey J, Skatrud J, Weber S, Badr S, et al. The occurrence of sleep-disordered breathing among middle-aged adults. N Engl J Med 1993;328:1230-5.
4. Hoffestein V, Szalai J. Predictive value of clinical features in diagnosing obstructive sleep apnoea. Sleep. 1993;16:118
5. Dempsey JA, Veasey SC, Morgan BJ, O'Donnell CP. Pathophysiology of Sleep Apnea. Physiol Rev 2010; 90: 47–112.
6. Kato M., Adachi T., Koshino Y., Somers V. K. Obstructive sleep apnea and cardiovascular disease. Circ J. 2009;73(8):1363–1370. [PubMed] [Google Scholar]
7. Victor L. D. Obstructive sleep apnea. Am Fam Physician. 1999;60(8):2279–2286.
8. Morrison D. L., Launois S. H., Isono S., Feroah T. R., Whitelaw W. A., Remmers J. E. Pharyngeal narrowing and closing pressures in patients with obstructive sleep apnea. Am Rev Respir Dis. 1993;148(3):606–611
9. Horner R. L., Mohiaddin R. H., Lowell D. G., et al. Sites and sizes of fat deposits around the pharynx in obese patients with obstructive sleep apnoea and weight matched controls. Eur Respir J. 1989;2(7):13–22.
10. Lowe A. A., Ono T., Ferguson K. A., Pae E. K., Ryan C. F., Fleetham J. A. Cephalometric comparisons of craniofacial and upper airway structure by skeletal subtype and gender in patients with obstructive sleep apnea. Am J Orthod Dentofacial Orthop. 1996;110(6):653–664.
11. Tangel D. J., Mezzanotte W. S., Sandberg E. J., White D. P. Influences of NREM sleep on the activity of tonic vs. inspiratory phasic muscles in normal men. J Appl Physiol. 1992;73(3):1058–1066.
12. Martin S. E., Marshall I., Douglas N. J. The effect of posture on airway caliber with the sleep-apnea/hypopnea syndrome. Am J Respir Crit Care Med. 1995;152(2):721–724.

13. Wiegand, D.A., Latz, B., Zwillich, C.W., and Wiegand, L. Upper airway resistance and geniohyoid muscle activity in normal men during wakefulness and sleep. *J Appl Physiol*. 1990; 69: 1252–1261
14. Rowley, J.A., Zahn, B.R., Babcock, M.A., and Badr, M.S. The effect of rapid eye movement (REM) sleep on upper airway mechanics in normal human subjects. *J Physiol*. 1998; 510: 963–976
15. Berthon-Jones, M. and Sullivan, C.E. Ventilation and arousal responses to hypercapnia in normal sleeping humans. *J Appl Physiol*. 1984; 57: 59–67
16. Shea, S.A., Edwards, J.K., and White, D.P. Effect of wake-sleep transitions and rapid eye movement sleep on pharyngeal muscle response to negative pressure in humans. *J Physiol*. 1999; 520: 897–908
17. Agarwal K, Mehta C, Sen MK, Suri JC. Evaulation & workup to detect regions of disproportionate anatomy and its subsequent management in patients of snoring and OSA. Indian J Sleep Med 2006; 1.1: 45-9 20.
18. Suri JC, Sen MK, Ojha UC, Adhikari T. Epidemiology of sleep disorders in the elderly – A questionnaire survey. Indian J Sleep Med 2009; 4.1:12-8 21.
19. World Health Organisation: Global Health Ob s e r v a t o r y (GHO) .Av a il a b l e a t: http://www.who.int/gho/ncd/risk_factors/obesi st ty_text/en/. Accessed on September 21 , 2013. 22.
20. National Family Health Survey (NFHS-3). National Fact Sheet. INDIA (Provisional data). International Institute for Population Sciences, Mumbai. Ministry of Health and Family Welfare. Government of India. 2005-2006
21. Obstructive sleep apnea. Available at: https://www.mayoclinic.org/diseases-conditions/obstructive-sleep-apnea/symptoms-causes/syc-20352090
22. Gyulay S, Olson LG, Hensley MJ, King MT, Allen KM, Saunders NA, et al. A comparison of clinical assessment and home oximetry in the diagnosis of obstructive sleep apnea. Am Rev Respir Dis. 1993;147:50–3
23. Obstructive sleep apnea. Available at https://www.mayoclinic.org/diseases-conditions/obstructive-sleep-apnea/diagnosis-treatment/drc-20352095
24. Flemons WW, Tsai W. Quality of life consequences of sleep disordered breathing. J Allergy Clin Immunol 1997; 99:S750-S756.
25. Paiva T, Farinha A, Martins A, Batista A, Guilleminault C. Chronic headaches and sleep disorders. Arch Intern Med 1997; 157:1701-5.
26. Wright J, Johns R, Watt I, Melville A, Sheldon T. Health effects of obstructive sleep apnea and the effectiveness of continuous positive airway pressure: a systemic review of the research evidence. BMJ 1997; 314:851-860.
27. Loube DI, Loube AA, Mitler MM. Weight loss for obstructive sleep apnea: The optimal therapy for obese patients. J Am Diet Assoc. 1994;94:1291–5.
28. Sullivan CE, Issa FG, Berthon-Jones M, Eves L. Reversal of obstructive sleep apnoea by continuous positive airway pressure applied through the nares. Lancet. 1981;1:862–5.
29. Schmidt-Nowara W, Lowe A, Wiegand L, Cartwright R, Perez-Guerra F, Menn S, et al. Oral appliances for the treatment of snoring and obstructive sleep apnea: A review. Sleep. 1995;18:501–10.
30. Thatcher GW, Maisel RH. The long-term evaluation of tracheostomy in the management of severe obstructive sleep apnea. Laryngoscope. 2003;113:201–4.
31. Friedman M, Ibrahim H, Bass L. Clinical staging for sleep-disordered breathing. Otolaryngol Head Neck Surg. 2002;127:13–21.