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REVIEW ARTICLE

OBSTRUCTIVE SLEEP APNEA

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ABSTRACT

Obstructive sleep apnea hypopnea syndrome is an important medical condition which is on the increase in the past 50 years. It causes significant morbidity and mortality in both developed and developing nations of the world.. Literature review was carried out on the pathophysiology, clinical presentation, complications, diagnostic modalities, and various treatment options. Additional information was obtained by cross referencing on other related journals using computerized search.

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INTRODUCTION

Our understanding of the nature and consequences of upper airway obstruction in adults during sleep has evolved considerably over the past two decades. Sleep apnea , defined as repeated episodes of obstructive apnea and hypopnea during sleep, together with day time sleepiness or cardiopulmonary function ,is common (Obstructive sleep apnea, 1996). Obstructive sleep apnea syndrome is defined by an apnea-hypopnea index (total no. of episodes of apnea and hypopnea per hour of sleep) of 5 or higher in association with excessive day time somnolence. OSA is becoming increasingly common with at least moderate disease now evident in 17% of middle aged men and 9% of women in middle aged population (Recent advances in OSA, 2015; Increased prevalence of sleep disordered breathing adults, 2013). The differential diagnosis include simple snoring, central sleep apnea, and other disorders that cause day time sleepiness. Eg: insufficient sleep, a circadian rhythm abnormality, narcolepsy, periodic limb movement disorder etc (Obstructive sleep apnea, 2002).

Symptoms

Signs and symptoms of obstructive sleep apnea include:

- Excessive sleepiness during day time
- Loud snoring
- episodes of breathing cessation during sleep
- Abrupt awakenings with gasping or choking
- dry mouth or sore throat
- Morning headache
- Difficulty in concentrating during the day
- mood changes, such as depression or irritability
- hypertension
- Nighttime sweating
- Decreased libido

Distinctive features of OSA

- Cessation of airflow for >10 seconds despite continuing ventilatory effort.
- 5 or more episodes per hour of sleep.
- Usually associated with a decrease of >/= 4% of oxyhaemoglobin saturation.

Etiology

- OSA occurs when the muscles of throat and tongue are more relaxed causing the airway to become blocked.
- High surface tension of upper airway liquid lining.
- Craniofacial skeletal restriction, enlarged upper airway soft tissues.

Risk factors

- **Obesity:** adipose tissue deposits around the laryngeal and pharyngeal areas may obstruct the airway

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- **Narrowed airway:** naturally narrow airways may be inherited or due to enlarged tonsils and adenoids.
- **Hypertension:** Obstructive sleep apnea is relatively common in people with hypertension.
- **Chronic nasal congestion:** Obstructive sleep apnea occurs twice as often in those who have consistent nasal congestion at night.
- **Smoking:** People who smoke are more likely to have obstructive sleep apnea.
- **Diabetes:** Obstructive sleep apnea may be more common in people with diabetes.
- **Sex:** In general, men are twice as likely as women to have obstructive sleep apnea.
- **A familial history of sleep apnea:** genetically determined craniofacial features or abnormalities in ventilator control may account for this pattern
- **Asthma.** Recent research has found an association between asthma and the risk of obstructive sleep apnea.

Pathogenesis: Narrowing or closure may occur at one or more sites in an unstable upper airway (i.e., in the velopharynx, oropharynx, or hypopharynx. Upper-airway dysfunction and the specific sites of narrowing or closure are influenced by the underlying neuromuscular tone, upper airway muscle synchrony, and the stage of sleep. These events are generally most prominent during rapid-eye-movement (REM) sleep because of the hypotonia of the upper-airway muscles characteristic of this stage of sleep. If the soft palate is exposed to recurrent vibratory trauma (snoring) and high negative inspiratory pressure, the result can be lengthening of the soft palate due to stretching and thickening caused by edema. It is possible that the changes in the soft palate of patients with sleep apnea may thus be a consequence of breathing against increased upper-airway resistance rather than the cause of that increased resistance.[3]

Pathophysiological consequences

- Day time fatigue and sleepiness
- Cardiovascular disorders-
- sudden decrease in blood oxygen levels during OSA increases blood pressure and strains cardiovascular system
- increases the risk of abnormal heart rhythms(arrhythmia). These abnormal rhythms can lower blood oxygen levels. If there is underlying heart disease, these repeated multiple episodes of low blood oxygen could lead to sudden death from a cardiac event
- Occular disorders- some researchers have found a connection between OSA and certain eye conditions like glaucoma
- Sleep deprived patterns- loud snoring can keep those around the patient from getting good rest and eventually disrupt the relationship

Diagnosis

- Loud snoring, fatigue, or both are frequently the patient's only symptoms
- Other patient characteristics associated with sleep apnea include male sex; age of more than 40 years; habitual snoring; nocturnal gasping, choking, or resuscitative snorting; observed apnea; and a history of systemic hypertension.
- Symptoms of daytime somnolence, unrefreshing sleep, morning headaches, cognitive impairment, depression, nocturnal esophageal reflux, and nocturia are commonly reported, but do not distinguish sleep apnea from other, nonpulmonary, sleep disorders.
- The presence of certain physical characteristics should heighten the physician's suspicion of upper-airway dysfunction during sleep. Retrognathia and discrete upper-airway abnormalities, such as an enlarged soft palate or tonsillar hypertrophy, are clinical clues.
- An increased body-mass index, hypertension, and an increased neck circumference often characteristic of patients with sleep apnea.
- A sleep study should be strongly considered for two groups of patients:
- those who habitually snore and report daytime sleepiness
- those who habitually snore have observed apnea (regardless of daytime symptoms).

Sleep studies

- A sleep study is performed to confirm the presence of upper-airway closure during sleep
- A full night of polysomnography, conducted by a technologist in a sleep laboratory, has traditionally been regarded as the gold standard for diagnosing sleep apnea.
- Portable, unattended monitoring systems that can be used outside the hospital premise is a more cost-effective system.
- The use of polysomnography requiresrecording the following physiologic signals
  - EEG
  - EOG(Electro Occulogram)
  - Chin electromyogram
  - Airflow
  - Oxygen saturation
  - Respiratory effort
  - ECG
- Additional recommended parameters include body position and leg EMG derivation
- Anterior tibilais EMG is useful to assist in detecting movement arousal and may have added benefit of assessing periodic limb movements, which co-exist with sleep relates breathing disorders.

Treatment: Therapeutic strategies for patients with sleep apnea may be grouped into three general categories: behavioral, medical, and surgical.

The goals of treatment

- to establish normal nocturnal oxygenation and ventilation.
- abolish snoring
- eliminate disruption of sleep due to upper-airway closure.

Behavioral Treatment: Avoiding factors that increase the severity of upper-airway obstruction such as sleep deprivation, the use of alcohol, sedatives, and hypnotic agents and obesity.

- Alcohol selectively reduces upper-airway muscle tone and increases the frequency of abnormal breathing during sleep. Alcohol also prolongs apnea by delaying arousal.
- In obese patients, weight loss can significantly decrease the severity of the apnea.
- In some patients, upper-airway dysfunction is present only during sleep in the supine position; training these patients to sleep exclusively in the lateral recumbent position may be useful.

Medical treatment

CPAP – continuous positive airway pressure delivered through a mask, is the initial treatment of choice in clinically important sleep apnea. The level of positive pressure required to sustain patency of the upper airway during sleep should be determined in the sleep laboratory. Patients treated with continuous positive airway pressure delivered nasally have repeatedly demonstrated improvement in neuropsychiatric function and a lessening of daytime sleepiness. Serious complications of therapy with continuous positive airway pressure are rare. Side effects reported by patients usually involve discomfort or irritation related to the nasal mask.. Patients may complain of nasal congestion, dryness, or occasional rhinorrhea. Nasal congestion can be treated with antihistamines or topical corticosteroids. Topical saline sprays or humidification will improve nasal dryness. Some patients may complain of increased resistance to exhalation or a sensation of too much pressure in the nose. A system that is fitted with a ramp may solve this problem. The ramp allows a gradual increase in the positive pressure to the prescribed level over a period of 5 to 45 minutes. Medication: Protriptyline and fluoxetine have been used with varying degrees of success in mild cases but are of little help in more severe cases. In hypothyroids, thyroxine replacement may significantly improves airway function during sleep. Nocturnal oxygen therapy for patient who have severe desaturation. Oxygen will decrease the nadir of oxyhaemoglobin saturation. Application of exogenous topical surfactants to the upper airway have shown reduction in sleep apnea. Nasal breathing have been shown to increase oral mucosal wetness and decrease the surface tension of upper airway liquid lining, where as the oral breathing route has the opposite effect.

Oral Appliances: Though positive airway pressure is often an effective treatment, oral appliances are an alternative for some people with mild or moderate obstructive sleep apnea. The appliances are worn only during sleep and are generally well tolerated. Patients with mild sleep apnea who do not tolerate therapy with positive airway pressure are good candidates for a trial of an oral appliance. Close collaboration between the physician and the dental consultant is necessary to ensure optimal patient selection and to avoid any alteration of dental occlusion or temporomandibular- joint discomfort.[10]

Tongue retaining device: This device has a room for the tongue and pulls it forwards. It enlarges the airway by keeping the tongue in an anterior position during sleep

Mandibular advancement devices: These appliances forces the mandible forwards and enlarges the airway. Eg: antisnoring splint. Herbst appliance

Tongue retaining device

Mandibular advancement devices

Surgical Treatment

Upper airway bypass

Tracheostomy: The availability and acceptance of positive-pressure therapy have lessened the need for tracheostomy. There remains a small subgroup of patients with severe apnea who cannot tolerate positive pressure and for whom other interventions are ineffective or unacceptable. A tracheostomy can provide dramatic improvement and can be lifesaving.

Palatal surgery: Surgery to modify, rather than bypass, a specific site of upper-airway closure, although less disfiguring than tracheostomy, offers more variable results. The most commonly performed procedure, uvulopalatopharyngoplasty, is curative in less than 50 percent of patients. Laser-assisted uvulopalatopharyngoplasty has been used as an outpatient treatment for snoring.

Even when the technical results of surgery are good, obstruction may continue at the site of surgery in the soft palate, or elsewhere in the upper airway. This procedure is currently not recommended for the treatment of sleep apnea.

Maxillofacial surgery: Genioglossal advancement, with or without resuspension of the hyoid bone, and may be performed in conjunction with a uvulopalatopharyngoplasty.

Patients with sleep apnea who have major craniofacial abnormalities, or who have had an unsuccessful genioglossal advancement, with or without uvulopalatopharyngoplasty, may benefit from a maxillomandibular advancement surgery.

Implantable devices: Nocturnal activation of lingual muscles by hypoglossal nerve stimulation has been investigated as a mean to overcome the sleep related decrease in pharyngeal dilator muscle tone to maintain patent airway. Stimulation of genioglossal motor activation has shown to improve airflow. Hypoglossal nerve stimulation to activate the lingual muscles is available via implantable devices. These devices consist of pulse generator implanted in the chest wall with an electrical lead extending unilaterally to an electrode cuff that wraps around the hypoglossal nerve.

Conclusion

Obstructive sleep apnea is a significant public health problem with increasing prevalence along with aging population and increasing obesity levels. It is increasingly appreciated that OSA is a heterogenous disorder and multiple pathophysiologic causes are now recognized. Upper airway anatomy and collapsibility remains a fundamentally important patho physiological factor. There are noe a number of different treatment options to address OSA with variable treatment effectiveness. Patients phenotyping of OSA pathophysiology is therefore likely to inform treatment options and improves treatment outcome in future (Recent advances in OSA, 2015).

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