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Obstructive Sleep Apnoea: A Comprehensive Review

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Introduction

Sleep is that golden chain that ties health and our bodies together; any disruption in its pattern is detrimental to health.

Obstructive Sleep Apnea (OSA) is a common yet often underdiagnosed sleep disorder characterized by intermittent episodes of complete or partial upper airway obstruction during sleep. These disruptions lead to frequent arousals, decreased sleep quality, and various health complications. The disorder affects both adults and children, with significant long-term implications if left untreated. Obstructive sleep apnoea (OSA) affects 2% to 4% of middle aged adults.[1]

Obstructive sleep apnoea is the most common type of sleep apnoea and is caused by obstruction of the upper airway. It is characterized by repetitive pauses in breathing during sleep, despite the effort to breathe, and is usually associated with reduction in blood oxygen saturation. These pauses in breathing, called "apnoeas" typically last 20 to 40 secs. OSA causes both fragmented sleep and oxygen desaturation. It is characterized by repetitive pharyngeal collapse at the level of the soft palate or base of tongue. Obstructive sleep disordered breathing is relatively common medical problem. It consists of a spectrum ranging from apnoea to hypopnea. Obstructive apnoea refers to the temporary cessation of airflow during sleep for 10secs or more despite continuos effort, whereas hypopnea means reduction of 30% to 50% in airflow for 10secs or more. Obstructive sleep apnoea (OSA) affects 2% to 4% of middle aged adults.[2]

Classification

The severity of OSA is classified on the basis of the patient's AHI index into 3 categories:[2]

1. Mild OSA (5 to 15 events per hour)
2. Moderate OSA (15 to 30 events per hour)
3. Severe OSA (more than 30 events per hour)

Prevalence

Obese and overweight individuals have much higher chance of snoring or having obstructive sleep apnoea , more commonly seen in males , post-menopausal women are a higher risk ,highly prevalent in people older than 65yrs of age.[3]

Pathophysiology: Fig 1

The definitive event in OSA is occlusion of the upper airway at the level of oropharynx. This can be because of large tongue, small air pathway or abnormal throat anatomy. OSA occurs when the muscles in the throat relax excessively during sleep, causing a temporary blockage of the upper airway. [2] This leads to pauses in breathing (apneas) that can last from a few seconds to minutes and may occur hundreds of times during a night. As a result, oxygen levels in the blood decrease, and the brain partially wakes up to restore normal breathing. These disruptions prevent the body from entering deep, restorative stages of sleep, which is why individuals with OSA often feel fatigued despite having an apparently full night's rest. It is frequently accompanied by snoring but not everyone with sleep apnea snores.[3]

Etiology

Multifactorial sleep disorder. People with narrow airways more likely to get affected by OSA.

Reduced upper airway caused by:

1. Obesity
2. Adenotonsillar hypertrophy
3. Mandibular deficiency
4. Macroglossia
5. Upper airway tumors

- Excessive pressure across the collapsible segment, attributed to nasal obstruction
- Alcohol or tranquillizers: important cofactor because of its selective depressant influence on the upper airway muscles. They lower "respiratory drive" in the nervous system, thereby reducing breathing rate and strength.[4]

Risk factors

- Male sex hormones – cause changes in the size or structure of upper airway
- Pregnancy – weight gain can affect breathing pattern during sleep, particularly in third trimester.
- Family history – known to run in families although no genes or gene associated disorder have been identified.
- Smoking – causes inflammation and narrowing of upper airway.

Signs and symptoms

Snoring, Nocturnal asphyxia, Daytime sleepiness, Morning headaches, Disorientation upon waking, Poor judgement, Personality changes, Night sweats and nocturia.

Clinical Features

- Narrow oropharynx or oropharyngeal edema or erythema
- Obesity
- Macroglossia
- Enlarged, low-lying, edematous or erythematous uvula
- Adenotonsillar enlargement
- Retrognathia or micrognathia
- Upper airway tumours
- Systemic hypertension

- Signs of pulmonary hypertension or cor pulmonale
- Plethora

Diagnosis

Patients present with vague, nonspecific complaints such as fatigue and impaired concentration with daytime somnolence. Diagnosing OSA typically involves a combination of patient history, physical examination, and polysomnography (a sleep study). [5]

Medical diagnosis

Medical history will include questions about alcohol or tranquilizer use, snoring, morning headaches or disorientation.[6,7]

1. It is important to ask the bed partner about interrupted snoring patterns, apneic periods, nocturnal gasping or choking attacks.
2. BMI and neck circumference as indications of obesity are good predictors of OSA.
3. Multiple sleep latency test – to establish how rapidly the patient falls asleep to distinguish from narcolepsy.
4. Epworth sleepiness scale – to screen for sleep apnoea[8] Fig 2
5. Mallampati score – higher the grade, smaller the air passage, indicating sleep disordered breathing[9]Fig 3
6. Enlarged tongue
7. Lateral cephalometric radiographs – reveal diversion of airway column, position of hyoid bone and craniofacial skeleton for any maxillomandibular deficiencies.
8. Fibro-optic nasopharyngoscopy – reveal anatomic obstruction of airway
9. Hypertension is common in OSA patients.

Laboratory investigations

Polysomnography is considered the gold standard for diagnosis, where various parameters, including brain activity, eye movement, heart rate, and respiratory effort, are monitored during sleep. However, access to sleep studies can be limited, and home sleep tests (HST) have been introduced as more convenient and cost-effective options, although they may lack some of the detail of in-lab testing.[10]

1. Polysomnography:

Conducted during an overnight stay in a specialized sleep laboratory. It is considered the gold standard for diagnosis of OSA.

Important parts of study include measurements of:

- Heart rate
- Airflow at the mouth and nose
- Respiratory effort
- Sleep stage (light sleep, deep sleep, dream sleep,etc.)
- Oxygen level in blood

2. Split- Night Polysomnographic studies:

- Normally, patients are studied over two nights; first – for diagnosis, second – for CPAP titration.
- Instead split the night into two halves; first half – diagnosis and second half – to determine optimal continuous positive airway pressure.
- For mild OSA – not accurate
- Follow up necessary to confirm the diagnosis and effectiveness of treatment.

3. Portable Home Monitoring of Sleep
4. Other tests:

- Nasopharynoscopy
- Neurophysiologic tests
- Throat microphone
- Respiratory tests
- Cardiological tests

- Apnoea/ hypopnoea index (AHI)
- Fiberoptic endoscopy

Dental diagnosis

Common clinical findings

- Excess fat deposition in the palate, tongue and pharynx leading to narrowing of airway
- Individuals with micro and retrognathic jaws
- Macroglossia
- Elongated soft palate
- Lateral cephalometric radiographs - to confirm retropositioned maxilla and mandible, shortened cranial base, inferiorly set hyoid bone, enlarged soft palate and tongue, narrowed posterior airway spaces
- Image-intensification fluoroscopy in conjunction with tooth-guided mandibular protrusion with patient in supine position.

Diagnosing OSA typically involves a combination of patient history, physical examination, and polysomnography (a sleep study). Despite these diagnostic tools, OSA is often underdiagnosed, particularly in populations that do not fit the stereotypical profile of obese, middle-aged men. As a result, many individuals live with undiagnosed OSA, which can lead to long-term health complications.[11]

Health Implications

The health risks associated with untreated OSA are numerous. It has been linked to hypertension, cardiovascular disease (including heart failure and arrhythmias), diabetes, stroke, and even premature mortality. Furthermore, OSA impairs cognitive function, leading to daytime sleepiness, reduced attention span, and poor memory.[12] This can negatively affect both personal life and professional productivity. Mental health disorders, including depression and anxiety, are also more prevalent in individuals with OSA. Additionally, OSA has a significant impact on overall quality of life. Patients often experience difficulty concentrating, irritability, and a reduced ability to perform daily tasks due to fatigue.

Treatment of OSA

Treatment can be divided into General measures: applied to all patients, Specific therapy: tailored to each patient individually.[13]

The treatment for obstructive sleep apnea (OSA) aims to relieve symptoms, improve quality of sleep, and prevent complications. Here are common treatment approaches:

Lifestyle Changes

Weight loss: Losing weight can reduce fatty tissue around the neck, which can help ease airway obstruction.

Position therapy: Sleeping on your side rather than your back may reduce airway collapse.

Avoiding alcohol and sedatives: These substances relax the muscles of the throat, worsening sleep apnea.

Smoking cessation: Smoking contributes to inflammation and fluid retention in the upper airways.

Exercise: Regular exercise can improve overall muscle tone and sleep quality.

2. Positive Airway Pressure (PAP) Therapy

Continuous Positive Airway Pressure (CPAP): A CPAP machine delivers a constant stream of air through a mask, keeping the airways open while you sleep**. Fig 4**

Bilevel Positive Airway Pressure (BiPAP): Similar to CPAP but adjusts pressure when you inhale and exhale, which may be more comfortable for some patients.

Auto-CPAP: Adjusts the air pressure automatically based on your needs throughout the night.

Medical Treatments

Medications: While medications don't treat sleep apnea directly, certain drugs (e.g., modafinil,anti hypertensives, tricyclic antidepressants, serotonergic

agents etc) may be prescribed to treat daytime sleepiness.

Nasal Steroid Sprays: Help reduce nasal congestion, making it easier to breathe through the nose.

Surgical Treatments

Surgical options are considered if other treatments are ineffective or if there are anatomical issues contributing to sleep apnea.[14]

Uvulopalatopharyngoplasty (UPPP): A surgical procedure that removes excess tissue from the throat or uvula to open the airway.it can be done with lasers too.

Genioglossus advancement (GA): A surgery to reposition the muscles that control the tongue to help keep the airway open.

Tracheostomy: In severe cases, a tube is inserted into the windpipe to allow air to bypass the blocked upper airway.

Hyoid suspension surgery: Performed in conjunction with tracheostomy in event of severe OSA. This method enhances anterior superior repositioning of the tongue base, enlarges the airway in a lateral dimension, partially separates the tongue base from the lower airway by an infrahyoid myotomy.

Midline glossectomy: Reduces the bulk of tongue,Used in patients who do not respond to UPPP procedure and in those demonstrating narrow airway at the base of the tongue.

Mandibular advancement splint: Sagittal mandibular osteotomy is performed to affect anterior mobilisation and insertion of tongue at the genioid tubercle, thus enlarging the retrolingual space.

Maxillo Mandibular advancement: The degree of mandibular advancement without maxillary advancement will lead to prognathism and dental malocclusion. The maxilla and mandible are both advanced by sagittal osteotomy, which enlarges the retrolingual as well as retropalatal space.

Septoplasty and turbinate reduction: Most common surgery for sleep apnoea in patients with nasal obstruction, including deviated nasal septum and enlarged turbinates.

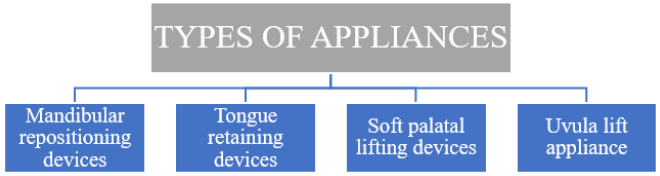
Oral appliance therapy

The goal these appliances is to enlarge the airway or reduce the collapsibility between soft palate and the posterior pharyngeal wall

Mechanism

- Narrow airway – mandibular advancement - increased volume of airway and decreased resistance to airflow
- The base of the tongue is moved farther from the airway tissues
- ↑ airway volume causes a slower rate of flow
- Raises the hyoid bone
- Greater stability of the airway musculature is achieved and resistance to airflow ↓

Oral appliances can be divided based on their mode of action. Fig 5



Soft palate lifters aim to reduce vibrations from the soft palate by elevating both the soft palate and uvula. However, there is little evidence regarding their effectiveness.

Uvula lift appliances

Tongue retaining devices (TRD) use a suction pressure to hold the tongue in a forward position during sleep and thereby prevent the tongue from falling back into the pharyngeal airway.

Mandibular advancement devices (MADs), mandibular advancement appliances (MAAs), mandibular repositioning appliances (MRAs), or mandibular advancement splints (MASs) [12]. The MAD is the most common type of oral appliance therapy used for the treatment of OSA [14]. The mechanism of action of the MAD is usually assumed to cause the enlargement of the cross-sectional upper airway dimensions by anterior displacement of the mandible and the attached tongue, resulting in improved upper airway patency .[15,16]

Several appliances move the mandible anteriorly, eg. Herbst, Klearway, Mandibular repositioner, PM positioner, Snore Guard, TheraSnore.

Criteria for evaluation of oral appliances

- Reliability at stopping snoring
- Titratability
- Simplicity of delivery
- Low bulk
- Lip seal
- Tongue space
- Non-interference with sleep
- TMJ or tongue symptoms easily adjusted
- Low cost
- Lateral freedom

Complications -During initial period of adjusting to the use of the oral appliance, patients usually complain of soreness of the jaws and teeth, either excessive salivation or dry mouth and minor occlusal changes, Long term mandibular protrusion may lead to complications with temporomandibular joint and dental occlusion.[16]

Challenges in Management

One of the key criticisms of OSA management is the focus on treating the symptoms (e.g., using CPAP) rather than addressing underlying risk factors such as obesity, which is a major contributor to the disorder. Weight loss and physical activity are critical but often neglected aspects of OSA treatment[17]. There is also a growing concern about the long-term cardiovascular implications of untreated OSA. Evidence suggests that untreated severe OSA significantly increases the risk of hypertension, coronary artery disease, stroke, and arrhythmias, yet patients with mild OSA often go undiagnosed or untreated due to a lack of awareness or clinical urgency.[18]

There is also limited public awareness about the disorder, and many people with OSA are unaware of its impact on their health or quality of life. Public health campaigns and more routine screenings in high-risk populations could help mitigate this.[19]

Emerging Research and Future Directions

Recent research has focused on the pathophysiology of OSA, including exploring genetic factors, the role of inflammation, and the effects of sleep fragmentation on cognitive function. Advances in neuroimaging and biomarkers may help identify individuals at risk for OSA earlier, potentially leading to earlier intervention.

Additionally, there is growing interest in personalized treatments, such as tailored CPAP therapy settings or genetic approaches to predict which patients may benefit most from surgical or non-surgical interventions. Newer, less invasive technologies, such as adaptive servo-ventilation (ASV) and bi-level positive airway pressure (BiPAP), are being tested and may offer advantages in managing complex OSA cases.[20]

Conclusion

Obstructive sleep apnoea is a multifaceted condition with significant health implications. While there are effective treatments available, challenges remain in terms of patient compliance, early diagnosis, and

management of underlying risk factors. Greater awareness, improved diagnostic methods, and personalized treatment strategies are essential to addressing the growing burden of OSA and its associated health risks. As research continues to evolve, there is hope that more effective and less invasive treatment options will become available, improving outcomes for individuals affected by the disorder. As dental professionals, we play a significant role in early diagnosis, management and care of patients suffering from sleep apnoea.

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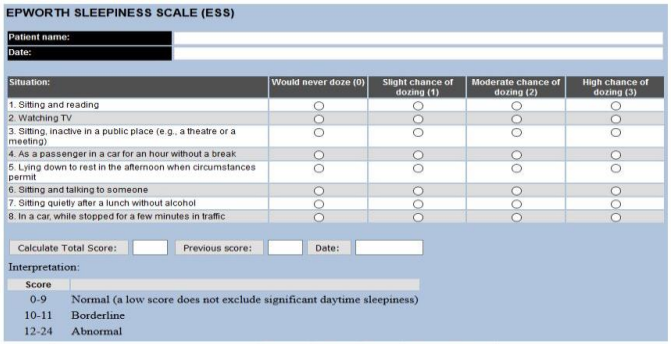
Legend Figure

Figure 1



EPWORTH SLEEPINESS SCALE (ESS)

Patient name:
Date:

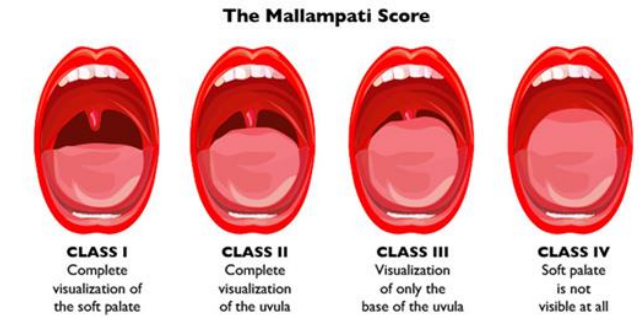
Situation:	Would never doze (0)	Slight chance of dozing (1)	Moderate chance of dozing (2)	High chance of dozing (3)
1. Sitting and reading	○	○	○	○
2. Watching TV	○	○	○	○
3. Sitting, inactive in a public place (e.g., a theatre or a meeting)	○	○	○	○
4. As a passenger in a car for an hour without a break	○	○	○	○
5. Lying down to rest in the afternoon when circumstances permit	○	○	○	○
6. Sitting and talking to someone	○	○	○	○
7. Sitting quietly after a lunch without alcohol	○	○	○	○
8. In a car, while stopped for a few minutes in traffic	○	○	○	○

Calculate Total Score: | Previous score: | Date:

Interpretation:

Score	
0-9	Normal (a low score does not exclude significant daytime sleepiness)
10-11	Borderline
12-24	Abnormal

Figure 2



Mallampati score – higher the grade, smaller the air passage, indicating sleep disordered breathing

Figure 3

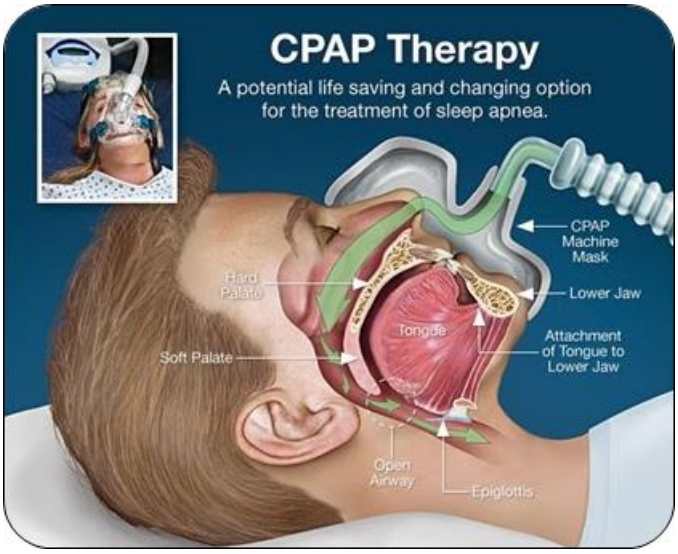


Figure 4

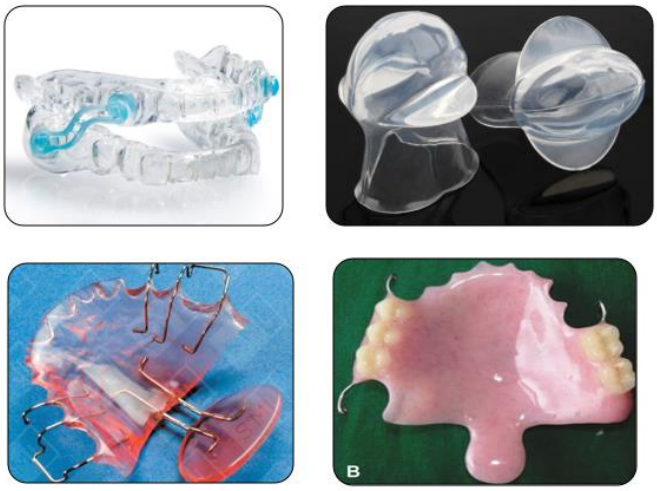


Figure 5