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Review Article

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## Review article on sleep apnea

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Article History	Abstract
Received on: 18-01-2021 Revised On : 24-02-2021 Accepted on : 28-02-2021	Obstructive sleep apnea (OSA) is a sleep-related breathing disorder that involves a reduces or complete halt in airflow despite an ongoing effort to breathe. It occurs when the muscles relax during sleep, causing soft tissue in the back of the throat to collapse and block the upper airway. This results in partial reductions (hypopneas) and complete block (apneas) in breathing that lasts a minimum of 10 seconds during sleep. Most pauses last between 10 and 30 seconds, but some may persist for one minute or longer. This can cause abrupt reductions in blood oxygen saturation. The brain responds to the lack of oxygen by alerting the body, causing a brief arousal from sleep that restores normal breathing. This pattern can occur many times in one night. The result is a fragmented quality of sleep that often produces an excessive level of daytime sleepiness. Most people with OSA snore loudly and regularly, with periods of silence when airflow is reduced or blocked. They then make choking, snorting or gasping sounds when their airway reopens. A common measurement of sleep apnea is the apnea-hypopnea index (AHI).
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### Introduction

Sleep apnea is a serious, potential life-threatening condition. It is a breathing disorder characterised by repeated collapse of the upper airway during sleep, with consequent cessation of breathing [1]. Virtually all sleep apnea patients have a history of loud snoring [2]. They may also unknowingly experience frequent arousals during the night, resulting in chronic daytime sleepiness or fatigue [3]. Sleep apnea occurs in about 25% of men and 10% of women. Sleep apnea can affect people of all ages, including neonates and children but is particularly common in people over the age of forty and those who are overweight [4]. Certain physical traits and clinical features are risk factors of obstructive sleep apnea [5]

These include excessive weight, large neck and structural abnormalities reducing the diameter of the upper airway such as nasal obstruction, a low – hanging soft plate, enlarge tonsils or a small jaw with an overbite [6].

There are two sorts of sleep apnea: obstructive and central. Obstructive sleep apnea (OSA) is that the more common of the two. OSA is characterised by repetitive episodes of complete or partial upper airway blockage during sleep [7]. During an apnea episode, the diaphragm and chest muscles work harder ass the pressure increases to open the airway. Breathing usually resumes with a loud gasp or body jerk [8]. These episodes can disturb sound sleep, reduce the flow of oxygen to vital organs, and cause heart rhythm irregularities [9].

In CSA, the airway is not blocked but the brain fails to signal the muscles to breath due to instability in the

respiratory control centre [10]. Central apnea is named as such because it is related to the function of the central nervous system [11].

### Symptoms

- Snoring
- Daytime sleepiness or fatigue
- Restlessness during sleep
- Sudden awakening with a sensation of gasping or choking
- Dry mouth or sore throat upon awakening
- Intellectual impairment, such as trouble concentrating, forgetfulness or irritability
- Night sweats
- Sexual dysfunction
- Headache
- Insomnia

Symptoms in children might not be as obvious and include:

- Poor school performance
- Sluggishness or sleepiness, often misinterpreted as laziness
- Inward movement of the ribcage when inhaling
- Unusual sleeping positions, such as sleeping on the hands and knees or with the neck hyper-extended
- Excessive sweating at night
- Learning and behavioural disorders
- Bed wetting

### Pathogenesis

#### Central sleep apnea

There are several forms of central sleep apnea. Periodic breathing develops in most individuals ascending to high altitude if the altitude is high enough [12]. This is a sort of ventilatory instability produced by ambient hypoxia. Hypoxia at altitude results in high controller gain, leading to hyperventilation and hypocapnia. In this situation the increased control gain is sufficient to overcome the somewhat reduced plant gain (low  $P_{CO_2}$ ) and ventilation becomes unstable as a result, ventilation waxes and wanes between apnea and hyperpnea. For this to occur, there must be in hypocapnia [13]. Thus, the periodic breathing at altitude occurs more commonly in individuals with high controlled gain [12]. However, most such individuals don't have periodic breathing at sea level. Thus, inherently high loop gain that is augmented further by altitude is required for this

respiratory pattern to emerge. Over time at altitude, the periodic breathing resolves due primarily to further decrements in plant gain that cannot be offset by a high controller gain [14]. Idiopathic central sleep apnea is a relatively uncommon disorder seen at sea level in individuals with high controller gain, generally on elevated hypercapnic ventilatory response  $P_{CO_2}$  drives ventilation during sleep at sea level as it does at altitude, and patients with idiopathic central sleep apnea tend to have low  $P_{CO_2}$  levels, even during wakefulness [15,16]. Another form of CSA called Cheyne-Stokes respiration is seen in patients with congestive heart failure and is a product of high controller gain, hypocapnia resulting from lung edema and a long circulation time. This combination of traits is particularly destabilizing to ventilation and yields a characteristic crescendo-decrescendo pattern of breathing, with a cycle time approximately 1 minute. As expected in a high loop gain disorder  $CO_2$  administration can regularize ventilation [17]. As in idiopathic CSA, Cheyne-Stokes respiration occurs primarily during non-REM [Rapid eye movement] sleep, although it can be detected during wakefulness if carefully sought. However, it is uncommon during REM sleep, again likely secondary to decreased controller gain, because both traits described above are generally required to manifest Cheyne-Stokes respiration, it is not seen in all patients with congestive heart failure, even those with severe congestive heart failure [18]. Patients with walking hypercapnia primarily due to ventilatory abnormalities or neuromuscular disease may have central apneas during sleep as well [19,20].

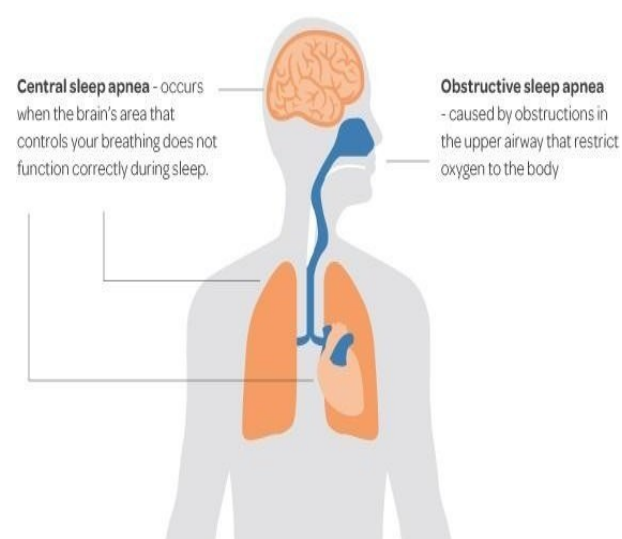
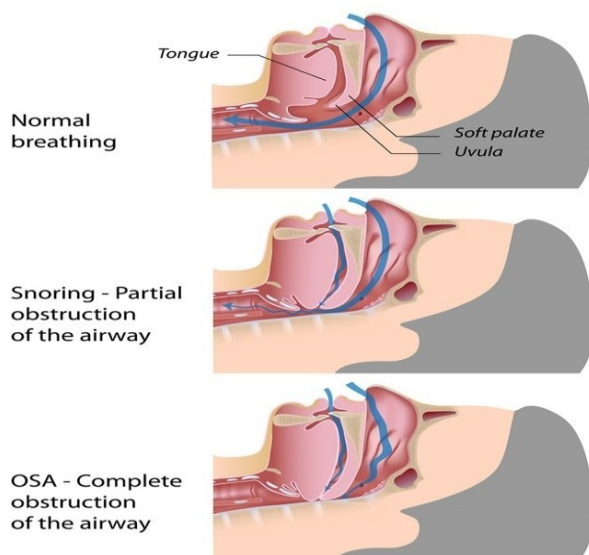


Fig 01: Central Sleep Apnea

## Obstructive sleep apnea

Obstructive apnea have an anatomically small pharyngeal airway like due to either increased soft tissue surrounding the airway or a small bony compartment in which the airway is enclosed [21]. During wakefulness, pharyngeal patency is maintained primarily by reflex-driven, augmented pharyngeal dilator muscle activity, which offsets the positive extraluminal tissue pressure collapsing the airway [22,23]. Normal ventilation is maintained, at sleep onset and/or during REM sleep, reflex muscle activation is reduced as is arousal-modulated excitatory output to the upper airway musculature [24]. Lung volume falls as well [25]. If the airway anatomy is quite deficient, these events alone will likely lead to substantial or complete airflow obstruction, yielding a hypopnea or apnea. As a result, hypoxia and hypercapnia develop, ventilation is stimulated, and often arousal from sleep in response to respiratory activation is required to reestablish airway patency to allow a recovery of ventilation [26,27].



**Fig 02: Obstructive Sleep Apnea**

## Diagnosis

If there's a high suspicion of sleep apnea after evaluating a patient, a sleep study is indicated to determine a diagnosis. Currently, polysomnography, which requires an overnight stay in a sleep laboratory, is the optimum test for diagnosing sleep apnea. It includes evaluation of sleep staging, airflow and ventilatory effort, arterial oxygen saturation, electrocardiogram, body position, and periodic limb movements. Polysomnography, however, may not be readily available. Other options to consider are evaluation using

pulse oximetry and portable (home) monitoring of cardiopulmonary channels. Although oximetry is currently being used to diagnose sleep apnea, its sensitivity and specificity are controversial [28]. A variety of home monitors are currently available or being developed that can record both cardiopulmonary parameters (for example, airflow, ventilatory effort, heart rate, and oxygen saturation) and sleep parameters and may be useful in diagnosing sleep apnea [29].

## Treatment

The goals of treatment for sleep apnea patients include both physiologic and symptomatic components. Physiologic goals of treatment include eliminating sleep fragmentation, apneas and hypopneas, and oxygen desaturation. Symptomatic goals include eliminating snoring and sleepiness, improving quality of life, and reducing or eliminating comorbidities. Symptomatic improvement, particularly decreased snoring, does not necessarily correlate with physiologic improvement or decreased morbidity [30].

## Behavioural approach

Behavioural measures may be the only treatment needed for patients with mild sleep apnea. Behavioral interventions include losing weight, eliminating evening alcohol and sedatives, and proper positioning (avoiding the supine position in bed). Although weight loss (accomplished through a comprehensive program or surgery) may be difficult to achieve, it can be very effective and, in some cases, even curative [31,32]. Patients with mild symptoms may experience improvement using behavioural techniques alone. Appropriate behavioural treatment should be implemented for all patients, even those requiring additional interventions. Patients treated with behavioural techniques should be reevaluated periodically after initiation of treatment [33]. For patients who have improved, continued support and positive reinforcement can sustain their adherence and success. In those patients who continue to experience symptoms, other therapies are warranted [34].

## Nasal continuous positive airway pressure

Continuous positive airway pressure (CPAP) is the most effective non-invasive therapy for sleep apnea. To use CPAP the patient must wear a sealed mask over the nose or in some cases, over the nose and mouth during sleep [35]. The mask is connected to a blower forcing air through the nasal passages. CPAP acts as a pneumatic

splint by increasing the pressure in the oropharyngeal airway, thereby maintaining airway patency throughout the ventilatory cycle [36]. This treatment is usually prescribed after polysomnography has first determined the therapeutic level of CPAP pressure required to reduce or eliminate sleep apnea [37]. CPAP is effective in reversing daytime somnolence and eliminating cardiopulmonary sequelae [38]. CPAP used properly, produces rhythmic breathing resulting in the patient feeling dramatically better and being able to function more efficiently. Compared with no treatment or other treatment modalities patients treated with CPAP have a lower mortality rate [39]. Although very effective, CPAP may be difficult for some patients to use [40]. Adherence to CPAP treatment varies greatly but tends to be higher in patients with severe symptoms [41]. The most common reasons for discontinuing CPAP are intolerance of the mask, nasal-related complaints, and the inconvenience of being connected to a machine. Common side effects include nasal stuffiness, rhinitis, facial skin discomfort, and discomfort with the pressure. Humidifiers, nasal steroids or decongestants, intranasal anticholinergics, or different masks may relieve side effects. Variations in pressure application have been developed to offer patients options for improving comfort. Assisting patients to focus on symptom reversal and working with home care companies to ensure proper-fitting and effective equipment will enhance adherence. Followup after the primary month of CPAP treatment should include checking the status kit, assessing patient symptoms and adherence, and assessing the status of coexisting conditions like hypertension. In patients who have achieved significant weight loss, the CPAP pressure may have to be adjusted. If the patient reports continued snoring, the pressure may need to be increased [42]

### **Oral/ dental appliances**

Oral or dental appliances could also be an option for patients with mild-to-moderate sleep apnea. However, they are not effective in all patients. Appliances have also been used for patients who snore but do not have sleep apnea. There are various devices that displace the tongue forward or move the mandible to an anterior and forward position to improve patency of the airway [43]. Reported side effects of the devices include excessive salivation and temporomandibular joint discomfort. A dentist or orthodontist experienced in the use of these devices should fit the patient, and a sleep

study should be done after the device is fitted to evaluate its effectiveness [44].

### **Surgical procedure**

Patients need to understand that no surgical procedure has universal success, and all are invasive and carry risk. Several procedures or a combination of procedures may need to be performed to help sleep apnea patients. It is important that sleep studies be repeated after each surgical procedure to confirm its effectiveness, once there is evidence of adequate healing. When weighing treatment options, it may be useful to let the patient know that CPAP is highly effective when used properly and is safe and reversible [45,46]. Uvulopalatopharyngoplasty (UPPP). During UPPP, an inpatient procedure, the uvula and portions of the soft palate are resected to widen the oropharyngeal airway. Although snoring is temporarily relieved in most cases, apnea may persist. The overall success rate of UPPP is reported to be about 40 percent (when success is defined as achieving an AHI of less than 20) [47]. It is difficult to predict which patients will benefit from this procedure, and long-term side effects and benefits are unknown.

### **Nasal Surgery**

Nasal surgery may be used alone or in conjunction with other procedures. However, it is rarely curative alone.

### **Tonsillectomy**

In children and adolescents adenotonsillectomy may be useful, even curative [48]. Tonsillectomy alone in adults is not usually helpful [49]. but is often done in conjunction with UPPP. Laser-Assisted Uvulopalatoplasty (LAUP): LAUP has received much attention recently as a treatment for snoring. However, its effectiveness in treating sleep apnea is unknown. LAUP differs from traditional UPPP in both surgical technique and setting (office-based). LAUP excises only part of the uvula and associated soft-palate tissues. The resultant shortening of the palate and reduction of the uvula may reduce or eliminate snoring. As with UPPP, relief of snoring may occur without improvement in apneic events. Therefore, patients who elect LAUP for snoring may risk delaying the diagnosis of sleep apnea because snoring, a primary symptom, is eliminated [50].

### **Maxifacial surgery**

(Genioglossal Advancement, Maxillary and Mandibular Advancement). These are specialized procedures that are currently not widely available, although they appear

to be effective in treating sleep apnea [51]. Genioglossal advancement enlarges the airway at the base of the tongue. This procedure may be combined with a UPPP. Maxillary and mandibular advancement enlarges the airway at the level of the soft palate as well as the tongue. Tracheostomy. Tracheostomy is highly successful in eliminating sleep apnea but is very invasive, both physically and psychologically. This procedure is reserved for severe cases where other treatments have failed [52].

### Pharmacological treatment

Currently, there are no safe and effective medications indicated in the routine treatment of sleep apnea.

### Oxygen

Administration of supplemental oxygen may improve nocturnal desaturation but is not a satisfactory treatment option by itself because it does not reduce sleep disruption and subsequent daytime sleepiness [53].

### Management considerations

The efficacy of a chosen treatment modality should be periodically and objectively verified. Sleep apnea patients who undergo surgical interventions need to have sleep studies repeated postoperatively, after healing has occurred. Once effective treatment has been initiated, all patients should be periodically reevaluated for recurrence of symptoms such as snoring and excessive daytime sleepiness as well as cardiopulmonary complications. The primary care physician can play a key role in determining if patients are adhering to treatment and in monitoring comorbidities such as hypertension and coronary artery disease. For example, hypertension treatment may need to be adjusted once sleep apnea has improved. Patients who are adherent to treatment for sleep apnea need positive reinforcement, and those who are not adherent may require different treatment options. Patients who are on CPAP need to have their equipment evaluated periodically to ensure that the machine and mask are functioning properly [54].

### Conclusion

there are a number of phenotypic traits that predispose an individual to the development of sleep apnea. In the case of central apnea, this generally relates to loop gain and circulation time. For obstructive apnea, pharyngeal anatomy, upper airway muscle responsiveness during

sleep, arousal threshold, and loop gain may all contribute to apnea presence and severity. The relative contribution of each may vary between patients. It is unclear at this time whether defining these traits in patients with apnea would have therapeutic implications, although this seems possible. Thus, as always, more information is needed.

### Authors contribution

all authors contributed equally to this work.

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