Obstructed Breathing During Sleep and Obstructive Sleep Apnoea Syndrome – Assessment and Treatment

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INTRODUCTION

During normal sleep the tone of the pharyngeal airway dilator muscles is decreased resulting in upper airway narrowing and increased resistance to airflow[1]. Nasal obstruction may result from a variety of anatomical abnormalities such as septal deviation, nasal polyps, adenoid hypertrophy and rhinitis such as allergic rhinitis, acute viral rhinitis, vasomotor rhinitis and non-allergic rhinitis with nasal eosinophilia syndrome[2]. Disordered breathing during sleep can both result from and be worsened by nasal obstruction[3]. In children, nasal obstruction due to enlarged tonsils and adenoids results in a switch to oral breathing which may lead to the *adenoid faces* because of changes in the craniofacial structures during growth that predispose to disordered breathing during sleep.

Obstructed breathing during sleep

Obstructed breathing during sleep is a spectrum of abnormal breathing related to increased airway resistance during sleep that includes pure snoring, the upper airway resistance syndrome and the obstructive sleep apnoea syndrome (OSAS)[1,4,5].

Snoring

Pure snoring, the most benign extremity in this continuum of obstructed breathing during sleep, affects 19 to 37% of the general population and more than 50% of middle-aged men[6]. Snoring which does not produce clinical symptoms or cause disruption in sleep is termed *primary snoring*. Habitual snoring occurs in up to 25% of men and 20% of women in the normal adult population. A semi-blocked nose requires increased inspiratory effort which lowers intrapharyngeal pressures that tends to suck the pharyngeal walls together causing increased snoring[3]. Many individuals experience a worsening of their snoring when they suffer from an upper respiratory tract infection or rhinitis.

Upper airway resistance syndrome (UARS)

The UARS is characterized by sleep-related airflow limitation and increases in upper airway resistance that precipitates arousals resulting in fragmented sleep and excessive daytime sleepiness[5]. The individual with UARS does not have apnoeas, hypopnoeas or oxygen desaturations documented during overnight polysomnography. In this syndrome, reduced oral/nasal airflow is accompanied by an increased inspiratory effort that causes arousals as shown on the electroencephalogram (EEG) and immediate reduction in upper airway resistance after arousal. UARS events are typically short - lasting one to three breaths. Such events are termed *respiratory effort-related arousals* (RERAs). The prevalence of UARS in the adult general population, as defined by adults with snoring and excessive daytime sleepiness, is estimated to be as high as 10 to 15%.

Obstructive sleep apnoea syndrome (OSA)

OSA is characterised by repetitive episodes of partial or complete upper airway obstruction during sleep, resulting in sleep fragmentation and oxygen desaturation[7]. The fragmentation of sleep results in daytime symptoms of tiredness and drowsiness with diminished alertness and performance. The obstructive sleep apnoea-hypopnoea syndrome (OSAHS) is the occurrence of repetitive obstructed breathing events during sleep (any combination of apnoeas, hypopnoeas or RERAs) associated with daytime symptoms, particularly excessive somnolence. Excessive daytime sleepiness is recognised as an important cause of motor vehicle and industrial accidents, decreased productivity, interpersonal difficulties and cognitive dysfunction with memory and concentration problems.

There is also increasing epidemiological evidence linking OSAS to long-term cardiovascular morbidity, including hypertension, myocardial infarction and stroke[8-13]. Untreated severe OSA increases the risk of fatal and non-fatal cardiovascular events compared with healthy controls[14]. People with OSAS have an increased the risk of sudden death from cardiac causes during night time hours (midnight to 6.00 am)[15].

Patients with OSAS have narrower upper airways. Increased muscle tone during waking hours prevents upper airway obstruction while the subject is awake. Collapse occurs with the onset of sleep and relaxation of this support. As the apnoea continues, oxygen desaturation occurs, inducing hypoxemia, vasoconstriction of the pulmonary vascular bed and increase in pulmonary artery pressures. Attempted inspiration against the obstruction decreases intrathoracic pressure, which eventually triggers a micro-arousal and stimulates the sympathetic nervous system[7]. Heart rate and blood pressure both rises in response, and oxygen saturation improves with the resumption of breathing. Repetitive episodes of hypoxaemia-reperfusion, hypercarbia, sympathetic activation and intrathoracic pressure swings in OSA may trigger cellular and biochemical processes which predispose to atherosclerosis. Repetitive surges of sympathetic activity related to repeated episodes of arousals

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may directly promote endothelial/vascular injury and enhanced coagulability[16-18]. In addition to cardiovascular disorders, OSAS is also recognised as a risk factor for the metabolic syndrome[19]. OSAS is related to obesity and associated with insulin resistance and type 2 diabetes mellitus[19,20]. Furthermore, serum leptin and insulin levels have been found to be elevated in patients with OSAS independently of obesity.

Epidemiology

The estimated prevalences of OSAS in middle aged men and women are 9% and 4%, respectively[4]. The incidence of OSAS in the general population is between 2% and 4%. The demographics and possibly the pathogenesis of OSA may be different for Asians in comparison with whites[21-23].

Risk factors

The most significant risk factor for OSAS is obesity, especially upper body obesity[7]. Other risk factors for OSAS include male gender, age between 40 and 65 years, cigarette smoking, alcohol consumption and craniofacial abnormalities such as small posteriorly placed mandible, narrow oropharnyx, enlarged tongue and soft palate. Anatomical lesions that cause pharyngeal obstruction, such as tonsillar hypertrophy, are relatively uncommon in adult patients with OSAS, in contrast to children. Bony factors are more important in lean subjects while soft tissue abnormalities are important in obese individuals.

Risk factors for sleep-disordered breathing (SDB) in children
A number of common features of childhood predispose to SDB. Allergic rhinitis is common in children and so is adenoidal and tonsillar hypertrophy. Epidemiological studies have shown that children with habitual snoring frequently have allergic rhinitis. While adenoidal hypertrophy is a major factor for SDB in younger children (aged 1 to 4 years), adenoidal hypertrophy and bony changes combine to increase risk in older children (5 years or older).

Clinical features

Patients with OSAS typically present with a history of loud snoring and apnoeas witnessed by the bed partner[7]. Habitual snoring is present in 70% to 95% of these patients. Snoring is cyclical, with loud snoring noises alternating with periods of silence when the snorer struggles to reopen his/her airway. Finally, a loud snort - the so-called 'resuscitative snort' - occurs and the patient's airway is opened and breathing resumes. The main daytime manifestation is excessive sleepiness, but other symptoms such as unrefreshing sleep, poor concentration or memory and fatigue are commonly reported. The most common physical finding is a crowded oropharynx with an oedematous uvula and difficulty in visualising the posterior pharyngeal wall[7]. Varying degrees of obesity, thick neck, retrognathia, micrognathia and hypertension are other common findings.

DIAGNOSIS AND INVESTIGATIONS FOR PATIENTS WITH OSA

Overnight polysomnography (PSG)

The diagnosis of obstructed breathing during sleep is based on overnight PSG in a sleep laboratory with a technician in attendance during which a number of physiological parameters are monitored[24,25]. A minimum of 6 hours of monitoring during a diagnostic nocturnal PSG is required to capture variability related to sleep stage and position with respect to the frequency of obstructive respiratory events and the occurrence of other nocturnal events such as periodic limb movements. The polysomnogram is then analysed for sleep stages; respiratory events based on the oronasal airflow channel, respiratory effort channels and oximetry; heart rate variability and arrhythmias; and snoring and body position. Sleep staging based on data from the EEG, EOG and chin EMG channels is done according to the criteria of Rechtschaffen and Kales[26]. Arousals and awakenings from sleep are quantified. Arousals are scored according to the American Sleep Disorders Association (now known as the American Academy of Sleep Medicine) criteria[27].

American Sleep Disorders Association Criteria for Measurements, Definitions, and Severity Ratings of the Sleep-Related Breathing Disorders Task Force Report

Based on the American Sleep Disorders Association (now known as the American Academy of Sleep Medicine) Criteria for Measurements, Definitions, and Severity Ratings of the Sleep Related Breathing Disorders Task Force Report, an *apnoea* is defined as a cessation of flow at the nose and mouth for at least 10 seconds while a *hypopnoea* is a decrease for at least 10 seconds in the amplitude of airflow signal to at least 50% of the level prevailing before the event, or if a decrease to more than 50% should be associated with a fall in oxygen saturation of at least 3% or accompanied by an arousal[24,28]. Both apnoeas and hypopnoeas have similar pathophysiology and are generally considered to be similar with respect to their clinical impact. There are three types of apnoeas or hypopnoeas. While *obstructive* apnoea or hypopnoea is the complete cessation or reduction in airflow despite persistent inspiratory effort, *central* apnoea or hypopnoea is the absence or reduction in airflow due to an absence or reduction in respiratory effort. *Mixed* apnoea or hypopnoea starts as central apnoea or hypopnoea followed by obstructive apnoea or hypopnoea. In practice, mixed apnoeas are considered as obstructive and the majority of cases of sleep apnoea syndrome are obstructive. A RERA is an event characterised by increasing respiratory effort for at least 10 seconds leading to an arousal but which does not fulfill the criteria for an apnoea or hypopnoea. RERAs are detected with nocturnal oesophageal catheter pressure measurement which demonstrates a pattern of progressive negative oesophageal pressure terminated by a change in pressure to a less negative pressure level associated with an arousal. Acceptable and validated non-invasive methods that can replace oesophageal pressure in the majority of the situations include nasal pressure[29] and pulse transit time (PTT) measurements[30].

Scoring of events can be fully automated, or automated with a manual review that allows for editing of results or only manual. The apnoea-hypopnoea index (AHI) which is the sum of all apnoeic and/or hypopnoeic events divided by the total sleep time as measured in hours is used to assess OSAH severity. An AHI greater than 5 is considered abnormal. The respiratory disturbance index (RDI) is the number of obstructive apnoeas, hypopnoeas, and RERAs per hour averaged over the course of at least two hours of sleep[28]. Sleep

disruption can be assessed either by the arousal index (the number of arousals per hour of sleep), the number and frequency of sleep stage changes, the percentage of time spent in stage 1 (light sleep) or stage 0 (awake), the sleep efficiency (amount of time asleep divided by the duration of the recording), or the time spent awake after sleep onset. Diagnostic PSG should be interpreted by a physician trained in the evaluation and treatment of patients with OSAS. Decisions about treatment revolve around the degree of obstructed breathing during sleep and the amount of sleep disruption.

In-laboratory attended PSG is labour-intensive, time-consuming and expensive. Furthermore, timely access is a problem for many patients. With the advance of computers and microchip technology, portable and simple screening devices are now available to screen for OSA without requiring the patient to come into a sleep laboratory and be monitored by a technician.

Limited-channel PSG

Limited-channel diagnostic nocturnal PSG which should monitor the following parameters: oronasal airflow, chest wall effort, ECG, and oxygen saturation may be performed on patients with a high probability of OSA based on typical symptoms[31,32]. Limited-channel PSG is not able to determine sleep stage and does not detect non-OSA sleep disorders that may coexist with OSA. As sleep cannot be determined without EEG and EMG signals, breathing events are usually quantified per hour of monitoring time as an RDI. Symptomatic patients with an inconclusive or negative limited-channel PSG study should undergo full attended PSG to determine the cause of their symptoms.

Treatment of OSAHS

The treatment approaches to OSAHS are listed in Table I[7]. Treatment of underlying medical illnesses such as hypothyroidism can reduce the severity of OSAHS. Advice on weight reduction should be given to patients who are overweight or obese although it is difficult for these patients to achieve and sustain significant weight loss. For patients with mild to moderate OSA which is position-dependent, positional therapy, i.e. preventing them from sleeping in the supine position by sewing one or two tennis balls into the back of their nightshirts may alleviate the apnoea and snoring[33]. As alcohol and sedatives aggravate upper airway obstruction during sleep, these should be avoided. Improvement in nasal patency in patients with allergic rhinitis with nasal corticosteroids may ameliorate OSAS[34].

Continuous positive airway pressure (CPAP) treatment

Nasal CPAP is the most effective treatment for patients with OSAS[7]. Introduced in 1981, nasal CPAP acts as a pneumatic splint to prevent collapse of the pharyngeal airway[35]. With CPAP therapy, upper airway shape and/or dimension improve over time[36]. Incremental levels of CPAP result in progressive increase of upper airway size primarily in the lateral direction and thinning of the lateral pharyngeal walls. This enlargement is likely to be due to a combined effect of positive intrapharyngeal pressure and also an increase in end-expiratory lung volume due to CPAP that is associated with a reflex increase in the size of the pharyngeal lumen. CPAP application is associated with relaxation of the upper airway dilator muscles during both wakefulness and sleep. These dilator muscles must contract more forcefully in OSAS patients to counteract the high collapsing forces in the upper airway. Relaxation of these muscles with CPAP therapy probably limits the development of muscle fatigue. The finding that OSA is less severe in the latter part of the night if CPAP is used during the first part lends support for this possibility. The beneficial effects of CPAP are usually seen within a few days of starting therapy and consist of improved sleep quality, decreased daytime sleepiness, increased daytime alertness, improved cognitive function and improved quality of life[37,38]. Maximal improvement in neurocognitive symptoms can require as long as two months of treatment.

A review of randomised controlled trials showed CPAP therapy resulted in reduction in blood pressure in sleepy patients with severe OSAHS[39]. The magnitude of this benefit was largest in patients with OSAHS which is severe based on both sleep study and subjective sleepiness criteria. There was no benefit in subjects without daytime sleepiness. Becker *et al*[40] have shown that patients with severe OSAS in the active CPAP treatment arm achieved a reduction in mean systemic blood pressure of 9.9 mmHg over a period of nine weeks. This magnitude of reduction in mean blood pressure with nasal CPAP is predicted to reduce coronary heart disease event risk and stroke risk by 37% and 56%, respectively[40]. Other favourable effects of CPAP therapy include reduction of sympathetic activity and hypoxic/oxidative stress[41,42], with improvement of vasodilator response and endothelial function[43], and protection against death from cardiovascular disease in patients with OSAS[14,44].

Indication for CPAP treatment

CPAP treatment is indicated for all OSA patients with an RDI of 30 or more events per hour, regardless of symptoms, based on the increased risk of hypertension as shown by the Wisconsin Sleep Cohort data[45]. CPAP treatment is also indicated for patients with an RDI of 5 to 30 events per hour accompanied by symptoms of excessive daytime sleepiness, impaired cognition, mood disorders, insomnia, or documented cardiovascular diseases which include hypertension, ischaemic heart disease or stroke[46]. Treatment with CPAP is not indicated for asymptomatic patients without cardiovascular diseases who demonstrate mild OSA on diagnostic PSG.

Determination of effective CPAP

Traditionally, the effective CPAP is titrated in the sleep laboratory by means of polysomnography, and is defined as the lowest pressure level which abolishes most obstructive apneas, hypopneas, RERAs and snoring in all sleep positions and all sleep stages. However, this effective pressure can vary in a given individual from night to night and even during a given night, depending on body position, fatigue level, sleep stage, nasal patency, upper airway edema and ingestion of alcohol or sedative agents. Furthermore, regular maintenance CPAP therapy itself and weight loss may alter the effective pressure in the long term.

Auto-titrating CPAP

Auto-titrating or auto-adjusting CPAP devices are capable of continuously adjusting the pressure to the effective level by feedback control according to patterns of pressure, flow or other signals recorded during treatment[47]. Auto-CPAP can control OSAS as effectively as fixed pressure CPAP. The mean nightly pressure that patients sleep on is lower with auto-CPAP compared with fixed pressure CPAP[48]. Auto-CPAP devices also simplify the initiation and follow up of OSAS patients as attended pressure titration studies are not required in contrast with standard fixed CPAP. By reducing side-effects associated with air leaks and noise at higher pressures, auto-CPAP devices may increase compliance among patients who require CPAP higher than 10 cmH_2O[49]. A meta-analysis of nine published randomised trials from 1980 to 2003 comparing auto-CPAP to fixed CPAP[50] showed that auto-CPAP is associated with a reduction in mean pressure but similar to fixed CPAP in adherence, ability to eliminate respiratory events and to improve subjective sleepiness measured by Epworth Sleepiness Scale[51].

Bi-level positive airway pressure (Bi-level PAP)

Bi-level PAP allows for independent adjustment of inspiratory and expiratory pressures. Since they can treat obstructed respiratory events as well as hypoventilation, they are considered the treatment of choice for patients with obesity-hypoventilation syndrome[52]. They show little advantage in patients with pure OSA except for patients who cannot tolerate CPAP due to persistent massive nasal mask air leakage or discomfort exhaling against positive pressure.

Problems and side-effects associated with CPAP treatment

CPAP is rather obtrusive and the device is expensive and cumbersome. Patients have to put up with the inconvenience of using the device every night. Initial acceptance rates are generally in the region of 80%. Adherence to CPAP therapy ranges from 65 to 88%[53,54] and nightly use averages about five hours per night, although some studies show usage rates as low as three hours per night. Nasal blockage and/or dryness, irritation of the face by the mask, sore nasal bridge, sleep disruption by CPAP and reduced intimacy with the bed partner are the side-effects associated with the use of nasal CPAP. Nasal symptoms associated with the use of nasal CPAP can adversely impact on patients' tolerance, acceptance and adherence to therapy. The use of heated humidification is effective in alleviating these symptoms and improves patient comfort. Runny nose may require treatment with nasal steroids. A high percentage of mouth breathing documented during sleep at diagnostic PSG is a risk for low adherence to CPAP therapy[55]. Mouth leak with CPAP occurs in 10 to 15% of cases[56], and may compromise CPAP therapy[55]. The problem of mouth breathing can be overcome with the use of a chin strap or full face mask.

A high level of compliance with CPAP treatment may be accomplished with an education programme comprising educational brochures on OSA and CPAP, practical CPAP education and acclimatization sessions plus regular CPAP clinic review[57-59]. Adjustments or changes in the CPAP-patient interface may be indicated due to difficulties with mask fit leading to skin abrasion; massive air leak; or to breakage of mask, tubing or rebreathing valve. Radiofrequency tissue ablation of hypertrophied nasal turbinates in CPAP users has been shown to subjectively improve nasal obstruction and could lower the pressure requirements and increase self-reported CPAP adherence[60].

Oral appliances

Oral appliances and dental prostheses have also been used to treat obstructed breathing during sleep[61-63]. These devices which include tongue retaining devices and mandibular advancing devices, keep the upper and lower jaws opposed during sleep and advance the mandible forward. Anterior displacement of mandible and/or tongue enlarges the retroglossal space and thus reduces the degree of upper airway obstruction and pharyngeal collapse during sleep. According to the American Academy of Sleep Medicine[64], oral appliances are indicated for use in patients with primary snoring (in whom behavioural measures, such as weight loss or sleep-position change, are inappropriate or ineffective), patients with mild OSAS who do not respond to or are not appropriate candidates for behavioural measures, and those with moderate to severe OSAS who are intolerant of or refuse nasal CPAP and/or refuse or are not candidates for a surgical procedure.

Surgery

Adenotonsillectomy for enlarged adenoids and/tonsils results in marked improvement of SDB in more than 80% of children. Surgical procedures to correct nasopharyngeal anatomical obstruction include uvulopalatopharyngoplasty (UPPP), midline laser glossectomy, and nasal surgery. Correction of the nasal valve area, septoplasty and turbinate reduction may reduce nasal resistance. Nasal surgery has been shown to decrease CPAP requirements in a group of patients with OSAS; however, the degree of SDB is not significantly improved and snoring improves in only 34%. Variations of palatal surgery include YAG laser-assisted UPPP[65] and volume reduction of the soft palate (and tongue base) with radio-frequency ablation (or somnoplasty)[60,66]. Palatal procedures may benefit mild OSAS patients but are not likely to be effective for the majority of OSAS patients. Permanent soft palate implants (polyethylene terephthalate implants), an office-based procedure, was introduced in 2003 to treat snoring and mild to moderate OSAHS by reducing airway obstruction. Although non-randomised uncontrolled trials reported a moderate reduction in the number of breathing interruptions during sleep three to six months following the procedure and significant improvements in daytime sleepiness and snoring intensity,[67,68] there is currently insufficient published evidence to show that palatal implants are an effective treatment option for patients with mild to moderate OSAHS. Maxillo-mandibular advancement/ osteotomy, hyoid myotomy and suspension and tongue base reduction are the other modes of surgery which may be performed singly or in combination, in one stage or multiple stages[69]. In most patients with OSAS, upper airway obstruction involves multiple airway segments and upper airway surgery to one level alone is seldom successful in treating OSAS[70].

Table I: Treatment approaches for patients with the obstructive sleep apnoea syndrome

1.	**Treatment of underlying medical disorders**
2.	**Medical treatment**
	weight reduction
	avoidance of alcohol and sedatives
	smoking cessation
	nasal corticosteroids
3.	**Positional treatment**
	sleeping by the side
4.	**Nasal continuous positive airway pressure therapy**
5.	**Oral appliances**
	mandibular advancement / repositioning devices
6.	**Surgical treatment**
	correction of nasal septum deviation
	nasal polypectomy
	tonsillectomy/adenoidectomy
	uvulopalatopharyngoplasty
	YAG laser-assisted uvulopalatoplasty
	radiofrequency volumetric palatal tissue reduction (somnoplasty)
	soft palate implants
	mandibular advancement surgery

Drug treatment

Drug therapy has been proposed as an alternative to CPAP in some patients with mild to moderate sleep apnoea and could be of value in patients intolerant of CPAP. A Cochrane review of 26 randomised, placebo controlled trials (most of which were small studies) of 21 drugs reported positive effects of certain agents on short-term outcome[71]. In subjects with sleep apnoea and rhinitis, use of intranasal fluticasone was shown to reduce AHI and improve subjective daytime alertness. Physostigmine, mirtazipine, paroxetine, acetazolamide, naltrexone and nasal lubricant also reduced the AHI. Protriptyline led to symptomatic improvement but there was no change in the apnoea frequency. No significant beneficial effects were found for medroxyprogesterone, clonidine, mibefradil, cilazapril, buspirone, aminophylline, theophylline, doxapram, ondansetron or sabeluzole. Modafinil *(Provigil)* is a wake-promoting drug that is chemically dissimilar to and has a pharmacological profile that differs from central nervous system stimulants. It is an effective, well-tolerated and now standard treatment for excessive daytime sleepiness associated with narcolepsy[72]. Modafinil may be used for refractory or residual daytime sleepiness despite compliance with CPAP therapy and after exclusion of coexistent sleep disorders such as narcolepsy and medical conditions such as chronic pain as well as psychiatric conditions such as depression which may contribute to daytime sleepiness[73,74]. Modafinil does not treat the underlying disorder and therapy with modafinil alone without CPAP therapy leaves patients at risk for cardiovascular consequences.

CONCLUSIONS

OSAS is a common condition which is increasingly being linked to increased cardiovascular morbidity and mortality. In patients with mild to moderate OSAS, nasal CPAP has been shown to provide objective benefits, but compliance is relatively poor. Weight reduction improves OSAS, but is difficult to achieve. Sleep posture measures may benefit patients with predominantly supine OSA. Oral appliances are a reasonable alternative to CPAP in mild cases, and possibly in UARS. Surgery, such as UPPP, can be considered in selected patients with mild OSAS, but it is more suited to nonapnoeic snoring patients. The results of treatment with pharmacological agents have been generally disappointing. In patients with severe OSAS, nasal CPAP is the treatment of choice. Other treatment options should only be considered where CPAP fails or is not tolerated. Surgery to the upper airway such UPPP and radiofrequency ablation should not be considered in severe cases of OSAS. Compliance with nasal CPAP therapy may confer cardioprotective effects in addition to symptomatic relief and improved quality of life.

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Obstructed Breathing During Sleep and Obstructive Sleep Apnoea Syndrome – Assessment and Treatment

MCQ'S:

1. Simple snoring
   a. is a form of obstructed breathing during sleep.
   b. occurs in about a third of the general population.
   c. is more common in women.
   d. prevalence decreases with age.
   e. has been conclusively linked to increased cardiovascular morbidity and mortality.

2. Clinical features of obstructive sleep apnoea syndrome include
   a. habitual snoring.
   b. non-refreshing sleep.
   c. excesive daytime sleepiness.
   d. lower body obesity.
   e. crowded oropharnynx.

3. Obstructive sleep apnoea syndrome is associated with
   a. an increased risk for motor vehicle and industrial accidents.
   b. poorly controlled systemic hypertension.
   c. the metabolic syndrome.
   d. an increased risk for sudden death from cardiac causes.
   e. an increased risk for carotid artery atherosclerosis and stroke.

4. Nocturnal polysomnography
   a. is the monitoring and recording of various physiological parameters during sleep.
   b. is usually performed in a hospital-based sleep laboratory.
   c. is available in most Malaysian hospitals.
   d. detects sleep arousals by electroencephalographic changes.
   e. cannot differentiate non-apnoeic snorers from snorers with obstructive sleep apnoea.

5. The following statements regarding nasal continuous positive airway pressure therapy for the obstructive sleep apnoea syndrome are true.
   a. It is the most effective treatment for this condition.
   b. It improves sleep quality, daytime alertness, cognitive function and quality of life.
   c. Compliance is good even in patients without excessive daytime sleepiness.
   d. It reduces systemic blood pressure in all patients with obstructive sleep apnoea syndrome.
   e. It has a protective effect against death from cardiovascular disease.