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Obstructive sleep apnea

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Abstract
Sleep disorders are characterized by problems with the quality, timing and amount of sleep of a person, which causes various problems with functioning and distress during the daytime. Obstructive sleep apnea (OSA) is a sleep-related breathing disorder that involves a decrease or complete halt in airflow despite an ongoing effort to breathe**.** Snoring is the most frequent symptom in obstructive breathing during sleep. Daytime hypersomnolence is the usual reason for adult patients to seek medical attention. Diagnostic criteria for OSA of at least 10 apnoeic events per hour. If sleep apnea is mild, these lifestyle changes alone may be recommended. CPAP is considered as a first-line option243 and is extremely useful in reducing symptoms, increasing quality of life and reducing the clinical consequences of sleep apnoea.

Keywords: Obstructive sleep apnea, sleep disorder, pharyngeal airway, continuous positive pressure, snoring

Introduction
According to American academy of sleep medicine, "Obstructive sleep apnea (OSA) is a sleep-related breathing disorder that involves a decrease or complete halt in airflow despite an ongoing effort to breathe [1]." It occurs due to collapse of the pharyngeal airway due to lack of motor tonicity of the tongue or the dilator muscles or both.
Gastaut first studied Sleep apnea in 1965. He observed a relationship between breathing abnormalities, snoring and daytime sleepiness in patients during sleep [2]. "Polysomnography" was developed to better understand and document sleep apnea and sleep. Definitions of sleep apnea and EEG arousals were first published by the American Academy of Sleep Medicine in 1999.

Sleep Related Breathing Disorder
Sleep is an essential part of our day-to-day routine. We humans spend almost one third of our lives asleep. With many studies in this regard we now can evaluate and manage most of the sleep complaints and their underlying disorders.
Normal sleep consists of 4 to 6 cycles which are defined both behaviourally and electroencephalographically (EEG), it includes periods during which the brain is active (characterized by rapid movements of eyes, called REM sleep). This is preceded by four progressively deeper and quieter sleep stages graded 1 to 4 on the basis of decreasing rate of EEG patterns [3]. Stages 3 and 4 are that of deep sleep periods.
Sleep disorders are characterized by problems with the quality, timing and amount of sleep of a person, which causes various problems with functioning and distress during the daytime. Poor or insufficient sleep has been associated with a wide variety of dysfunction in most body systems, including endocrine, metabolic, higher cortical function, and neurological disorders. Sleep disorders are very prevalent in the general population and are associated with significant medical, psychological, and social disturbances [4].

Prevalence
The prevalence of sleep related breathing disorders and OSAS was investigated more systematically in the late seventies and early eighties.

The field survey by Lavie *et al*. in 1983, was conducted on a presumably healthy working population required to be alert.
Peter *et al*. in 1985, reported an extremely high prevalence of sleep related breathing disturbance in selected blue-collar workers with mild coronary risk factors and reported disturbances in sleep-wake-cycle [5].
In 1986 the same investigators demonstrated again a high prevalence in subjects with cardiovascular complaints, but they also showed a much lower prevalence in presumably healthy subjects [6].
Gislason *et al*. were the first to systematically select subjects at risk for sleep related breathing, by using a complaint specific questionnaire informing about snoring and daytime sleepiness [6]. Cirignotta *et al*. in 1989 also used a questionnaire and a telephonic survey to select patients for recording, and found an intermediate prevalence rate [7].
Recent epidemiologic data suggest that more than 25% of "healthy" persons older than 65 years have more than 5 apneas per hour of sleep [8, 9]. This does not mean that these have sleep apnea syndrome, because daytime sleepiness may not be present at all.
An extremely high prevalence of OSAS in relativel "obese" ommercial truck drivers was shown by Stoohs *et al*. [10]. Seventy-nine percent of their subjects had more than five desaturations per hour, while 38% complained of daytime somnolence. These data are alarming, because the relation between sleep apnea and traffic accidents is well established.
Certain specific anatomical abnormalities are related with the occurrence of obstruction of the upper airway and may result in OSAS. Micrognathia (Pierre Robin syndrome), macroglossia (acromegaly, Downs syndrome) and hypertrophy of tonsillar and adenoid tissue (frequently encountered in children) are only a few of the documented causes.

Pathogenesis
During NREM I and II sleep respiration is controlled by metabolic and state dependent mechanisms, both with a different $pC0_2$ set-point. Due to the physiologic properties of NREM I in particular, but of NREM II as well, fluctuations between metabolic and state dependent control exist. This means that respiratory control fluctuates between two $C0_2$ set-points, resulting in periodic breathing that is considered an important condition for the development of sleep apnea. The state dependent decrease in cortical activity decreases the nonautomatic dilatation of the upper airway during sleep. The tonic-arousal dilatation of the upper airway is also decreased due to deactivation of the reticular system. The absence of nonautomatic and automatic dilatations leads to narrowing of the upper airway, which can become occluded if other factors are present. The upper airway collapses due to the negative intraluminal force resulting from the negative intrathoracic pressure during inspiration, resulting in obstructive apnea.
Subsequently hypoxia, hypercapnia and increased inspiratory muscular activity induce arousal and the upper airway is opened again. With arousal the CO2 set-point of respiratory control is shifted upwards as a result of increase in cortical activity. Ventilation is increased, until a change in state dependent control with falling asleep decreases the set-point again, and also decreases ventilation and dilatation of the upper airway again. The cycle repeats. These mechanisms contribute to periodic breathing, causing a vicious circle, further enhancing the instabilities in the respiratory control system [11].
A Magnetic Resonance Imaging based study demonstrated that the pharyngeal space of snorers and OSAS patients had a circular or elliptic orientation with the long axis in the sagittal plane. The pharyngeal area of normal controls was also elliptic but with the long axis orientated in the coronal plane. The soft palate is the most common site of narrowing at the level of the nasopharynx in approximately 80% of patients.

Symptoms and Natural History
Snoring is the most frequent symptom in obstructive breathing during sleep. Snoring is a very common phenomenon affecting about 20% of the 30 to 35 year-old population, while by age 60, 60% of men and 40% of women will snore habitually [12]. With very few exceptions, all OSAS patients have loud pharyngeal snoring associated with snorting and apneic periods. The periods of silence are a frequent cause for alarm by the bed partner who may repeatedly try to arouse the patient to make him breath again.
OSAS patients are however often unresponsive, even to painful stimuli, and may be disorientated when finally aroused.
Daytime hypersomnolence is the usual reason for adult patients to seek medical attention. Other complaints frequently encountered in OSAS are: difficulties maintaining sleep caused by apneas, abnormal movements during sleep and nocturnal enuresis; headache, cognitive deficits and sexual dysfunction [13].
In a general population-based study, obesity, respiratory symptoms (cough or wheeze), gender and age were found to be related to daytime sleepiness, suggesting that other factors may be complementary to sleep fragmentation and nocturnal hypoxemia in the development of daytime sleepiness in OSAS. The clinical course of OSAS is rather typical. In a sample of 50 patients studied by Kales, 40 patients snored prior to any other symptom, 36 patients had EDS at the onset of their illness [13]. Other symptoms came later, but in 50% obesity and hypertension preceded the complaints for at least one year [13].
Martikainen in 1994 followed a relatively random sample of 626 responders to a questionnaire send to 1600 people and found that snoring indeed increased with age, as did daytime sleepiness [14]. These data indicate again that a continuum exists from snoring to OSAS, and that the number and severity of symptoms increase with age and severity of the syndrome. Guillemin Ault was the first to state that an increase in upper airway resistance may lead from habitual snoring to upper airway resistance syndrome (UARS) and to OSAS respectively[15]. UARS is characterised by habitual snoring and all the other complaints of OSAS, but without the presence of obstructive apneas to define OSAS [15].
A relation between snoring and angina pectoris was found in men aged 40 – 69 years. The same investigators found that frequent to habitual snorers had a relative risk of 1.7 for ischemic heart disease and 2.08 for ischemic heart disease and stroke combined.

Diagnosis
Diagnostic criteria for OSA of at least 10 apnoeic events per hour. An "event" can be either an apnoea, characterised by complete cessation of airflow for at least 10 seconds, or a hypopnoea in which airflow decreases by 50 percent for 10 seconds or decreases by 30 percent if there is an associated decrease in the oxygen saturation or an arousal from sleep.
To grade the severity of sleep apnea the number of events per hour is reported as the apnoea-hypopnoea index (AHI). An AHI of less than 5 is considered normal. An AHI of 5-15 is

mild; 15-30 is moderate and more than 30 events per hour characterizes severe sleep apnea.

Subjective assessment of sleepiness

Patients with significant sleep apnoea may not realise that they have a problem as many of the features may be reported by a spouse or partner. Subjective assessment of sleepiness (by both patient and partner) is important as it is unlikely that patients will accept treatment unless they can perceive benefit with a reduction in subjective sleepiness or improvement in work performance.

The Epworth sleepiness scale (ESS)

It is a validated method of assessing the likelihood of falling asleep in a variety of situations. The maximum score is 24. The score can be used to clinically subdivide the patients into:

- the normal range (ESS <11),
- mild subjective daytime sleepiness (ESS=11–14),
- moderate subjective daytime sleepiness (ESS=15 –18)
- severe subjective daytime sleepiness (ESS>18)

Objective assessment of sleepiness

The Multiple Sleep Latency Test (MSLT) measures the time to fall asleep (using EEG criteria) in a darkened room on at least four separate occasions across the day following an instruction to fall asleep. Each challenge is terminated at 20 minutes or upon sleep onset. The average adult requires 10 or more minutes to fall asleep during the day and an average time of seven minutes or less is regarded as evidence of pathological sleepiness.

Physical examination

Examination by itself cannot allow an accurate diagnosis of OSA but it does help to exclude other causes for the patient's symptoms.

- Approximately 50% of patients with OSA are obese (BMI >30 kg/m2)[16]
- Often have neck circumference greater than 16" in a woman or greater than 17" (43 cm) in a man.
- Increasing neck size.
- Small chin, maxilla and mandible.
- Assessment of nasal patency visually
- A crowded posterior airway. These patients may have an enlarged floppy uvula or tonsillar hypertrophy. The posterior pharyngeal erythema may be secondary to repeated trauma from snoring or gastroesophageal reflux[16].
- Assessment of upper airway for obvious obstruction using indirect laryngoscopy if available
- Inspection of the tongue for macroglossia and assessment of dentition
- Assessment of pharyngeal appearance (tonsillar size, uvular appearance, lumen size)
- Measurement of BP
- Measurement of forced expiratory volume (FEV1) and forced vital capacity (FVC) to detect any significant spirometric abnormalities
- The possibility of hypothyroidism, acromegaly and Marfan's syndrome

Diagnostic Tools

- **Polysomnography (Laboratory testing)**

Nocturnal polysomnography is the gold standard for diagnosing OSA & it measures multiple physiologic parameters while the patient sleeps in a laboratory. A standard PSG typically consists of:

- Electro-oculogram: to detect rapid-eye-movement sleep,
- An electroencephalogram (to determine arousals from sleep),
- Chest wall monitors (to document respiratory movements),
- Oronasal flow monitors,
- An electrocardiogram,
- A segmental (+/-) tibialis electromyogram (to look for limb movements that cause arousals)
- Oximetry (to measure oxygen saturation).

- **Oximetry**

It alone is often used as the first screening tool due to the universal availability of cheap recording pulse oximeters. They are spectrophometric devices that detect and calculate the differential absorption of light by oxygenated and deoxygenated haemoglobin in blood to produce a measurement called the SpO_2. This is an assessment of the oxygen saturation of the arterial blood arriving at the fingertip or earlobe with each pulse beat.

- **Radiological imaging**

Studying upper airway size or shape by computed tomography (CT), magnetic resonance imaging (MRI), or cephalometric radiology does not accurately differentiate patients with OSA from normal subjects and cannot be recommended in the routine assessment of patients with possible OSA.

- **Questionnaires**

Comparison of questionnaire sampling in OSA patients to full PSG reported a mean sensitivity and specificity of only 42% and 68% respectively. Questionnaires are useful in the initial assessment of the potential OSA patient but cannot, by themselves, make the diagnosis[17].

Treatment

There are variety of treatments for obstructive sleep apnea, depending on an individual's medical history, the severity of the disorder and, most importantly, the specific cause of the obstruction.

In children

Epidemiological reports suggest prevalence of OSA upto 2% and snoring of 8% to 27% in children. The common cause of OSA in children is enlargement of lymphoid tissues in waldeyers ring as the adenoids reach their maximum relative size at 5 yrs of age. According to Guilleminault, the severity of respiratory obstruction in children may be reflected more often in respiratory labour than in the AHI scores /the degree of oxygen saturation in the blood[18]

In most children with OSA, tonsillectomy and adenoidectomy is curative. The surgery tends to cure not only the apnea and but reverses the cardiovascular complications & allows subsequent normal growth and development.

In adults

The treatment in the case of adults with poor oropharyngeal airways secondary to heavy upper body type is varied.

If sleep apnea is mild, these lifestyle changes alone may be recommended.

- Losing weight
- Exercise
- Stopping smoking

- Sticking to a regular sleep schedule
- Avoiding alcohol and sleeping pills
- Sleeping on side

Physical Intervention

Positive Airway Pressure: The most widely used whereby a breathing machine pumps a controlled stream of air through a mask worn over the nose, mouth, or both. The additional pressure splints or holds open the relaxed muscles, just as air in a balloon inflates it. There are several variants:

C.P.A.P. - Continuous Positive Airway Pressure

It works by delivering a steady stream of air through a special mask to keep the airway open during sleep. A machine gently blows pressurized room air to the mask through a flexible tube. The constant flow of air pressure from the mask prevents airway from collapsing when breathing in. The C.P.A.P. unit pressure is set specific to each person's needs.

VPAP - Variable Positive Airway Pressure

Also known as bilevel or BiPAP, uses an electronic circuit to monitor the patient's breathing, and provides two different pressures, a higher one during inhalation and a lower pressure during exhalation. This system is usually used by patients who have other coexisting respiratory problems.

APAP- or Automatic Positive Airway Pressure

An APAP machine incorporates pressure sensors and a computer which continuously monitors the patient's breathing performance. It adjusts pressure continuously, increasing it when the user is attempting to breathe but cannot, and decreasing it when the pressure is higher than necessary.

Oral appliances

There are three types of oral appliances:

1. Mandibular advancing devices which act to advance or downwardly rotate the mandible and thus draw the tongue forward through its attachments to the genial tubercles.
2. Tongue retaining devices to hold the tongue, through negative pressure in an anterior position during sleep.
3. Palatal lift devices aim to reduce the vibration of the soft palate and thus snoring

Radio frequency tissue ablation (RFTA) called Somnoplasty

Radio waves are used to shrink the tissues in the throat, soft palate or tongue by piercing it with an electrode connected to a radio frequency generator. This generates heat at approx 85° C (185° F) to create finely controlled coagulative lesions at precise locations within the upper airway.

Surgical procedures

Conventional (regular) surgery

This usually involves removing tissues that block the airway during sleep and may involve operating on the nose, soft palate and tongue.

UPPP (Uvulopalatopharyngoplasty)

This is the most common surgery for sleep apnea which is intended to enlarge the airway by removing or shortening the uvula and removing the tonsils and adenoids, if present, as well as part of the soft palate or roof of the mouth.

Tracheostomy

It is the most effective surgery for OSA & is generally reserved for serious apnea that has failed other treatment. The hole is plugged during the day for normal breathing and unplugged during sleep so obstructions are bypassed.

Pharmaceuticals

Oral administration of the methylxanthine, theophylline can reduce the number of episodes of apnea, but can also produce side effects such as palpitations and insomnia. Theophylline is generally given in Central Sleep Apnea, and infants and children with apnea [19].

Conclusion

The aetiology of OSA is multifactorial, consisting of a complex interplay between anatomic and neuromuscular factors, causing upper airway collapsibility. It has been pointed out that there are other physiological factors eventually defining the incidence of the disease.

Many treatment options are now used for treatment of OSA. CPAP is considered as a first-line option and is extremely useful in reducing symptoms, increasing quality of life and reducing the clinical consequences of sleep apnoea.

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