Practical approach to sleep disordered breathing

Anup Bansal, Arun Samuel Ponnish, Nagarajan Ramakrishnan*

Email: ram@nithra.com

Abstract

It is not uncommon to have occasional difficulty in falling asleep or have fatigue and day time sleepiness. However, when sleep disturbances persist for over a month and are associated with significant daytime dysfunction for at least two weeks, it may be a warning of an underlying sleep disorder requiring further evaluation and treatment. The combination of snoring, nonrefreshing sleep and daytime sleepiness is usually a hallmark of Obstructive Sleep Apnea (OSA) which is the most common sleep related breathing disorder. This review article aims to provide a practical and comprehensive approach to the diagnosis and management of sleep related breathing disorders with a focus on OSA.

Keywords: Obstructive, polysomnography, sleep apnoea.

Introduction

Sleep related breathing disorders are classified into three categories as per international classification of sleep disorders.[1] They are obstructive sleep apnoea (OSA) syndromes, central sleep apnoea (CSA) syndromes and sleep related hypoventilation/hypoxia syndromes. Sleep disordered breathing is an important public health problem with significant health consequences for affected individuals. It is characterised by repetitive airflow cessation (apnoea) or reduction in airflow (hypopnoea) during sleep. It occurs when forces promoting airway collapse overcome mechanism that maintains airway patency.

Apnoea is defined as cessation of airflow for ≥ 10 s in adults and 6 seconds or more in children. Over years, it has been observed by clinicians and researchers that *hypopnoea* has a similar impact on sleep. However, the definition has varied over a period of time. The consensus conference (Chicago Criteria) is now widely accepted and defines a respiratory event as hypopnoea if it meets one of the following criteria: Substantial reduction in airflow (> 50%), a moderate reduction in airflow (< 50%) with desaturation (> 3%), or a moderate reduction in airflow (< 50%) with EEG evidence of arousal.[2]

Classification

There are three types of sleep apnoea: Obstructive (OSA), Central (CSA) and complex or mixed sleep apnoea. Sleep apnoea is called as obstructive when respiratory effort is present and central when effort is absent.

Regardless of type, most of the time an individual with sleep apnoea may be unaware of his breathing problem. The condition is often noticed by bed partners or others who witness and vividly describe the episodes. It is not uncommon for the patient to deny any problem initially although they usually admit to symptoms upon further specific questioning. Common symptoms reported by patients are dryness of throat, a sense of tiredness or fatigue, drinking excessive caffeinated beverages to keep awake and remaining irritable or sleepy during the daytime. The symptoms also include difficulty in staying awake or falling asleep during common situations such as watching television, reading and driving etc, difficulty in concentration and controlling emotions, sometimes being aware of their own snoring and

N. Ramakrishnan, AB (Int Med), AB (Crit Care), MMM, FACP, FCCP, FCCM
Director, Nithra Institute of Sleep Sciences, Number 29 (Plot Number 1997), J Block, 13th Main Road, Anna nagar, Chennai 600 040

How to cite this article: Bansal A, Ponnish AS, Ramakrishnan N. Practical approach to sleep disordered breathing. Ind J Resp Care 2013; 2:277-83.

waking up gasping for breath (sometimes described as choking sensation) and waking up with morning headaches and dry mouth. Some patients may adjust themselves to the daytime sleepiness and fatigue associated with sleep disturbances over a period of time not realising the medical condition and the fact that treatment is available. Creating awareness on common symptoms would help to diagnose and treat earlier and prevent complications.

Obstructive sleep apnoea

In Indian population, OSA is the most common sleep related breathing disorder. The estimated prevalence of OSA and obstructive sleep apnoea syndrome (OSAS) with symptoms in males was 13.4% and 4% respectively whereas in females, these were 5.6 and 1.5% respectively in an urban Indian population. Some of the early studies suggested that males have a higher prevalence as compared to females.[3]

The common risk factors for OSA include obesity (BMI > 30 kg/m^2), neck circumference (16 inches in females; 17 inches in males), male gender, postmenopausal status, craniofacial abnormalities upper airway anatomy (macroglossia or enlarged tonsils), diabetes and hypothyroidism.[4,5] Nearly all measures of obesity (BMI, waist circumference and waist to hip ratio) predict the presence of OSA. Comorbid conditions such as cardiac or pulmonary disease may also predispose patients to OSA. Other risk factors include alcohol, smoking and sedative/hypnotic use which could cause or worsen OSA.

The pharynx is a collapsible tube that is acted upon by intraluminal and extraluminal pressure, there by producing a transmural pressure.[6] The extraluminal forces due to surrounding tissues collapse the upper airway while an intraluminal force is reduced in patients of narrowed upper airway. Airway obstruction occurs if the dilating forces are exceeded by the collapsing forces. Pharyngeal dilator muscles such as genioglossus surprisingly have increased activity in OSA patients but this activity will be diminished after sleep onset. This causes a decrease in airway luminal cross-section and an increase in upper airway resistance to airflow. *Ventilatory controller instability* may also contribute to propensity for partial or complete airway closure.

Loud snoring, excessive day time sleepiness, frequent nocturnal awakenings, morning headaches, mood swings, learning or memory impairment, nocturia and sexual dysfunction are common symptoms of OSA. In children hyperactivity and poor school performance are commonly noted. Snoring is the most common symptom in patients with OSA. Not all snorers have sleep related breathing disorders. However, it may not be possible to distinguish this clearly based on patient's history alone and a polysomnographic evaluation may be essential. Snorers who report that varying character and cessation of breathing have been witnessed by others, those who have experienced choking sensation, morning headache, daytime sleepiness or uncontrolled hypertension should undergo a sleep study to evaluate for OSA.

The consequences of untreated OSAS include hypertension (resistant in those who already have hypertension), diabetes (poor glycaemic control in diabetics on treatment), coronary artery disease, cardiac arrhythmias, cerebrovascular accidents, and impairment in quality of life, mood changes, cognitive dysfunction and sudden death.[7]

Central sleep apnoea

Central sleep apnoea (CSA) is defined as the cessation of airflow along with absence of respiratory effort during sleep. Its prevalence is less common than obstructive sleep apnoea. Some sleep studies report that predominantly central apnoea is found in only 10% of individuals with sleep breathing disorders. Broadly it can be classified into 5 types: Primary alveolar hypoventilation (sleep hypoventilation syndrome), sleep transitional apnoea, treatment emergent central apnoea, narcotic induced central apnoea and Cheyne Stokes respirations (CSR).

The major abnormality in these syndromes is stoppage of breathing when carbon dioxide (CO_2) levels fall below the apnoea threshold. Cheyne Stokes respiration (CSR) is commonly seen in patients of congestive heart failure.[8] CSR-CSA patients have enhanced chemosensitivity to CO_2 which destabilises breathing during sleep. During sleep, partial pressure of carbon dioxide in arterial blood ($PaCO_2$) rises by 3-6 mm Hg, which results

in hyperventilation and then $PaCO_2$ decreases below the apnoea threshold and breathing ceases. This results in periodic breathing with recurring cycles of apnoea and hyperventilation.[9]

Complex sleep apnoea

Complex sleep apnoea, also called mixed sleep apnoea is an emergence of central apnoea after disappearance of obstructive events with CPAP therapy in OSA patients. Most proposed mechanism is anatomic vulnerability for OSA and respiratory control instability. Treatment with adaptive support ventilation (ASV) may be considered.[10]

Diagnosis

The screening of patients suspected of sleep apnoea-hypopnoea syndromes is based on the performance of detailed clinical history and proper physical examination. During evaluation of symptoms, *Epworth Sleepiness Scale (Table 1)* is one of the widely used subjective methods to assess sleepiness. It is a short questionnaire that includes a list of 8 social circumstances in which patient may have a tendency to fall asleep. Patient has to rate chances of dozing on a scale of 0-3. The maximum score is 24 and a score greater than 10 suggests excessive daytime sleepiness. The validity of the questionnaire in Indian patients has been debated and modified versions are used in some sleep centres.[11]

Table 1: Epworth Sleepiness Scale[11]

		Chances of dozing (Rate on a scale of 0 -3)
	Sitting and reading	
	Watching TV	
	Sitting inactive in a public place	
	As a passenger in a car for an hour without a break	
	Sitting and talking to someone	
	Sitting quietly after a lunch without alcohol	
	Lying down to rest in the afternoon	
	In a car, while stopped for a minute in traffic	

The weight history, including a significant recent increase and change in waist or neck circumference should be recorded. A family history of snoring may suggest an anatomic or genetic link. An oropharyngeal and nasal examination helps in determining anatomic contributions to sleep disordered breathing. The cardiopulmonary examination should include blood pressure measurement, oxygen saturation and lung function test.

While initial evaluation with above symptoms and signs could suggest the possibility of OSA, the confirmatory diagnosis should be based on sleep study. There is a wide variation in the type of polysomnography (PSG) equipment and type of sleep study used across the globe. These studies are categorised ranging from Level 1 to Level 4 based on the number of parameters recorded and data analysed *(Table 2)*.[12]

Table 2: Levels of sleep studies

Level 1	• Attended in laboratory - Full PSG • Gold standard
Level 2	• Unattended Full or Comprehensive portable PSG
Level 3	• Cardiorespiratory sleep study • 4 bioparameters- Airflow, SpO_2, respiratory effort, heart rate along with body position sensor
Level 4	• Continuous 1 or 2 bioparameters • Airflow and SpO_2

Multiple parameters and data are available for review from a sleep study. However, a quick and easy approach often adapted to evaluate the severity of OSA would be check the apnoea-hypopnoea index (AHI). This is calculated on the basis of average number of apnoeas and hypoapnoeas per hour of sleep *(Table 3)*. Caution must be used not to strictly base the severity only on AHI as severity of oxygen desaturations, symptoms, length of apnoeas, comorbidities and presence of target organ damage should be taken into account while planning treatment.

Table 3: Classification of severity of OSA based on AHI

AHI	Classification
≤ 5	Normal
5 - 15	Mild obstructive sleep apnoea
16 - 30	Moderate obstructive sleep apnoea
> 30	Severe obstructive sleep apnoea

Polysomnography

Polysomnography is considered the gold standard for evaluation of sleep related breathing disorder and CPAP titration.[13] Frequently referred as sleep study, it consists of simultaneous and continuous recording of several physiologic variables to

describe sleep and associated events. Recorded variables include electroencephalogram (EEG), electrooculogram (EOG), electromyogram (EMG), airflow, snoring, thoracic and abdominal movement and pulse oximetry (SpO_2). Usually PSG is done in the laboratory but can be done at home. In most cases, this would be done at night, but exceptions may be made for shift workers and those with circadian rhythm disorders.

Electroencephalography (EEG): The placement of the EEG electrodes is based on the International 10-20 system. The important anatomic landmarks for study are *nasion* (the bridge of the nose), *inion* (the occipital protuberance) and the *preauricular point*. Each electrode is provided with a letter representing a region of brain namely, frontal (F), occipital (O), central (C) and mastoid (M) and a numeric subscript. Odd and even numbers are given for left and right sided electrodes respectively. The voltage recorded from these electrodes and the resultant waves are classified into delta (<4 Hz), theta (4-7 Hz), alpha (8-13 Hz) and beta (>13 Hz). Delta waves have the highest amplitude (>75 mV) and alpha waves are 50 mV in adults.

Electro-oculography (EOG): It records the difference in potentials between cornea and retina. Generally two types of eye movements are recorded, namely, slow rolling eye movements and rapid eye movements. Slow eye movements mainly occur during early relaxed stage with eye closed or during brief awakenings and stage N1 sleep while rapid eye movements are seen during waking with open eyes and in rapid eye movement (REM) stage of sleep.

Electromyography (EMG): It records mainly muscle tension and excessive leg movements during sleep to rule out periodic limb movement disorder. Sleep onset is associated with reduction in muscle tone and a further decrease occurs in REM sleep. Generally, electrodes are placed on the chin for evaluation of REM sleep. Additional EMG electrodes are often placed on the anterior tibialis also to evaluate leg movements.

Electrocardiogram (ECG): Single channel ECG is recorded usually from Lead II. An electrode is placed below the right clavicle and another over the left lateral chest wall.

Respiratory measurements (airflow, respiratory effort, oxygen saturation): It is essential to measure both respiratory effort and airflow. With the help of change in airflow we can distinguish between apnoea and hypopnoea while respiratory efforts are helpful in distinguishing between types of sleep related breathing disorder. Oronasal thermal sensor or pressure transducers (nasal cannula) are often used with each one having its advantages and disadvantages. Obstructive events appear as flattening of inspiratory airflow signal whereas central respiratory events are associated with reduced but rounded signals. The recommended sensors for respiratory efforts and oxygen saturation (SpO_2) are inductance plethysmography and pulse oximetry.

Scoring sleep stages

PSG data are divided into 30 second periods or epochs. Each epoch is assigned a single sleep stage that comprises maximum percentage of the epoch. Criteria for scoring various stages of sleep are outlined in *Table 4*.

Table 4: Scoring of Sleep Stages

Stage W	More than 50% of epoch has alpha waves when eyes are closed. If alpha waves are absent, then wakefulness is defined by presence of vertical eye blinks with high chin EMG tone.
Stage N1 (Normal: 5%)	>50% epoch occupy low voltage mix frequency waves Vertex sharp waves of < 0.5 s duration over the occipital region Slow eye movements can occur Reduced EMG tone as compared to stage W
Stage N2 (Normal 45-55 %)	Sleep spindle and K complexes over the central leads
Stage N3 (Normal 20-25%)	20-50% of epoch contain slow wave (delta) activity
	> 50 % of epoch occupies delta waves
Stage REM (Normal 20–25%)	Low amplitude, mixed frequency EEG waves Rapid eye movements Low chin EMG tone

Polysomnographic terminology

1. **Bed time:** It is the time when a person gets into bed and attempts to fall asleep.
2. **Lights out:** Time when sleep recording started.
3. **Light on:** Time when sleep recording ended.
4. **Sleep onset latency:** Time from light out to sleep onset. It is generally < 15-30 min in normal individuals.
5. **REM sleep latency:** Time from sleep onset to first epoch of REM sleep. Normally, it is 60-120 min in healthy individuals.
6. **Time in bed:** Duration of monitoring between light out to light on.
7. **Total sleep time:** Time obtained by adding all the sleep stages in minutes
8. **Sleep efficiency:** Defined as ratio of total sleep time to time in bed. Normal is approximately 85% or higher.

Procedure

For the standard level 1 PSG, patient has to come to sleep laboratory for overnight sleep study. Usually patient comes in the late evening and then recording will be started when he/she falls asleep. A sleep technician is available throughout the night to monitor patient during study. During the study, technician monitors sleep activity and data is displayed on the computer screen. Most often, the sleep study is completed by 6 to 7 am. While it is common practice in the USA to use only Level 1 PSG, it is common practice in India to use Level 3 portable sleep study particularly when index of suspicion and pretest probability of OSA are high. After study is completed, physician analyses and interprets the data in conjunction with clinical history. Once interpreted, the sleep physician writes a report with specific recommendations.

Split night polysomnography

In this type of study, initial portion of the study is used to document sleep related breathing disorder and the later portion of study is used for CPAP titration. Although it saves time and expenses for the patient, a significant proportion of patients may undergo suboptimal CPAP titration. A split night study may be indicated when AHI of ≥ 40 events per hour is recorded during the initial 2 h of study and at least 3 h remain for adequate CPAP titration.[14]

Treatment of OSA *(Table 5)*

It is estimated that only 30-40% of patients who are diagnosed with OSA pursue further recommended treatment (CPAP in particular). It is imperative to discuss the severity of diagnosis and the need for treatment with patient as untreated OSA leads to complications as discussed above. Conservative measures such as weight loss, positional therapy avoidance of alcohol and smoking are beneficial for all people with sleep disordered breathing.[15] Even mild reduction in weight (10%) can lead to improvement of symptoms.

Table 5: Treatment approach for OSA patients

Behaviour changes	Position (sleep on the side) Avoidance of alcohol, smoking and sedatives Weight loss and life style modifications
Oral appliances	Mandibular advancement splints Tongue stabilising devices
Positive airway pressure (PAP)	Continuous Positive Airway Pressure (CPAP) – Manual or Automatic Bilevel Positive Airway Pressure (BiPAP) – Usually required in patients with coexisting pulmonary problems and/or Obesity Hypoventilation Syndrome (OHS) Adaptive Support Ventilation (ASV) may be considered in complex sleep disordered breathing
Surgical options	Tracheostomy - Gold standard surgical procedure for those who are intolerant to CPAP and at immediate risk Maxillomandibular advancement (MMA) surgeries (for those with anatomical issues of mandible) Adenotonsillectomy (effective in children with OSA) Bariatric surgery for the morbidly obese patients Others: Laser assisted uvulopalato-pharyngoplasty, Radiofrequency volumetric tissue reduction
Pharmacological treatment	Topical nasal corticosteroids: Patients with OSA and concurrent rhinitis Modafinil: Treatment of residual excessive daytime sleepiness in OSA patients

Continuous positive airway pressure (CPAP) therapy is treatment of choice for moderate to severe OSAS. It is also recommended for symptomatic patients with mild OSA with or without cardiovascular complications. Positive pressure acts as a pneumatic

splint that maintains the patency of upper airway. There are various manufacturers and designs of CPAP and these are increasingly becoming more compact, user friendly and portable. It consists of an interface (choice of which should be customised and plays a key role in compliance) which is connected by a flexible tube to CPAP machine. CPAP treatment improves several features such as sleep architecture, daytime sleepiness, quality of life and cardiovascular disease. Control of blood pressure and glycaemic control improve in patients with hypertension and diabetes respectively. Some problems such as dry mouth, claustrophobia, nasal congestion and mask leak may lead to reduced compliance. Systematic education to allay apprehensions and heated humidification for patients with troublesome dryness are well known to improve compliance.[16,17]

Oral appliances are mainly indicated in mild to moderate OSA or patients who are unable to tolerate or fail CPAP**.** The oral appliance works by advancing the mandible forward (mandibular advancement devices) or by lifting and maintaining the tongue away from the posterior pharynx (tongue retention devices). Jaw discomfort, excessive salivation and toothache are the common side effects of oral appliances. It should be fitted by trained and experienced dentist.

Several **surgical approaches** such as nasal surgery, tonsillectomy, uvulopalatopharyngoplasty, tongue reduction surgery, bimaxillary advancement, hyoid and tongue suspension and tracheostomy have emerged to treat sleep apnoea. Overall success of these procedures is variable. Tracheostomy, mandibular advancement surgery and bariatric surgery in appropriate patients are clearly the most effective surgical approaches in OSA.

Summary

Suspected OSA patients with daytime sleepiness working in critical occupations or with comorbid cardiac and lung diseases should be investigated without delay. Level 1 PSG remains the gold standard for evaluation of sleep related breathing disorder. Level 3 and level 4 PSG can be done in patients with high probability for OSAS to confirm the diagnosis. Positive pressure therapy (CPAP) is the treatment of choice in moderate to severe OSAS. CPAP is indicated in uncomplicated OSAS patients in the absence of comorbid diseases and conditions. Successful weight loss is associated with improvement in AHI. Bariatric surgery is helpful in management of OSAS in morbidly obese patients (BMI > 40 kg/m^2), and in those with a BMI > 35 kg/m^2 with comorbid conditions. Optimisation of pharmacological therapy is the first step in the management of CSAS in patients with heart failure. If CSAS still persists, trial of CPAP may be considered. BIPAP should be considered for patients with ventilator failure.[18]

References

1. American academy of sleep medicine. International classification of sleep disorders, 2nd ed: Diagnostic and coding manual. Westchester, IL: American Academy of Sleep Medicine 2005.
2. American academy of sleep medicine. Sleep related breathing disorders in adults: recommendations for syndrome definitions and measurement techniques in clinical research: the report of an American Academy of Sleep Medicine Task Force. *Sleep* 1999; **22**:667-89.
3. Reddy EV, Kadhivaran T, Mishra HK, Sreenivas V, Handa KK, Sinha S, *et al.* Prevalence and risk factors of obstructive sleep apnea among middle-aged urban Indians: A community based study. *Sleep Med* 2009; **10**:913-8.
4. Jha A, Sharma SK, Tandon N, Lakshmy R, Kadhivaran T, Handa KK, *et al.* Thyroxin replacement therapy reverses sleep-disordered breathing in patients with primary hypothyroidism. *Sleep Med* 2006; **7**:55-61.
5. Sakakibara H, Tong M, Matsushita K, Hirata M, Konishi Y, Suetsugu S. Cephalometric abnormalities in nonobese and obese patients with obstructive sleep apnoea. *Eur Respir J* 1999; **13**:403-10.
6. White DP. Pathogenesis of obstructive and central sleep apnea. *Am J Respir Crit Care Med* 2005; **172**:1363-70.
7. Peppard PE, Young T, Palta M, Skatrud J. Prospective study of the association between sleep-disordered breathing and hypertension. *N Engl J Med* 2000; **342**:1378-84.

8. Sin D, Fitzgerald F, Parker J. Risk factors for central and obstructive sleep apnea in 450 men and women with congestive heart failure. *Am J Respir Crit Care Med* 1999; **160**:1101-6.
9. Eckert DJ, Jordan AS, Merchia P, *et al*. Central sleep apnea: pathophysiology and treatment. *Chest* 2007; **131**:595-607.
10. Allam JS ,Olson EJ, Gay PC *et al*. Efficacy of adaptive servo ventilation in treatment of complex and central sleep apnea syndromes. *Chest* 2007; **132**:1839-46.
11. Johns MW: A new method for measuring sleepiness: The Epworth sleepiness scale. *Sleep* 1991; **14**:540-5.
12. Ferber R, Milliman R, Coppola M, *et al:* Portable recording in assessment of obstructive sleep apnea: ASDA Standards of Practice. *Sleep* 1991; **14**:378-92.
13. Iber C, Ancoli-Israel S, Chesson A, *et al*. for the American Academy of Sleep Medicine. The AASM manual for the scoring of sleep and associated events: rules, terminology and technical specifications. Westchester, IL: American Academy of Sleep Medicine 2007.
14. Kushida C, Chediak A, Berry R, *et al*. Clinical guidelines for the manual titration of positive airway pressure in patients with obstructive sleep apnea. *J Clin Sleep Med* 2008; **4**:157-71.
15. Greenburg DL, Lettieri CJ, Eliasson AH. Effects of surgical weight loss on measures of obstructive sleep apnea: A meta-analysis. *Am J Med* 2009; **122**:535–42.
16. Patel SR, White DP, Malhotra A, Stanchina M, Ayas NT. The effect of CPAP therapy on subjective and objective sleepiness in obstructive sleep apnea: A meta-analysis of randomized controlled trials. *Arch Intern Med* 2003; **163**:565–71.
17. Series F, Plante J, Lacasse Y. Reliability of home CPAP titration with different automatic CPAP devices. *Respir Res* 2008; 956.
18. Canadian Thoracic Society 2011 guideline update: Diagnosis and treatment of sleep disordered breathing. *Can Respir J* 2011; **18**: 31–5.