

# Chronic Obstructive Sleep Apnoea- A Review

Dr. Balakrishnan<sup>1</sup>, Dr. VijayEbenezer<sup>2</sup>, Dr. WasimAhmed<sup>3</sup>, Dr. Vigil Dev Asir<sup>4</sup>

*Department of Oral and Maxillofacial Surgery, Sree Balaji Dental College and Hospital, Bharath University, Chennai- 600100, Tamil Nadu, India.*

*Email- [drbalakrishnanomfs@gmail.com](mailto:drbalakrishnanomfs@gmail.com)*

## **ABSTRACT:-**

*obstructive sleep apnoea hypopnea syndrome is an important medical condition brought to limelight in the last five decades. It is a major cause of morbidity and significant cause of mortality worldwide, including developed and developing nations. OSA is a sleep disorder that involve cessation or significant decrease in airflow in the presence of breathing effort. It is most common type of sleep disordered and is characterized by recurrent episodes of upper airways collapse during sleep, usually at the level of soft palate. The golden standard diagnosis is by polysomnography. Treatment includes positive continuous airway pressure, oral appliance and several surgical procedure Conservative procedures includes alcohol & tobacco cessation, weight loss*

**KEYWORDS:-***Apnea, snoring, polysomnography, uvulopalatopharyngoplasty*

## **INTRODUCTION:-**

Repeated episodes of partial or complete upper airways obstructing during sleep .A sleep disorder characterized by recurrent episodes of narrowing or collapse of pharyngeal airways during sleep despite ongoing breathing effort. The first description of the disorder in the medical literature was in 1965<sup>(1)</sup>. Obstructive events are characterized by continued thoracoabdominal effort in the setting of partial or complete airflow cessation.

Mild Sleep Apnoea, AHI : 5-15 events per hour

Moderate Sleep Apnoea, AHI : 15-30 event per hour

Severe Sleep Apnoea, AHI : greater than 30 events per hour

Three different types of apnoea can be distinguished. Firstly, central sleep apnoea in which cessation of breathing is caused by a disturbance in ventilatory control at the level of the central nervous system(2); and there is no effort to breathe when asleep. Secondly, obstructive sleep apnoea in which the respiratory control is normal, but there is an obstruction, usually at the level of the pharynx, which physically interrupts the flow of air although vigorous effort to breathe on the part of patient is present. Lastly, mixed sleep apnoea which is a combination of both components. Obstructive sleep apnoea is by far the most common disorder. A typical patient with obstructive sleep apnoea syndrome will have 30 or more apnoeas per hour of sleep, each lasting for not less than 10 seconds; along with apnoea there will be a concurrent drop in oxygen saturation, sometimes to dramatically low levels. In middle age , the prevalence of over OSA is approximately 4% in men and 2% in women. It is estimated that 80% of the patient are undiagnosed with sleep study data estimating sleep disordered breathing having a prevalence of 24% in men and 9% in women. OSA severity is highest in Asian due to craniofacial characteristics OSA Is mainly due to soft tissue collapse in the pharynx. Transmural pressure is the difference between intraluminal pressure and the surrounding tissue pressure .If transmural pressure decreases, the cross sectional area of the pharynx decreases. If this pressure passes a critical point, pharyngeal closing pressure is reached .Exceeding pharyngeal critical pressure cause tissue collapsing inward .The airway is obstructed.

Anatomic factors includes Enlarged tonsil, Volume of the tongue, Soft tissue, Lateral pharyngeal walls, Length of the soft palate, Abnormal positioning of maxilla and mandible, decreased neuromuscular activity in the UA, Including reflex activity, reduced ventilatory motor output to upper airways muscles.

Etiology includes Retrognathia and micrognathia, Mandibular hypoplasia, Brachycephalic head form, Inferior displacement of the hyoid Pierre robin syndrome, Nasal obstruction-polyps, septal deviation, tumors, trauma and stenosis. Retropalatal obstruction-elongated, posteriorly placed palata and uvula, tonsil and adenoid hypertrophy OSA are independently linked to hypertension in pregnancy.. Some reports have associated fetal growth retardation with OSA

#### **DIAGNOSIS OF OSA:**

History of snoring, Restless sleep, Headaches And day time sleepiness should alert to the possibility of OSAS

#### **POLYSOMNOGRAPHY:-**

PSG examination includes Recording of heart rhythm, Electrocephalography, Blood pressure, Eye movement, Electromyography, Snoring volume, Oro – nasal airflow, Peripheral pulse oximetry(10) Consequences of OSA includes Hypoxia, Neurovascular endothelial dysfunction, Excessive daytime sleepiness and sleep fragmentation, Cardiovascular consequences of OSA, Hypertension, MI& stroke, CCF, Arrhythmia, pulm(5)

#### **TREATMENT OF OSA:-**

The decision to treat OS should be based on its severity, related symptoms and medical comorbidities:-

Treatment option available are positive airway pressure therapy, general measures and weight loss, intraoral devices, position therapy, nasal expiratory positive airway pressure, pharyngeal muscle stimulation, Surgery, Oxygen, Positive airway pressure therapy. Colin Sullivan et al first described the use of nasal CPAP to treat OSA in 1981. *Complication associated with CPAP includes* nocturnal arousals, Rhinitis, nasal irrigation and dryness, aerophagia, Mask and mouth leaks, facial rash or irritation, difficulty with exhalation(4) Oral devices helps to alter the position of the upper airway structures which enlarging airways calibre or reducing airways collapsibility during sleep, Tongue retaining devices, Palatal lifting device(3) Position therapy is indicated in position dependent sleep apnea. It is defined as SDB in which the AHI while asleep in the supine position is at least twice as high as in lateral position. The prevalence of position dependent sleep apnea may be as high as 50% to 70% in patients with OSA. The lateral position is associated with increased maximum cross sectional upper airways area and lower closing pressure. Lateral positioning can be attained by tennis ball, backpack, thoracic antisupine band, zoma positional sleepers(6)

#### **NASAL EXPIRATORY POSITIVE AIRWAY PRESSURE:-**

Nasal expiratory positive airway pressure is delivered via a novel device with one way mechanical valve that provide high expiratory resistance in the setting of very low inspiratory resistance(7). The nasal EPAP device is applied to each nostril using adhesive and is designed for one time use. It is approved by FDA in 2008

#### **SURGICAL TREATMENT FOR OSA:-**

A variety of surgical options are available, however, the success of these treatment, aside from tracheostomy is less well established and generally less effective than CPAP therapy. Fiberoptic laryngoscopy, drug induced sleep endoscopy, or MRI imaging can be used to classify the obstruction of the airways at the nasal, oropharyngeal and /or hypopharyngeal level. Phase 1 surgeries refers to obstruction nasal, palatal and lingual level(8). They are typically performed first with subsequent use of a phase 2 surgery such as maxillomandibular advancement if needed. Tracheotomy is virtually 100 % effective in eliminating obstructive. It is generally reserved for patient with severe OSA who manifest severe complication such as malignant arrhythmias without treatment. Laser assisted uvulopalatoplasty is not recommended as a treatment option for OSA by AASM

### **MEDICATION:-**

Specifically selective serotonergic reuptake inhibitor, protriptyline, methylxanthine derivatives and estrogen therapy are not recommended(9). Patients with hypothyroidism and acromegaly should be treated with thyroid replacement and somatostatin analogs, respectively as such treatment can improve AHI. Topical nasal steroids if rhinitis is present. Modafinil and armodafinil can be used

### **DISCUSSION:-**

It has been reported that 60%-70% of patients with OSA are obese. Obesity predisposes to the development of OSA by causing a narrowing of the pharynx and aggravates the hypoxemia of OSA by reducing functional residual capacity, thus creating areas of ventilation/ perfusion mismatch. Weight loss of even 5 kg- 10 kg is beneficial. Although the mechanism of improvement is not fully understood, it is probably multifactorial, consisting of improvement in pharyngeal structure and function, neuromuscular control, and an increase in functional residual capacity(11,12). The extent of weight loss and the degree of improvement are not always directly related, although it has been shown that a 1% change in weight is associated with a 3% change in AHI. Bariatric surgery should be considered in all patients with BMI 35 mg/kg<sup>2</sup> or more.<sup>14</sup> A number of medications, such as nasal decongestants, respiratory stimulants, and tricyclic antidepressants have been used in treating patients with OSA. However, the efficacy of this therapy has not yet been established. One agent which has been tested in a small, double-blind, controlled trial is protriptyline. It works by suppressing the REM sleep, where most episodes of severe hypoxemia occur. Recent animal work suggests that this drug may also increase the tone of the upper airway muscles, thus stabilizing the airway and making it less susceptible to collapse. The anticholinergic side effects of protriptyline(13), such as dry eyes, dry mouth, difficulty in initiating urination, and difficulty with ejaculation are common with the dosages used (10-30 mg) and limit the usefulness of this medication. Stimulants like Amphetamines, Methylphenidate, and Modafinil increases alertness. They are most often used in the treatment of Excessive Daytime Sleepiness (EDS) seen in patients with OSA. Modafinil, like other CNS stimulants, increases the release of monoamines, specifically the catecholamines, norepinephrine and dopamine from the synaptic terminals. However, Modafinil also elevates the hypothalamic histamine levels which make it a "wakefulness promoting agent" rather than a classic amphetamine-like stimulant. Several other drugs like acetazolamide, medroxyprogesterone, theophylline, doxapram and almitrine are under investigation. the surgeries that can be offered to patients with OSA include nasal surgeries (septoplasty, sinus surgery and others), Tonsillectomy with/without adenoidectomy, uvulopalatopharyngoplasty (UPPP), laser assisted uvuloplasty (LAUP), radiofrequency volumetric tissue reduction, sliding genioplasty and maxillo-mandibular advancement osteotomy. In patients with idiopathic OSA (i.e., without obvious anatomical deformities) uvulopalatopharyngoplasty (UPPP) has recently become popular. UPPP was introduced by Fujita and colleagues in 1981 in US for the first time. This operation involves tonsillectomy, uvulectomy, partial resection of the soft palate, and removal of redundant pharyngeal mucosa.

### **CONCLUSION:-**

In recent few years the field of sleep medicine has undergone numerous changes; advances as far as the diagnosis, consequences and management. Different treatment options are now available for effective management of OSA. CPAP is highly effective in controlling symptoms, improving quality of life and reducing the clinical consequences of sleep apnoea and we must consider it as a first-line option(14). Bilevel PAP and Auto-CPAP can be used in those patients intolerant to CPAP or when high treatment pressures are necessary. Mandibular advancement devices can be offered as a viable alternative to patients with mild to moderate OSA, intolerant to PAP. The role of surgery remains controversial. Tonsillectomy and adenoidectomy are useful in children and in adults with enlarged tonsils. Uvulopalatopharyngoplasty is a well established procedure to be considered as a second-line option when PAP has failed. Maxillary mandibular surgery is extremely effective and can be suggested to patients with craniofacial malformations. All patients with obesity should be encouraged to lose weight and bariatric surgery can be considered in patients with BMI over 40. A multidisciplinary approach and the implementation of educational programs will significantly improve the management of the disease.

Ethical clearance – Not needed as it is review article

Source of funding- Nil

Conflict of interest- Nil

**References:-**

1. Dickens C. The posthumous papers of the Pickwick club. London: Capman and Hall, 1836- 1837.
2. Douglas NJ, Thomas S, Jan MA. Clinical Value of Polysomnography. *Lancet* 1992;339:347.
3. Douglas NJ. Systematic review of the efficacy of nasal CPAP. *Thorax* 1998;53:414
4. Duran J, Esnaola S, Rubio R, et al. Obstructive sleep apnoeahypopnoea and related clinical features in a population based sample of subjects aged 30-70 yr. *Am J Respir Crit Care Med* 2001;163: 685-689.
5. Engleman HM, Martin SE, Deary IJ, et al. Effect of CPAP therapy on day-time function in patients with mild sleep apnoea/hypopnoea syndrome. *Thorax* 1997;52:114.
6. Gastaut H, Tassinari CA, Duran B. Etude polygraphique des manifestations episodique (hypnique et respiratoires), diurne et nocturne, du syndrome de Pickwick. *Rev Neurol* 1965;112:568-579.
7. Guileminault C, Abad VC. Obstructive sleep apnoea syndromes. *Med Clin North Am* 2004;88:611-630.
8. Guileminault C, Kirisoglu C, Poyares D, et al. Upper airway resistance syndrome: A long-term outcome study. *J Psychiatr Res* 2006;40:273-279.
9. Hirshkowitz M, Sharafkhaneh A. Positive airway pressure therapy of OSA. *Semin Respir Crit Care Med* 2005;26:68-79.
10. Hoffstein V, Szalai J. Predictive value of clinical features in diagnosing obstructive sleep apnoea. *Sleep* 1993;16:118.
11. Hudgel DW, Harasick T, Katz RL, et al. Uvulopalatopharyngeoplasty in obstructive apnoea: value of pre-operative localization of site of upper airway narrowing during sleep. *Am Rev Respir Dis* 1991;143:942.
12. Keenan SP, Burt H, Ryan F, et al. Long-term survival of patients with obstructive sleep apnoea treated by uvulopalatopharyngoplasty or nasal CPAP. *Chest* 1994;105:155.
13. Li KK. Surgical therapy for adult obstructive sleep apnoea. *Sleep Med Rev* 2005;9:201-209.
14. Mortimore IL, Marshall I, Wraith PK, et al. Neck and total body fat deposition in non-obese and obese patients with sleep apnoea compared with that in control subjects. *Am J Respir Crit Care Med* 1998;157:280