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Adherence To CPAP In Obstructive Sleep Apnea Syndrome

Essay

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List of Abbreviations

ADHD	Attention Deficit Hyperactivity Disorder
AHI	Apnea hypopnea index
aOCT	Anatomic Optical Coherence Tomography
APAP	autotitrating positive airway pressure
BMI	Body mass index
BPAP	bilevel positive airway pressure
CBT	Cognitive behavior therapy
COPD	Chronic obstructive pulmonary disease
CPAP	Continuous positive airway pressure
CRs	Conditioned responses
CS	Conditioned stimulus
CT scan	Computerized tomography scan
EEG	Electroencephalography
EMG	Electromyography
EOG	Electrooculography
EPAP	Expiratory positive airway pressure
ESS	Epworth Sleepiness Scale
fMRI	Functional magnetic resonance imaging
FOSQ	Functional outcomes of sleep questionnaire
FSS	Fatigue Severity Scale
GERD	Gastroesophageal reflux disease
IPAP	inspiratory positive airway pressure
MET	Motivational enhancement therapy
MI	Motivational Interviewing
MRA	Mandibular repositioning appliances
MRI	Magnetic resonance image

NC	Neck circumference
OAs	Oral appliances
ODI	Oxygen Desaturation Index
OSAS	Obstructive sleep apnea syndrome
PAP	Positive Airway Pressure
PCP	Primary care physician
PLMS	Periodic limb movements of sleep
PSG	Polysomnography
PVT	Psychomotor vigilance task
RDI	Respiratory disturbance index
REM	Rapid eye movement
RERA	Respiratory effort related arousals
RFA	Radiofrequency ablation
RIP	Respiratory Inductance Plethysmography
RLS	Restless legs syndrome
SDB	Sleep disordered breathing
SLE	Systemic Lupus Erythmatosis
SS	Sleep specialist
TCRFA	Temperature controlled radiofrequency tissue ablation
TRD	Tongue retaining devices
UARS	Upper airway resistance syndrome
Uc	Calculated subjective time of CPAP use per night
UCRs	Unconditioned responses
UCS	Unconditioned stimulus
Ue	Estimated CPAP usage time per night
UPPP	Uvulo-palato-pharyngo-plasty
Ur	Reported mean usage time of CPAP per night

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Introduction

Obstructive sleep apnea hypopnea syndrome (OSAHS) is a chronic sleep disorder characterized by repeated episodes of upper airway obstruction during sleep (*McNicholas, et al, 2007*).

Sleep apnea can occur at different degree of severity, but the OSAS is defined by presence of at least five obstructive events per hour, with associated day time sleepiness, OSAS is frequent in general population, affecting more than 2% of adult females and 4% of males (*Tishler, et al,2003*). Moreover, prevalence rates increase with age, with OSAS occurring in 30%-80% of elderly population (*Tarasiuk, et al, 2008*).

The resulting sleep fragmentation can cause day time symptoms including sleepiness, headaches and cognitive dysfunction (*Young, et al, 2002*). Apart from the short term negative consequences caused by disturbed breathing, a growing body of evidence indicates that OSAS is also a risk factor for hypertension, cardiac failure, stroke and occupational accidents due to sleepiness (*Shamsuzzaman, et al, 2003*).

The treatment of choice in OSAS is continuous positive airway pressure (CPAP). By maintaining upper airway patency, CPAP is almost always effective in controlling the apneaic events and has been shown to improve the symptoms of OSA(*Gilles, et al, 2006*). Mortality rates with CPAP are low(3%) as compared to untreated patients (20%) (*Marti, et al, 2002*).

Adherence to CPAP treatment is important since sufficient use can eliminate apneas completely and improve sleep quality, excessive day

time sleepiness, and quality of life. Furthermore, it can reduce morbidity and mortality in cardiovascular diseases as well as consumption of health care resources (*Haniffa, et al, 2004*).

The adherence rates to CPAP, however, are low without a clear consensus of causes (*Weaver et al, 2006*).

The *World Health Organization* defines adherence as the "extent to which a person's behavior-taking medications, following a diet, and/or executing life style changes corresponds with agreed recommendations from a health care provider". The term has come to replace the term compliance as while compliance suggests a passive role, adherence emphasizes an active role (*Sabate, et al, 2003*).

Lutefy and Wishner,1999 suggest that the difference in terminology between compliance and adherence, though minor, indicates a shift from the medical model, whereby patients are seen to comply with their physicians' instructions to the social model, whereby patient adherence is dependent upon multiple economic and social constraints.

Adherence to CPAP is considered as regular use of the CPAP machine; however, the precise frequency of use to attain therapeutic effect is unknown. Recommended use is between 6-8 h per night, but researchers have defined CPAP adherence as anywhere from an average of 4 h a night for 70% of nights (*Pruitt, et al, 2009*).

It is not surprising that reports of CPAP adherence range from as low as 28% to more than 83% (*Weaver, et al, 2008*).

CPAP non adherence is a significant barrier to OSA treatment. Adherence failure is defined as "use of CPAP for less than 4 h/night on 70% of nights and/or lack of symptomatic improvement " as these criteria support significant improvement in the reduction of symptoms. Adherence failure ranges from 5% to 89% in the first week to 6 months (*Alicia, et al, 2010*).

CPAP adherence can be monitored using built-in smart cards, communication by modem, or a web based system that can differentiate the hours that CPAP was running from when it was used (*Pruitt, et al, 2009*).

Aim of the work

The aims of this Essay are to study the multiple factors that influence adherence to CPAP use in treatment of OSAS and different protocols used for better adherence, from which, extraction of suggested protocol will be done for follow up of CPAP adherence of patients attending Mansoura sleep disordered breathing clinics.

Obstructive Sleep Apnea Syndrome

Definitions

Obstructive sleep apnea (OSA) is a common chronic disorder that often requires lifelong care (*American Academy of Sleep Medicine, 2005*). Cardinal features of OSA in adults include Obstructive apneas, hypopneas, or respiratory effort related arousals (RERAS), daytime symptoms attributable to disrupted sleep and signs of disturbed sleep (*Epstein et al., 2009*).

Obstructive sleep apnea syndrome includes obstructive sleep apnea in adults and OSA in children. OSA in adults is defined as either more than 15 apneas, hypopneas, or RERAs per hour of sleep (ie, an apnea hypopnea index (AHI) or respiratory disturbance index (RDI) >15 events/hr) in an asymptomatic patient, or more than 5 apneas, hypopneas, or RERAs per hour of sleep (ie, an AHI or RDI >5 events per hour) in a patient with symptoms (eg, sleepiness, fatigue and inattention) or signs of disturbed sleep (eg, snoring, restless sleep, and respiratory pauses). More than 75 percent of the apneas or hypopneas may have an obstructive pattern (*Epstein et al., 2009*).

Apnea is the cessation, or near cessation, of airflow. It exists when airflow is less than 10 percent of baseline for at least 10 seconds in adults. At least 90 percent of the duration showed diminished airflow less than 10 percent of baseline. Apnea can produce arousals from sleep, increased arterial carbon dioxide, and decreased oxygen levels (*Iber et al., 2007*).

Hypopnea is a reduction of airflow to a degree that is insufficient to meet the criteria for an apnea. A precise definition has varied over

time, according to technology, expert opinion, and guidelines. *American Academy of Sleep Medicine*, 2012 recommends that hypopnea can be scored when all of the following four criteria are met which includes that the peak signal excursions drop by ≥ 30 percent of pre-event baseline using nasal pressure (diagnostic study), PAP device flow (titration study) or an alternative hypopnea sensor (diagnostic study), the duration of the ≥ 30 percent drop in signal excursion is ≥ 10 seconds and there is a ≥ 3 percent oxygen desaturation from pre-event baseline or the event is associated with an arousal (*Warren et al., 2012*).

Alternative scoring criteria are also approved. Airflow decreases at least 50 percent from baseline, there is diminished airflow lasting at least 10 seconds, at least 90 percent of the duration of diminished airflow is spent with airflow that is at least 50 percent less than baseline and decreased airflow is accompanied by at least 3 percent oxyhemoglobin desaturation or an arousal (*Iber et al., 2007*).

Respiratory effort related arousals (RERAs) exist when there is a sequence of breaths that lasts at least 10 seconds, is characterized by increasing respiratory effort or flattening of the nasal pressure waveform, and leads to an arousal from sleep, but does not meet the criteria of an apnea or hypopnea (*Iber et al., 2007*). The inspiratory airflow or tidal volume is maintained during these episodes, but requires increased respiratory effort. RERAs are often accompanied by a terminal snort or an abrupt change in respiratory measures (*American Academy of Sleep Medicine, 2005*).

RERAs (>5 events per hour) that are associated with daytime sleepiness were previously called *upper airway resistance syndrome*

(*UARS*), a subtype of obstructive sleep apnea (OSA). These patients may exhibit abnormal sleep and cardio-respiratory changes typical of OSA. Patients previously diagnosed with UARS are now considered to have OSA (*American Academy of Sleep Medicine, 2005*).

According to *Goetting and Downey, 2010* the *apnea hypopnea index (AHI)* is the number of apneas and hypopneas per hour of sleep while the *respiratory disturbance index (RDI)* is the total number of events (eg, apneas, hypopneas, and RERAs) per hour of sleep. The RDI is generally larger than the AHI, because the RDI considers the frequency of RERA, while the AHI does not.

Epidemiology

It is estimated that 26 percent of adults are at high risk for OSA (*Punjabi et al., 2008*).

The prevalence of OSA in the general population is approximately 20 percent if defined as an AHI greater than five events per hour. In contrast, it is 2 to 9 percent if defined as an AHI greater than five events per hour accompanied by at least one symptom that is known to respond to treatment (eg, daytime sleepiness) (*Epstein et al., 2009*). This difference suggests that it is common to be asymptomatic and have an AHI greater than five events per hour. Three to four percent of women and six to nine percent of men have OSA, when defined as an AHI greater than five events per hour accompanied by daytime sleepiness or a cardiovascular morbidity (eg, hypertension) (*Jennum et al., 2009*).

The prevalence of OSA increases from 18 to 45 years of age, with a plateau occurring at 55 to 65 years of age. There is a two- to three-fold

higher prevalence among individuals who are 65 years and older, compared to those who are 30 to 64 years old (*Jennum et al., 2009*).

OSA is more prevalent in African, Americans who are younger than 35 years old, compared to Caucasians of the same age group. This observation is independent of body weight. The prevalence of OSA in Asia is similar to that in the United States, despite having a lower mean body weight. African American individuals appear to be more predisposed to *sleep disordered breathing (SDB)* than white persons. This increased predisposition varies according to age. The odds ratio is greater than three in children younger than 13 years and is 1.88 in persons younger than 25 years. In elderly African Americans, the risk is increased two-fold. Examination of craniofacial morphology found that brachycephaly is associated with an increased AHI in whites but not in African Americans (*Cakirer et al., 2001*).

Chinese patients with OSA have a more crowded upper airway and relative retrognathia compared with their white counterparts, with statistical controls for BMI and neck circumference (*Patil et al, 2007*).

Asians are known to have a shorter cranial base and a more acute cranial base flexure, increasing OSA risk, with BMI and neck circumference being roughly equal. Therefore, interestingly, obesity plays a more prominent role in OSA predisposition in whites than in Chinese persons. This may serve to underscore the role that craniofacial factors have in Chinese patients. Other populations that may be at increased risk include Mexican Americans and Pacific Islanders. (*Wellman et al., 2004*).

Some of the *gender differences* may be age-related. Males have a higher AHI during adulthood, although there is little gender difference among adolescents or after the sixth decade (*Flemons, 2002*).

Pathophysiology

Brainstem nuclei coordinate the ventilatory actions of upper airway muscles, chest wall muscles, and the diaphragm. Phasic (inspiratory, expiratory) neural output induces cyclic increases and decreases of ventilatory muscle activation; the result is a series of breaths that comprise the ventilatory rhythm. Whether this rhythm is regular, irregular, or periodic over time is determined by the ventilatory control system (*Patil et al., 2007*).

Upper airway patency is maintained by the bony and cartilaginous structures surrounding the naso-pharynx and oro-pharynx, plus twelve pairs of skeletal muscles. Patients with OSA have a reduced upper airway size due to excess surrounding soft tissue or a highly compliant airway. A reduced airway size, combined with diminished neural output to the upper airway muscles during sleep and at apnea onset, can result in partial or complete upper airway collapse. The results are obstructive and mixed apneas. The tendency of the upper airway to collapse is determined by its critical closing pressure, which is designated as P_{crit} (*White, 2005*).

Apneas recur, at least in part, through a process referred to as "loop gain". This can be conceptualized as apneas occur when the respiratory drive is less than the threshold for inspiratory muscle activation and for maintaining upper airway patency during sleep. As apnea progresses, respiratory drive increases until a threshold is passed. Inspiration then occurs. If an overshoot in ventilation drives down carbon

dioxide levels, the respiratory drive may fall below the threshold for inspiration to occur. Thus, the next apnea results from overcompensation for the prior apnea (*White, 2005*).

Individual variations in P_{crit} and loop gain may explain why there exists a spectrum of OSA severity, as well as the appearance of central apneas during positive airway pressure titration (complex sleep apnea) (*Wellman et al., 2004*).

Pathogenesis of interplay between central apnea and OSAS

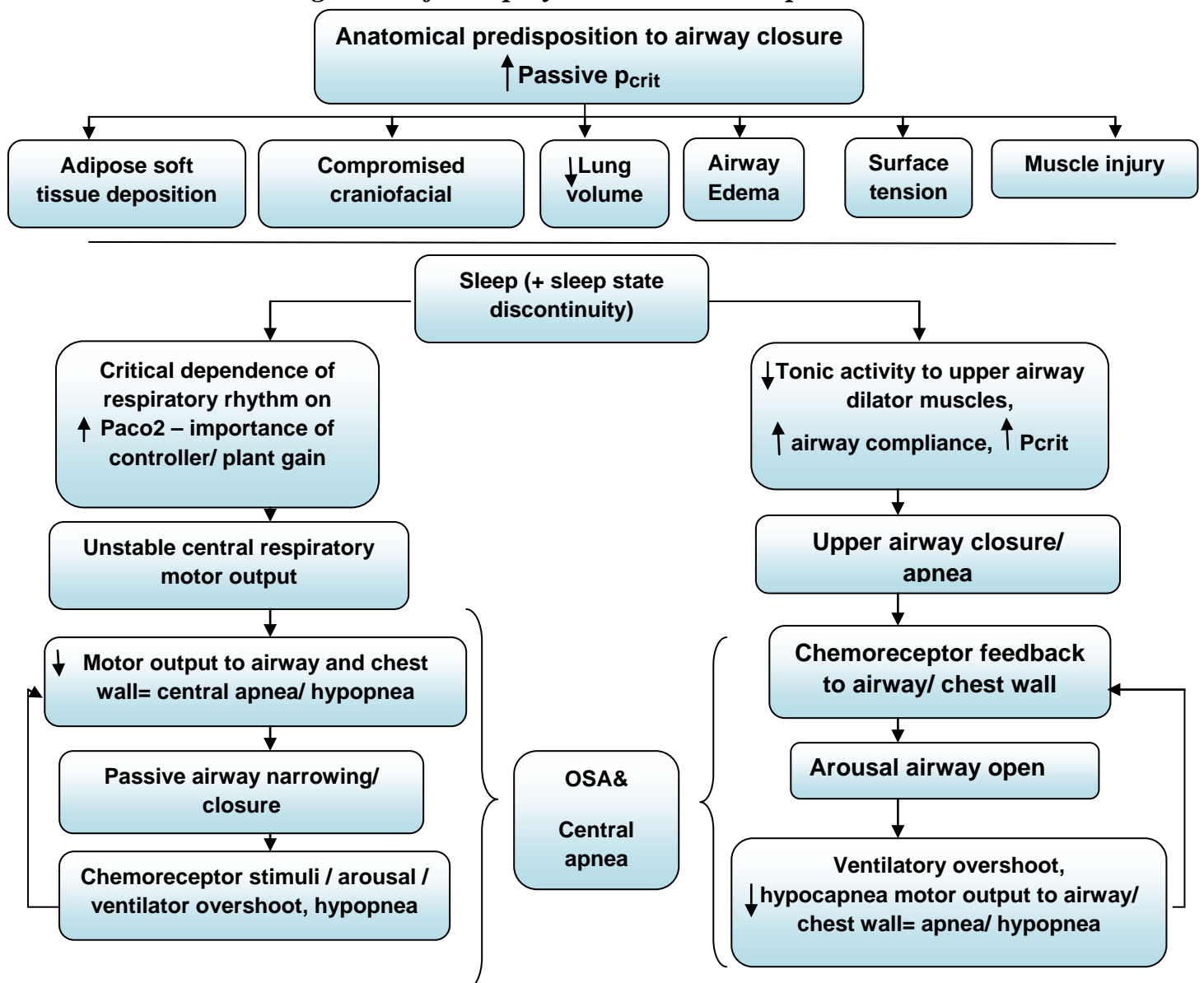


Figure 1. pathogenesis of cyclical obstructive sleep apnea (*Dempsey et al., 2010*).

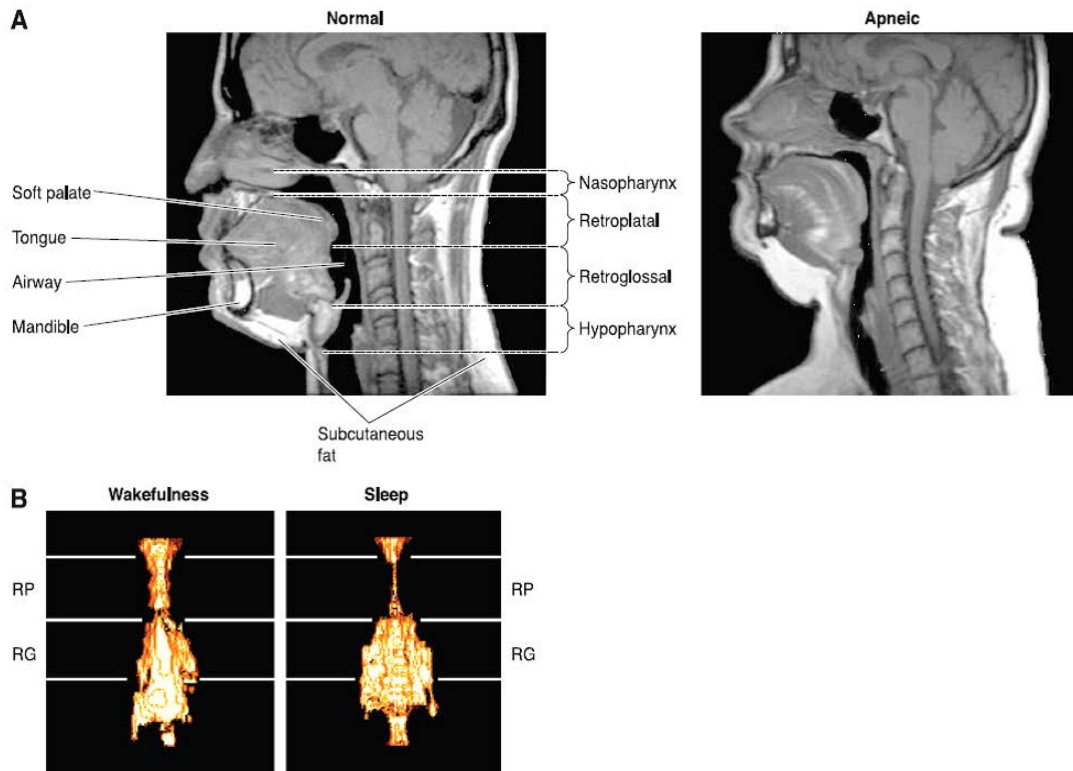


Figure 2. *A:* midsagittal magnetic resonance image (MRI) in a normal subject (*left*) and in a patient with severe OSA (*right*). In the apneic patient: the upper airway is smaller, in both the retroplatal and retroglossal region; the soft palate is longer and tongue size is larger; and the quantity of subcutaneous fat is greater. *B:* state dependence of upper airway size in a normal subject as assessed via three-dimensional reconstructions of MRI images. Images represent averages taken over several respiratory cycles during eupneic breathing in sleep and wakefulness. Airway volume during NREM sleep is smaller in the retroplatal (RP) region, not in the retroglossal (RG) region. (*Dempsey et al., 2010*).

Such images show the marked effect of sleep, per se, on the loss of upper airway muscle dilator tone and also show that the upper airway does not narrow as a homogeneous tube during sleep (*Dempsey et al., 2010*).

Risk factors

- **Definite risk factors**

Definite risk factors for OSA include obesity, craniofacial abnormalities, and upper airway soft tissue abnormalities (*Young et al., 2004*).

Obesity is the best documented risk factor for OSA. The prevalence of OSA progressively increases as the body mass index and associated markers (eg, neck circumference, waist-to-hip ratio) increase. *Craniofacial and upper airway soft tissue abnormalities* each increase the likelihood of having or developing OSA. Examples of such abnormalities include an abnormal maxillary or short mandibular size, a wide craniofacial base, tonsillar hypertrophy, and adenoid hypertrophy (*Young et al., 2004*).

The inheritance of obesity and craniofacial abnormalities explains only a fraction of the two- to four-fold increased likelihood of OSA or an elevated AHI among the family members of patients with OSA (*Young et al., 2004*).

- **Potential risk factors**

Potential risk factors include heredity, smoking, nasal congestion, acromegally, hypothyroidism and diabetes. Current smokers (but not past smokers) are nearly three times more likely to have OSA than never smokers. Nasal congestion confers an approximately two-fold increase in the prevalence of OSA compared to controls, regardless of the cause of nasal congestion. This is probably related to increased resistance due to decreased nasal patency (*Marshall et al., 2008*).

It is important to recognize that while these factors may be associated with OSA, their elimination is not necessarily curative of OSA. As examples, weight loss or correction of a craniofacial abnormality may resolve OSA, but smoking cessation and management of nasal congestion or septal deviation have not been shown to have a high yield as primary therapy (*White, 2005*).

Diagnosis of OSAS

- **Clinical presentation**

Snoring and daytime sleepiness are the most common presenting complaints of OSA. Additional symptoms and signs include restless sleep, periods of silence terminated by loud snoring, poor concentration, nocturnal angina, and awakening with a sensation of choking, gasping, or smothering (*American Academy of Sleep Medicine., 2005*).

Most patients with OSA first come to the attention of a clinician due to a complaint about snoring. This may be the presenting complaint, a symptom associated with another complaint, or a symptom detected during health maintenance screening. Regardless of how it is identified, all patients who snore should be asked about excessive daytime sleepiness (*Sansa et al., 2010*).

Daytime sleepiness is a common feature of OSA. However, it may go unnoticed or its significance may be underestimated because of its insidious onset and chronicity. The patient may not describe the symptom as sleepiness, but may use other terms, such as fatigue. Careful questioning of the patient typically reveals a pattern of feeling sleepy or falling asleep in boring, passive, or monotonous situations. As an example, the patient may admit to falling asleep while reading, watching

television, or operating a motor vehicle. In addition, embarrassing or inappropriate episodes of sleep may be reported (*Chervin, 2000*).

It is often unclear whether a patient's complaint of daytime sleepiness represents true sleepiness or fatigue. Sleepiness is the inability to remain fully awake or alert during the wakefulness portion of the sleep-wake cycle. Fatigue is a subjective lack of physical or mental energy that is perceived by the individual or caregiver to interfere with usual and desired activities (*Hossain et al., 2005*).

The Berlin Questionnaire is a simple sleep apnoea screening questionnaire used to quickly identify the risk (low to high) of sleep disordered breathing. The questionnaire consists of 3 categories and risk is based on the responses to individual items and overall scores in the symptom categories (*Punjabi et al., 2003*).

A simple questionnaire, the Epworth Sleepiness Scale (ESS), subjectively measures sleepiness as it occurs in ordinary life situations. It can be used as a screening test for excessive sleepiness or to follow an individual's subjective response to an intervention. It is usually helpful to have the patient's bed partner or a family member present during the interview because they may have greater insight than the patient. The patient is asked about his liability to doze off or fall asleep in some situations. Using a scale, he is asked to choose the most appropriate number for each situation (0 = would never doze, 1 = slight chance of dozing, 2 = moderate chance of dozing, 3 = high chance of dozing). The scores for the eight questions are added together to obtain a single number. A number in the 0–9 range is considered to be normal while a number in the 10–24 range indicates that expert medical advice should be sought. For instance, scores of 11-15 are shown to indicate the possibility

of mild to moderate sleep apnea, where a score of 16 and above indicates the possibility of severe sleep apnea or narcolepsy (*Punjabi et al., 2003*).

Table 1. Epworth Sleepiness Scale (*Punjabi et al., 2003*).

Situation	Chance of dozing (0–3)			
	0	1	2	3
Sitting and reading	0	1	2	3
Watching television	0	1	2	3
Sitting inactive in a public place (e.g. a theater or meeting)	0	1	2	3
As a passenger in a car for an hour without a break	0	1	2	3
Lying down to rest in the afternoon when circumstances permit	0	1	2	3
Sitting and talking to someone	0	1	2	3
Sitting quietly after a lunch without alcohol	0	1	2	3
In a car, while stopped for a few minutes in the traffic	0	1	2	3

The Fatigue Severity Scale ((FSS) is a method of evaluating fatigue_in multiple sclerosis and other conditions including Chronic Fatigue Immune Dysfunction Syndrome and Systemic Lupus Erythmatosis (SLE). It is designed to differentiate fatigue from clinical depression, since both share some of the same symptoms. Essentially, the FSS consists of answering a short questionnaire that requires the subject to rate his or her own level of fatigue. A total score less than 36 suggests that he/she may not be suffering from fatigue, but if it is 36 or more, the patient is in need for further assessment by physician. The obvious problem with this measure is its subjectivity can be combined with the Epworth Sleepiness Scale to objectively determine whether a patient is sleepy, fatigued, or both. The presence or absence of excessive daytime sleepiness is a key variable in deciding which patients should undergoing diagnostic testing for OSA (*Hossain et al., 2005*).

Table 2. Fatigue severity Scale (*Hossain et al., 2005*).

During the past week, I have found that:	Score						
1. My motivation is lower when I am fatigued.	1	2	3	4	5	6	7
2. Exercise brings on my fatigue.	1	2	3	4	5	6	7
3. I am easily fatigued.	1	2	3	4	5	6	7
4. Fatigue interferes with my physical functioning.	1	2	3	4	5	6	7
5. Fatigue causes frequent problems for me.	1	2	3	4	5	6	7
6. My fatigue prevents sustained physical functioning.	1	2	3	4		6	7
7. Fatigue interferes with carrying out certain duties and responsibilities.	1	2	3	4	5	6	7
8. Fatigue is among my three most disabling symptoms.	1	2	3	4	5	6	7
9. Fatigue interferes with my work, family, or social life.	1	2	3	4	5	6	7

Daytime sleepiness, fatigue, or inattention can result from micro-arousals (ie, electroencephalographic activation lasting three seconds or less), despite the absence of apneas or hypopneas. Snoring may or may not be a prominent complaint. These symptoms are reduced by treatment that alleviates RERAs (*Downey et al., 1993*).

Other associated symptoms and historical features include awakening with a sensation of choking, gasping, or smothering, awakening with a dry mouth or sore throat Restless, interrupted sleep, episodes of cessation of breathing, periods of silence terminated by loud snoring, moodiness, lack of concentration, morning headaches, decreased libido and impotence, awakening with angina pectoris, nocturia and history of hypertension, cardiovascular disease, cerebrovascular disease, renal disease, type 2 diabetes mellitus, or gastroesophageal reflux disease (*Margel et al., 2006*).

Table 3. Clinical features of obstructive sleep apnea-hypopnea (*Sansa et al., 2010*).

<i>Symptoms</i>	<i>Signs</i>
Daytime sleepiness	Obesity
Non restorative sleep	Large neck circumference
Witnessed apneas by bed partner	Systemic hypertension
Awakening with choking	Hypercapnia
Nocturnal restlessness	Cardiovascular disease
Insomnia with frequent awakenings	Cerebrovascular disease
Lack of concentration	Cardiac dysrhythmias
Cognitive deficits	Narrow or "crowded" airway
Changes in mood	Pulmonary hypertension
Morning headaches	Corpulmonale
Vivid, strange, or threatening dreams	Polycythemia
Gastroesophageal reflux	

- **Physical examination**

The physical examination is frequently normal, except for obesity (body mass index >30 kg/m²) and a crowded oro-pharyngeal airway. The obesity may be only moderate, since 18 to 40 percent of patients are less than 20 percent above their ideal body weight (*Epstein et al., 2009*).

Additional physical findings that are suggestive of OSA include elevated blood pressure, narrow airway, large neck and/or waist circumference, signs of pulmonary hypertension or corpulmonale. Approximately 50 percent of patients with OSA have coexisting hypertension, which is often most elevated in the morning (*Bixler et al., 2001*).

Conditions associated with increased upper airway resistance may cause the airway to appear narrow or crowded. These include

retrognathia, micrognathia, lateral peritonsillar narrowing, macroglossia, tonsillar hypertrophy, an elongated or enlarged uvula, a high arched or narrow palate, and nasal abnormalities (eg, septal deviation or polyps) diagnosed by E.N.T. examination (*Epstein et al., 2009*).

OSA correlates with an increased neck size or waist circumference more than general obesity (*Carmelli et al., 2000*). It is particularly prominent among men who have a collar size greater than 17 inches (43.2 centimeter) and women who have a collar size greater than 16 inches (40.6 centimeter) (*Epstein et al., 2009*).

Signs of pulmonary hypertension or cor pulmonale (eg, peripheral edema) may exist if OSA coexists with obesity hypoventilation syndrome or an alternative cause of daytime hypoxemia (*Arias et al., 2006*).

- **Laboratory**

Routine laboratory data are not helpful in establishing or excluding the diagnosis of OSA. Nonspecific abnormalities that are occasionally found include elevated liver enzymes, proteinuria, hypercapnia, cardiac dysrhythmia, hypothyroidism, acromegaly. Less than 10 percent of patients with OSA have proteinuria, but it may be severe. Although hypercapnia is uncommon in OSA alone, awake hypercapnia and hypoxemia may be present if obesity hypoventilation syndrome coexists. OSA can be caused or exacerbated by hypothyroidism (*Flemons et al., 2004*).

- **Diagnostic evaluation**

Diagnostic testing is essential to confirm or exclude OSA, since the clinical features of OSA are nonspecific and the diagnostic accuracy of clinicians' subjective impression is poor (*Epstein et al., 2009*).

It should be performed on any patient who snores and has excessive daytime sleepiness. In the absence of excessive daytime sleepiness, diagnostic testing should be done if the patient snores and either has two or more of the clinical features of OSA or works in a mission-critical profession (eg, airline pilots, bus and truck drivers) (*American Academy of Sleep Medicine., 2005*).

According to *Morgenthaler et al., 2006*, this approach is illustrated in the following algorithm for diagnosis of obstructive sleep apnea-hypopnea (OSAH) (**figure 3**).

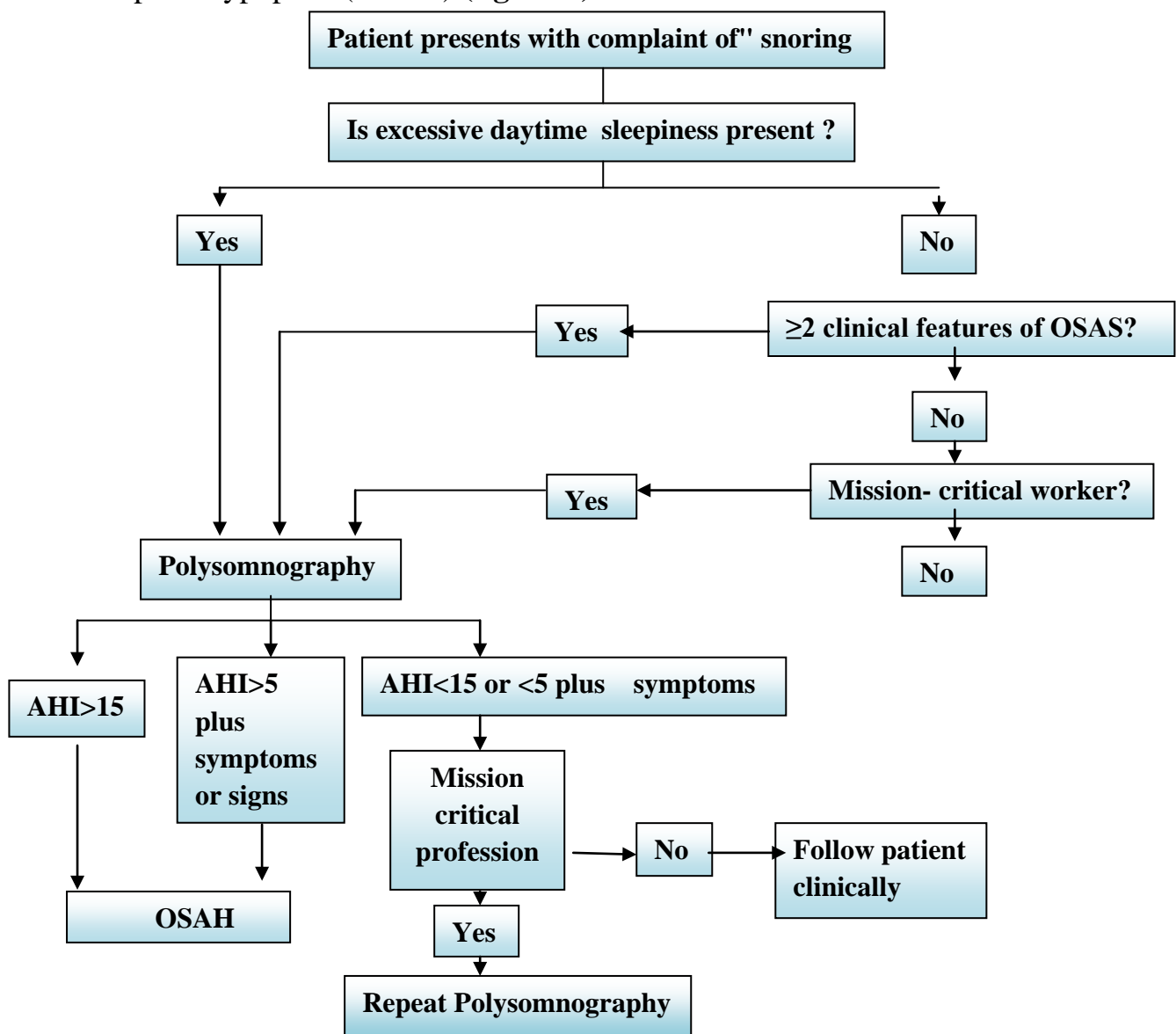


Figure 3. Approach for diagnosis of OSAS (*Morgenthaler et al., 2006*).

- ***Polysomnography***

For most patients, *full-night* (ie, diagnostic only) or *split-night* (ie, diagnostic and therapeutic), attended, in-laboratory polysomnography which is a diagnostic test for OSA. During it, the patient sleeps while connected to a variety of monitoring devices that record physiologic variables (*Skomro et al., 2010*).

Physiologic sensor leads are placed on the patient in order to record brain electrical activity, eye and jaw muscle movement, leg muscle movement, airflow, respiratory effort (chest and abdominal excursion), ECG, and oxygen saturation (*Rodsutti et al., 2004*).

Information is gathered from all leads and fed into a computer and outputted as a series of waveform tracings which enable the technician to visualize the various waveforms, assign a score for the test, and assist in the diagnostic process (*Sivan, 2005*).

Physiologic Variables measured and recorded. Assessment of sleep stages requires three studies including electroencephalography (EEG), electrooculography (EOG), and surface electromyography (EMG). For EEG six electrodes (labeled C₃, C₄, M₁, M₂, O₁, and O₂) and one ground electrode are placed around the cranium to record electrical activity across the brain. These leads are used to determine the stage of sleep the patient is in during any given period of the night (*Silber et al., 2007*).

Two EOG channels are used to monitor both horizontal and vertical eye movements. Electrodes are placed at the right and left outer canthi, one above and one below the horizontal eye axis. The electrodes pick up the inherent voltage within the eye; the cornea has a positive

charge and the retina has a negative charge. Evaluation of the eye movements is necessary for 2 reasons. First is for documentation of the onset of rapid eye movement (REM) sleep, and second is to note the presence of slow-rolling eye movements that usually accompany the onset of sleep (*Ayas et al., 2010*).

For EMG three leads are placed on the chin (one in the front and center and the other two underneath and on the jaw bone) and two are placed on the inside of each calf muscle 2-4cm apart. These leads serve to demonstrate muscle movement during sleep. This is helpful in documenting a wake period, an arousal, or just a spastic movement (*Campbell and Neill, 2011*).

Two ECG electrodes are placed on the chest right infraclavicular and left inframamary in seventh intercostals space. These record the heart rate and rhythm and serve to alert the technician to a possible emergency situation. They also demonstrate whether apneic desaturation leads to arrhythmias or not(*Silber et al., 2007*).

Airflow (thermistor or thermocouple sensor) a device that looks similar to a nasal cannula is secured just under the patient's nose. It senses the amount of air moving into and out of the airways and sends a signal to a physiological recorder. This tracing is used to determine the presence and extent of apneic episodes (*Skomro et al., 2010*).

Respiratory Inductance Plethysmography (RIP) is a method of evaluating pulmonary ventilation by measuring the movement of the chest and abdominal wall. A Respiratory Inductance Plethysmograph consists of two sinusoid wire coils insulated and placed within two 2.5 cm (about 1 inch) wide, lightweight elastic and adhesive bands. The

transducer bands are placed around the rib cage under the armpits and around the abdomen at the level of the umbilicus (belly button). They are connected to an oscillator and subsequent frequency demodulation electronics to obtain digital waveforms. During inspiration the cross-sectional area of the rib cage and abdomen increases altering the self-inductance of the coils and the frequency of their oscillation, with the increase in cross-sectional area proportional to lung volumes. The electronics convert this change in frequency to a digital respiration waveform where the amplitude of the waveform is proportional to the inspired breath volume (*Campbell and Neill, 2011*).

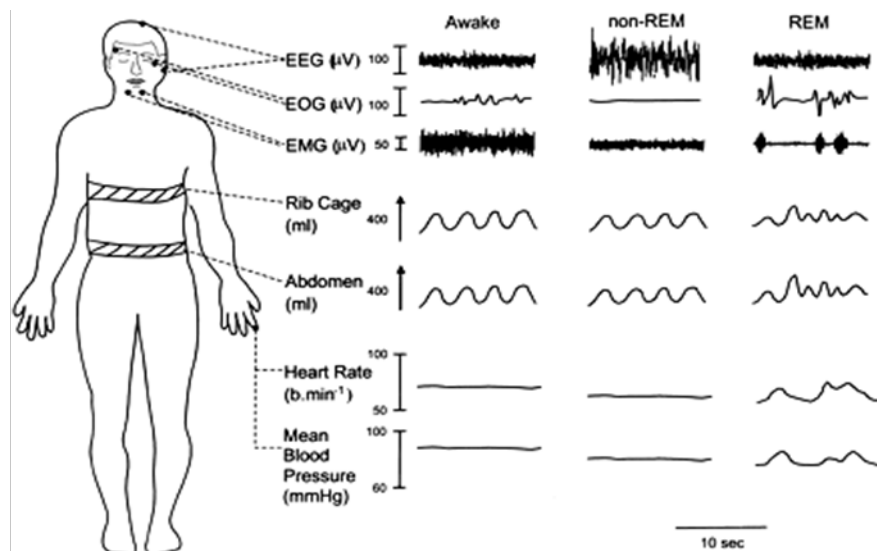


Figure 4. Typical electrode placements to distinguish sleep-wake states from the electroencephalogram (EEG), electrooculogram (EOG), and electromyogram (EMG) signals. Respiratory inductance plethysmography can be used to measure breathing noninvasively from bands placed around the rib cage and abdomen. Blood pressure and heart rate can also be continuously monitored, noninvasively, via a finger cuff and photoplethysmography. Note the typical low-voltage/high-frequency EEG signal in wakefulness and rapid eye-movement (REM) sleep and the high-voltage/low-frequency signal in non-REM sleep. Note also the progressive decline in postural EMG activity from wakefulness to non-REM sleep, with minimal EMG activity in REM, except for brief muscle twitches, associated with the rapid eye movements recorded by the EOG. REM sleep is also associated with rapid and irregular breathing and variable heart rate and blood pressure (*Stephen et al., 2001*).

The O₂ saturation is measured by a pulse oximeter probe placed on the patient finger, earlobe (*Parrino et al., 2004*).

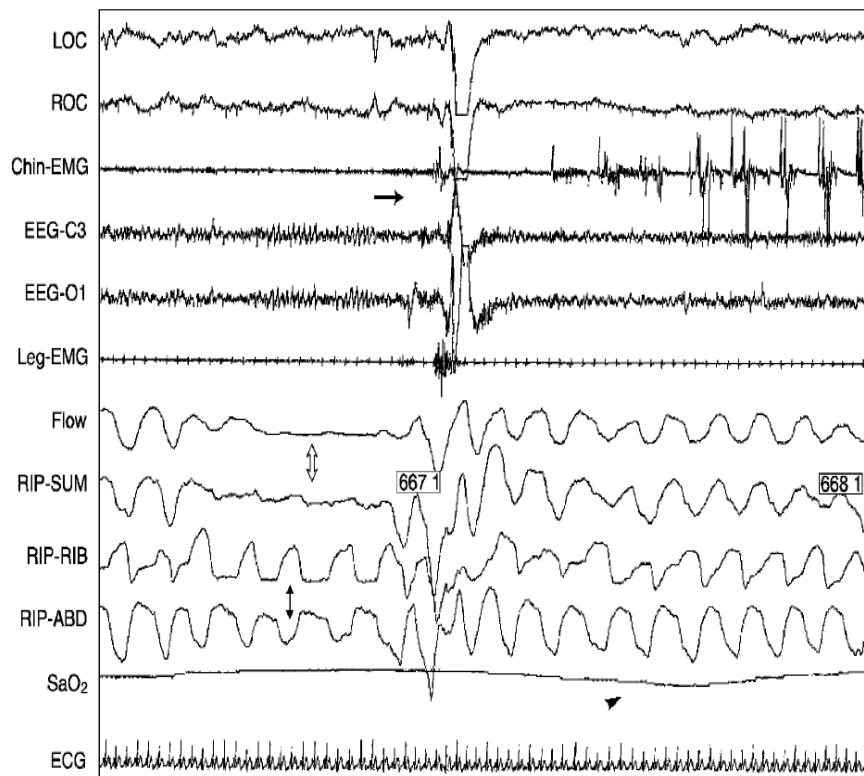


Figure 5. Sample record of the overnight traces in a patient with obstructive sleep apnea. LOC = left electro-oculogram; ROC = right electro-oculogram; Chin EMG = submental electromyogram; EEG = electroencephalogram; Leg EMG = anterior tibialis electromyogram; Flow = airflow; RIP-SUM = respiratory inductance plethysmography recording of the sum of the rib cage plus abdominal excursion (qualitatively reflecting ventilatory effort); RIP-RIB = respiratory inductance plethysmography recording of the rib cage excursion; RIP-ABD = respiratory inductance plethysmography recording of the sum of the abdominal excursion; SaO₂ = oxyhemoglobin saturation by pulse oximetry; ECG = electrocardiogram (usually a modified chest lead) (*Stephen et al., 2001*).

Full-night, attended, in-laboratory polysomnography (PSG) is considered the gold-standard diagnostic test for OSA. It involves monitoring the patient during a full night's sleep. Patients who are diagnosed with OSA and choose positive airway pressure therapy are subsequently brought back for another study, during which their positive airway pressure device is titrated (*Iber et al., 2007*).

Split-night, attended, in-laboratory polysomnography is similar, except the diagnostic portion of the study is performed during the first

part of the night only. Those patients who are diagnosed with OSA during the first part of the night and choose positive airway pressure therapy can have their positive airway pressure device titrated during the second part of the night. An absence of REM sleep and /or less than three hours of sleep recorded during a split night study can lead to significant underestimation of sleep apnea severity(*Flemons et al., 2003*).

Despite its reputation as the gold-standard, negative PSG should be viewed with skepticism if the clinical suspicion of OSA is high. In this situation, repeating PSG may be worthwhile. In an observational study, 11 patients with negative PSG but a high clinical suspicion of OSA underwent repeat PSG. Six of the patients had positive repeat PSG, suggesting that PSG can vary significantly from night-to-night(*Kushida et al., 2005*).

Unattended, in-home *portable monitoring* is a reasonable alternative for patients in whom there is a high likelihood of moderate or severe OSA, as well as no co morbidities (*Epstein et al., 2009*).

There are a variety of devices that are used for in-home, unattended, portable monitoring. Many have been validated against standard PSG, typically by testing the same patient with both modalities in the sleep laboratory. Generally speaking, the sensitivity and specificity seem to be high in populations at high risk of OSA on the basis of clinical symptoms, assuming there are no comorbid medical disorders or sleep disorders (*Collop et al., 2007*).

The clinical practice guidelines from the *American Academy of Sleep Medicine, 2007*, indicates that portable monitoring may be used as

an alternative to polysomnography for the diagnosis of OSA in patients with a high pre-test probability of moderate to severe OSA.

Portable monitoring should not be used in patients who have comorbid medical conditions that predispose them to non-OSA sleep related breathing disorders (eg, heart failure) or in whom another sleep disorder is suspected. The portable monitoring device must record airflow, respiratory effort, and blood oxygenation (*Collop et al., 2007*).

The increased recognition of OSA has led to more requests for inpatient consultations regarding sleep-related conditions, especially among perioperative or cardiac patients. The judicious use of portable monitors in this setting can identify inpatients who are at high risk for OSA, which may influence management. This situation is likely to be common according to one case series. The series of 105 patients who had been admitted for acute myocardial infarction found that the prevalence of previously undiagnosed OSA was high (65.7 percent) (*Lee et al., 2009*).

The diagnosis of OSA is based on the frequency of respiratory events during sleep (i.e. apneas, hypopneas, respiratory effort related arousals), as measured by polysomnography or portable monitoring, as well as the presence or absence of related symptoms (*American Academy of Sleep Medicine, 2007*).

- ***Radiology***

For years, in part due to the significance and frequency of obstructive sleep apnea syndrome (OSAS), and also in part due to frustrations of defining optimal treatments for selected patients, imaging techniques have been used to attempt to gain insights into the disorder. In

hopes of defining the exact site of obstruction in a selected patient, a therapy could be tailored to that person (*Esslinger, 2008*).

Some of the more standard imaging techniques that have been used include x-ray cephalometry which measures the cranio-facial dimensions in relationship to oro-pharyngeal diameters (the area behind the tongue) on a person's head and neck. This attempts to predict OSA in patients by selecting those with reduced diameters in the throat area. Unfortunately these measures are low in both sensitivity and specificity for the disorder (*Fleetham, 2010*).



Figure 6. Lateral cephalometry in patient with OSA – Note a steep mandibular plane, a narrow posterior airway space, an increased length and width of the soft palate, and an inferiorly positioned hyoid bone (*Hora et al., 2007*).

Cephalometry has also been combined with CT scanning of the posterior tongue and soft palate to predict this better, but again this has been limited in defining the population at risk (*Stuck and Maurer, 2008*).

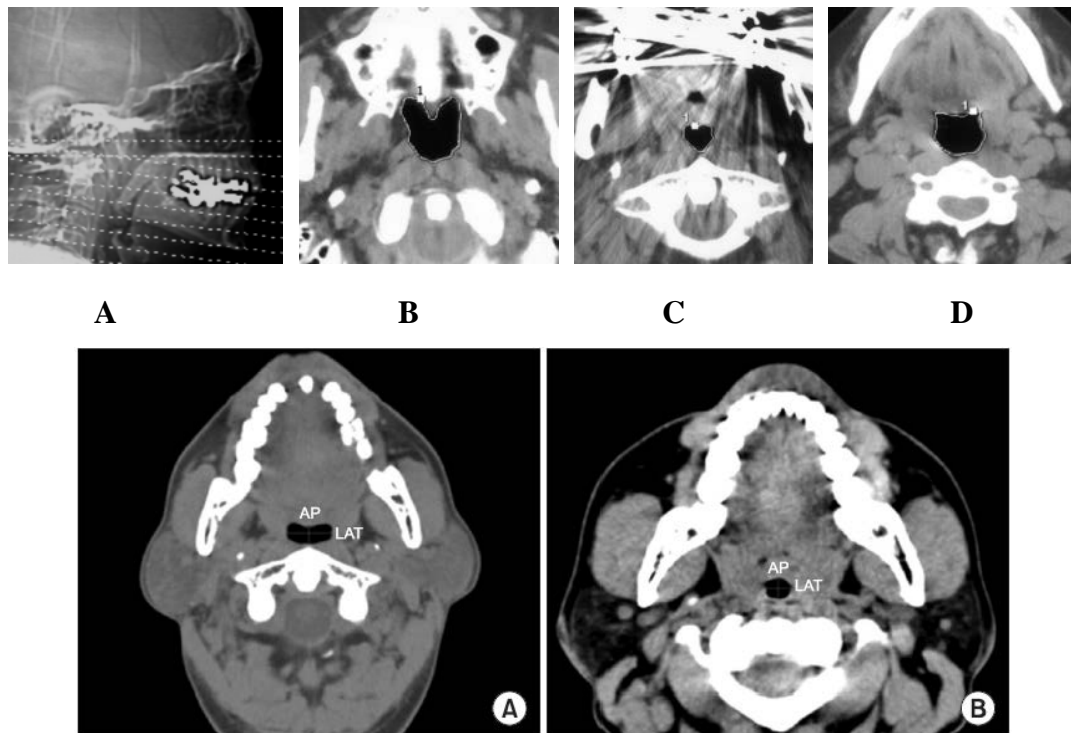


Figure 7. CT Tomography in patient with OSAS. Images show the parameters measured on CT scans. CT was performed to obtain measurements of the luminal area of the airway (outlined areas in **B**, **C** and **D**) at the levels of the nasopharynx, oropharynx, and hypopharynx. (**A**) Lateral scout view shows the levels (dashed lines) at which CT sections were obtained. CT scans obtained at the (**B**) hard palate level, (**C**) oropharyngeal level (20 mm caudal to **B**), and (**D**) hypopharyngeal level (50 mm caudal to **B**). The horizontal computed tomography views show the minimal anteroposterior diameter of the upper airway. A. The ratio of lateral to anteroposterior diameters is more than the 1.5 defined elliptical shape. B. The ratio of lateral to anteroposterior diameters is less than the 1.5 defined ovoid shape. (AP: anteroposterior diameter, LAT: lateral diameter) (*Stuck and Maurer, 2008*).

Nasopharyngoscopy

Another option is to have videoendoscopy with Muller's maneuver. This is where an otolaryngologist inserts a laryngoscope (flexible optical fiber) down one nostril into your nasal/throat area. The patient then plug the other nostril and mouth and try to breathe in. This creates a vacuum and can show how much his airway collapses. This has been more effective in predicting patients with OSA if the area behind the palate is less than 0.8cm squared in males and less than 0.54 cm squared in females (*Ghegan et al., 2006*).

Attempts including performing nasopharyngoscopy under sedation, and having the patient's jaw advanced during the procedure are used to assess if visualized obstruction and snoring improved. If so, these patients were either fitted with a dental appliance advancing the mandible, or underwent surgical mandibular advancement. This seems to have some beneficial results in small population studies ((*Giles et al., 2006*)).

MRI

Also MR techniques are being studied to assess the anatomical risks of patients who may have OSA, but pre- and post-operative exams by MRI have not been able to show consistent changes that allow predictability of success or failures of various treatments (*Fleetham, 2010*).

Optical Coherence Tomography

Anatomic optical coherence tomography is in real-time imaging technique which can be performed without sedation. A small probe is passed from the nose to mid-esophagus, and within this an optical probe is inserted. As it passes down it directly visualizes and measures the anatomy of the upper airway by creating images from the phase characteristics of light that bounces back from the surrounding tissues. This procedure still has limitations, but may hold the key for both better therapeutics and diagnostics since obstruction is being measured real-time (*Giles et al., 2006*).

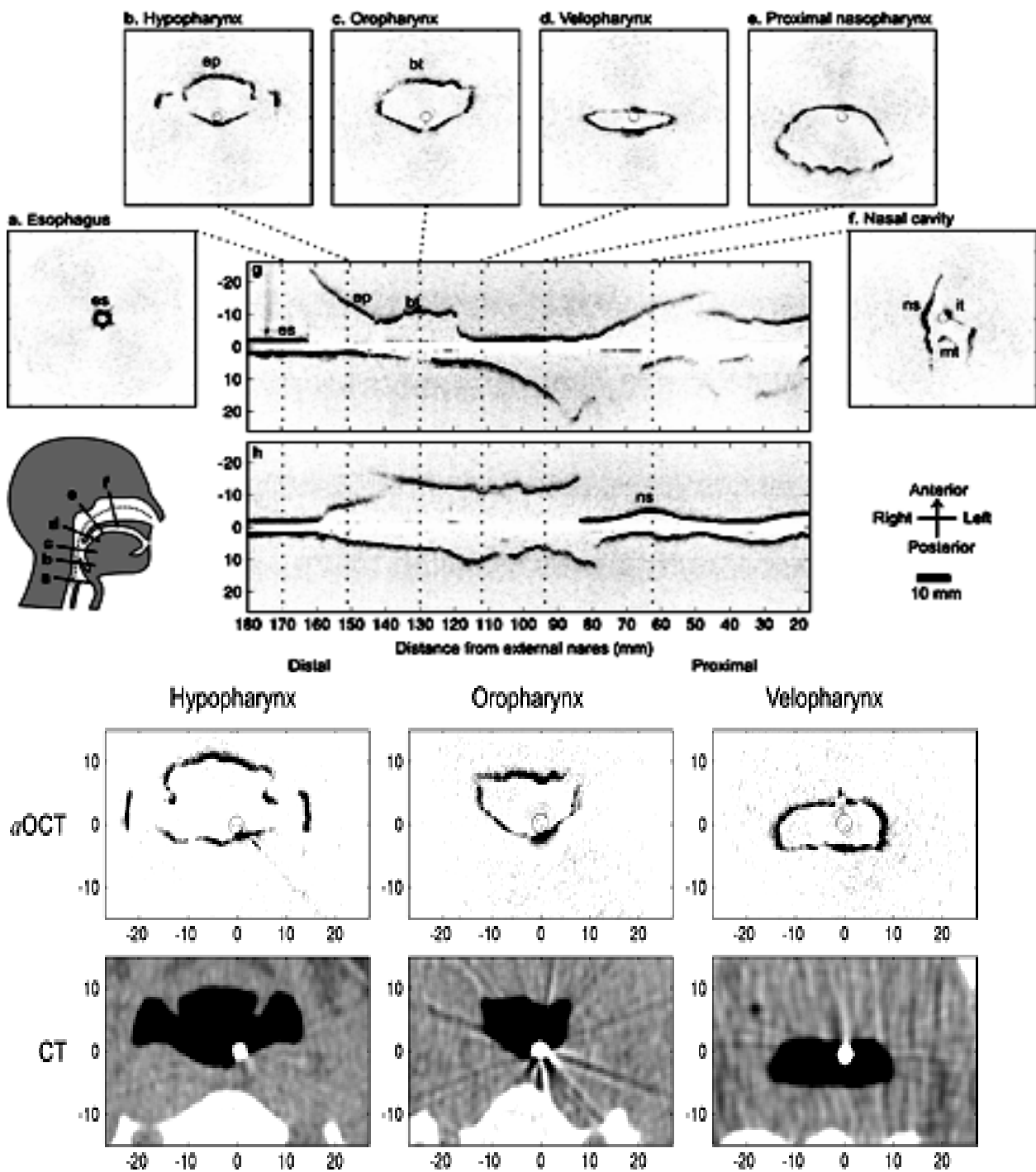


Figure 8. Simultaneous Anatomic Optical Coherence Tomography (*aOCT*) scans and CT scans obtained in the hypopharynx, oropharynx, and velopharynx in patient with OSAS versus normal person (*Julian et al., 2006*).

- **Differential diagnosis**

All of the possible causes of excessive daytime sleepiness, including OSA, should be considered in patients identified as having excessive daytime sleepiness (*Collop et al., 2007*).

Periodic limb movements of sleep (PLMS) are recurrent jerks of the legs and arms, associated with arousals. They are often observed in association with OSA and can further fragment sleep, even after successful therapy for OSA. Restless legs syndrome (RLS) refers to symptoms of spontaneous, continuous leg movements associated with unpleasant paresthesias. These sensations occur only at rest and are relieved by movement (*Lohmann et al., 2008*).

Upper airway resistance syndrome (UARS) was previously classified as an independent disorder, but it is now considered a type of OSA according to the International Classification of Sleep Disorders (*American Academy of Sleep Medicine, 2005*). It exists when there are arousals from sleep induced by airflow limitation due to increased upper airway resistance (i.e. RERA more than 10 events per hour sleep measured using esophageal pressure monitoring). Patients with UARS have few discrete respiratory disturbances (i.e. apneas or hypopneas) or episodes of desaturation. It is common in thin women with certain craniofacial abnormalities. The patient is present by somatic symptoms such as headache, insomnia, irritable bowel syndrome and psychiatric morbidity like depression, attention deficit disorders and a tendency to feel light headed (*American Academy of Sleep Medicine, 2005*).

Night-shift workers obtain approximately 7 hours/week less sleep than non-shift workers. They often revert to a daytime schedule on their leisure days, which adds to their sleep deprivation (*Collen et al., 2010*).

Patients with symptoms suggestive of OSA may actually have *narcolepsy*. Sleep attacks are classically described, although there is a wide spectrum of clinical presentations. Many patients complain of

ongoing drowsiness. Narcolepsy is characterized by the classic tetrad of excessive daytime sleepiness, cataplexy, hypnagogic hallucinations, and sleep paralysis. This tetrad is seen only rarely in children. The term narcolepsy is derived from Greek, "seized by somnolence." Narcolepsy frequently is unrecognized, with a typical delay of 10 years between onset and diagnosis. Approximately 50% of adults with the disorder retrospectively report symptoms beginning in their teenage years (*Black et al., 2005*).

OSA must also be distinguished from disorders that mimic OSA such as primary snoring, underlying pulmonary disease, sleep-related laryngospasm, abnormal swallowing disorder, panic attacks, nocturnal asthma, GERD, sleep-choking disorder, hypothyroidism, nocturnal seizures and insomnia (repetitive awakenings) (*Collop et al., 2007*).

Patients who *snore* are typically asymptomatic and present due to complaints from their bed partners. Snoring is far more common than OSA. However, many patients who snore undergo polysomnography to exclude OSA, since snoring is a common symptom among patients with OSA (*Rodsutti et al., 2004*).

Respiratory diseases can cause excessive daytime sleepiness either alone or in combination with OSA. As an example, patients with either chronic obstructive lung disease or restrictive lung disease may have sleep-related desaturation. Sudden awakenings and dyspnea can result, imitating OSA. In addition, patients with neuromuscular disease depend on their accessory muscles to breathe and inhibition of muscle activity during REM sleep can cause profound desaturations and awakenings. Poorly controlled asthma is often worse at night, with

nocturnal bronchospasm and cough inducing sleep fragmentation and paroxysmal dyspnea (*Tawk, et al., 2006*).

Gastroesophageal reflux disease (GERD) can mimic OSA by producing a choking sensation and dyspnea. In addition, GERD can be exacerbated by OSA. Both GERD and OSA appear to improve with positive airway pressure therapy (*Harding, 2007*).

Disease spectrum

Healthy individuals may experience periods of absent or decreased respiration at sleep onset or during rapid eye movement (REM) sleep. This generally lasts less than 10 seconds and is not repetitive. Occasionally, an isolated apnea lasting up to 30 seconds may occur during REM sleep. Such episodes are infrequently accompanied by hypoxemia or followed by arousals or sleep-state changes. There are no known clinical squeale (*Young et al., 2004*).

Some patients who are diagnosed with OSA have an AHI greater than 15 events per hour, but do not exhibit excessive sleepiness and do not have sleep-related comorbidities. Such patients may have an increased risk of developing hypertension (*Young et al., 2004*).

In contrast, other patients who are diagnosed with OSA exhibit excessive sleepiness and/or have sleep-related comorbidities. Such patients are classified as having mild, moderate, or severe disease (*Flemons, 2002*).

Patients who should be classified as having *mild OSA* are those who have an AHI between 5 and 15 respiratory events per hour of sleep. Such patients typically have passive or sedentary daytime sleepiness,

which refers to sleepiness that becomes noticeable once the patient is sedentary and unstimulated. The daytime sleepiness often does not impair daily function and may be unapparent to the patient, although it may be recognized by family members. Alternatively, the daytime sleepiness may become apparent to the patient only after it improves due to weight loss, alcohol abstinence, or treatment of OSA. The sleep stages and slow wave sleep are generally preserved in mild OSA and systemic hypertension, corpulmonale, and polycythemia are generally absent. Approximately 30 percent of patients with mild OSA will tolerate and respond to treatment, compared to placebo. Specifically, positive airway pressure therapy will reduce daytime sleepiness and blood pressure (*Giles et al., 2006*).

Patients who should be classified as having *moderate OSA* are those who have an AHI between 15 and 30 respiratory events per hour of sleep. Such patients are typically aware of their daytime sleepiness and take steps to avoid falling asleep at inappropriate times (eg, taking a nap or avoiding driving). They are able to continue their daily activities, but at reduced levels, and they may have an increased incidence of motor vehicle violations or accidents. Systemic hypertension may exist, but daytime signs or symptoms of corpulmonale are usually absent. Sleep fragmentation is observed, but the progression of sleep stages is better conserved than with severe disease. Patients with moderate OSA usually respond to PAP therapy with an improvement in the daytime sleepiness, quality of life, and blood pressure (*Giles et al., 2006*).

Patients who should be classified as having *severe OSA* are those who have an AHI >30 respiratory events/hour of sleep and/or an oxyhemoglobin saturation < 90 % for >20 % of the total sleep time. Such patients typically have disabling daytime sleepiness that interferes with

normal daily activities. They tend to fall asleep during the day (often in a sitting posture) and are at risk for accidental injury. In addition, there are signs of cardiopulmonary failure, nocturnal angina, polycythemia, or cor pulmonale. Patients with severe OSA benefit from prompt therapeutic intervention. Treatment will improve daytime sleepiness, hypertension, and, possibly, other hypoxemia-related abnormalities, such as polycythemia and cor pulmonale (*Giles et al., 2006*).

Treatment

- **General approach**

The diagnosis of OSA should be firmly established and its severity determined prior to deciding whether therapy is indicated. The disease severity guides management by identifying patients who are at greatest risk for adverse outcomes and providing a baseline from which to measure the effectiveness of subsequent treatment (*Epstein et al., 2009*).

Once the diagnosis of OSA is confirmed and its severity determined, the results of all testing should be reviewed with the patient. The patient should be educated about the risk factors, natural history, and consequences of OSA. In addition, the patient should be warned about the potential consequences of driving or operating other dangerous equipment while sleepy and counseled to avoid activities that require vigilance and alertness if sleepy (*Phillips et al., 2005*).

Finally, it should be determined whether treatment is indicated and, if so, which therapy is most appropriate. The benefits of successfully treating OSA include clinical improvement (eg, less daytime sleepiness, better quality of life, improved hypertension), reduced health care utilization and costs and, possibly, decreased mortality (*Giles et al., 2006*).

- **Choosing a therapy**

The types of behavior modification that should be instituted depend upon the characteristics of the patient. Overweight or obese patients should be encouraged to lose weight. Patients with positional OSA should change their sleep position. All patients should abstain from alcohol and avoid medications that may worsen their OSA (*Gay et al., 2006*).

For patients with *severe OSA* (AHI >30 events per hour and/or severe clinical sequelae), Positive airway pressure is used as first-line therapy. This is based on the variable efficacy of oral appliances in this patient population (*Vennelle et al., 2010*).

For patients with *mild or moderate OSA* (AHI \leq 30 events per hour without severe clinical sequelae) who do not express a preference, positive airway pressure is preferred to an oral appliance because it is superior at reducing the frequency of obstructive events (*Vennelle et al., 2010*).

In contrast, for patients with *mild or moderate OSA* who prefer an oral appliance, it is initiated rather than positive airway pressure. This is based on recognition that most patients prefer an oral appliance, adherence is an essential aspect of successful treatment, both modalities are effective compared to no treatment or a sham treatment, and both modalities have a similar effect on symptoms (*Vennelle et al., 2010*).

Uvulo-palato-pharyngo-plasty (UPPP) or an alternative surgical therapy are considered when positive airway pressure or an oral appliance is declined, ineffective (after at least a three month trial of therapy), or not an option (*Walker et al., 2002*).

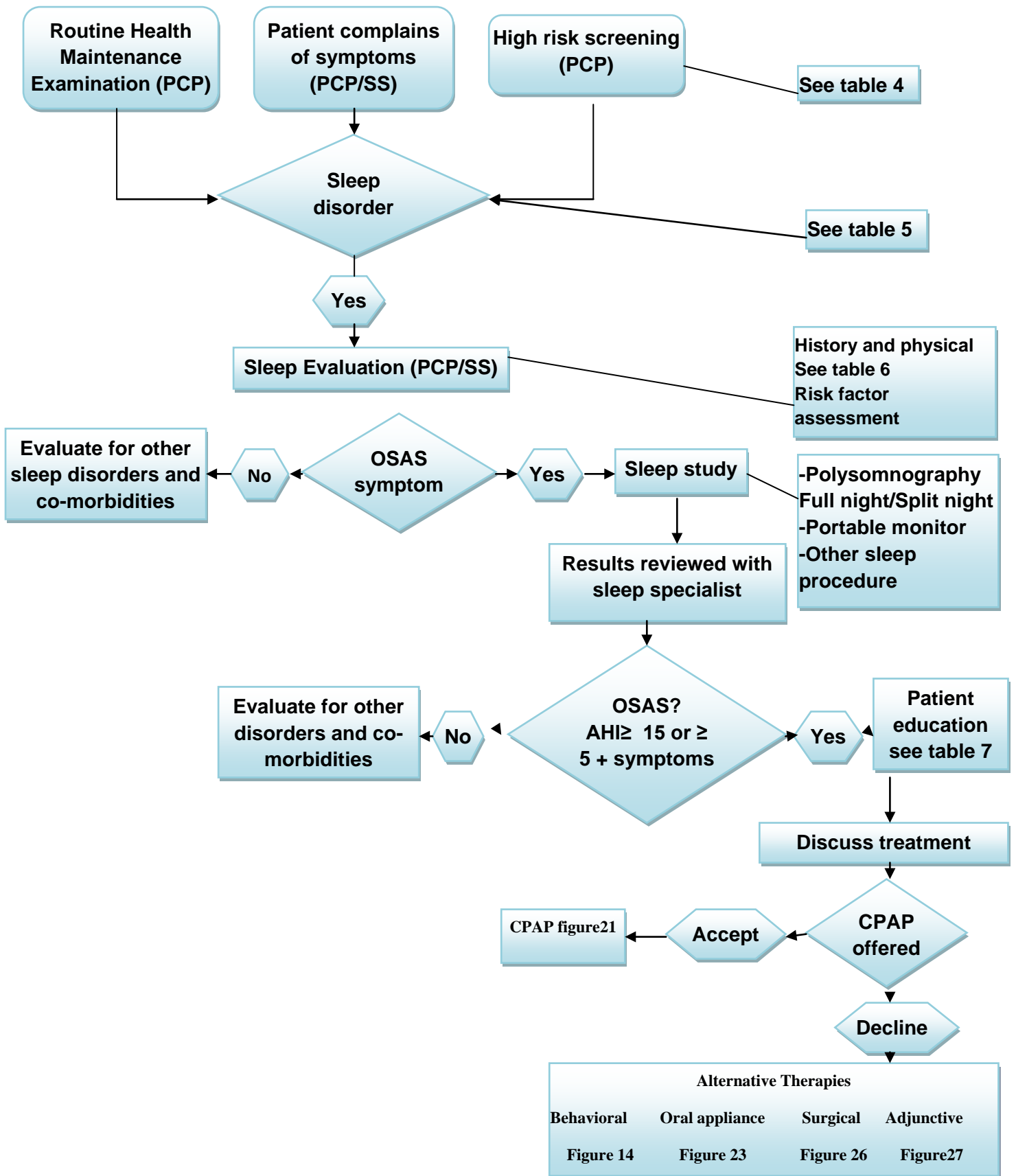


Figure 9. Flow chart for evaluation of patients suspected of having OSA. PCP= Primary care physician, SS= sleep specialist. (Lawrence et al., 2009).

Table 4. Patients at high risk for OSA who should be evaluated for OSA symptoms (*Lawrence et al., 2009*).

-Obesity (BMI >35).
-Congestive heart failure
-Atrial fibrillation
-Treatment of refractory hypertension
-Type 2 diabetes
-Nocturnal dysrhythmias
-Stroke
-Pulmonary hypertension
-High risk driving populations
-Preoperative for bariatric surgery

Table 5. Questions about OSA that should be included in routine Health Maintenance Evaluations (*Lawrence et al., 2009*).

-Does the Patient snore?
-Does the patient complain of day time sleepiness?
-Is the patient obese?
-Does the patient have hypertension ?
-Is the patient retrognathic?

Table 6. OSA symptoms that should be evaluated during a comprehensive sleep evaluation (*Lawrence et al., 2009*).

-Snoring
-Excessive sleepiness not explained by other factors
-Non-refreshing sleep
-Total sleep amount
-Sleep fragmentation/ maintenance insomnia
-Witnessed apneas
-Gasping/ choking at night
-Morning headache
-Nocturia
-Decreased concentration
-Memory loss
-Decreased libido
-Irritability

Table 7. Components of patient education programs (*Lawrence et al., 2009*).

-Findings of study, severity of disease
-Pathophysiology of OSA
-Explanation of natural course of disease and associated disorders
-Risk factor identification, explanation of exacerbating factors, and risk factor modification
-Genetic counseling when indicated
-Treatment options
-What to expect from treatment
-Outline the patient's role in treatment, address their concerns, and set goals
-consequences of untreated disease
-Drowsy driving/ sleepiness counseling
-Patient quality assessment and other feedback regarding evaluation

- **I-Behavior modification**

Behavior modification includes weight loss, sleep position, medication selection and alcohol avoidance. It is a corner stone in OSA management (*Tuomilehto et al., 2009*).

Behavior modification is indicated for all patients who have OSA. Whether OSA-specific therapy (as positive airway pressure, an oral appliance, upper airway surgery) is also indicated depends upon the frequency and severity of both respiratory events (eg, apneas, hypopneas) and oxyhemoglobin desaturation episodes during sleep, as well as the severity of any clinical sequelae. When indicated, OSA-specific therapy should be initiated concomitantly with behavior modification, rather than being delayed until the success or failure of behavior modification has been determined (*Epstein et al., 2009*).

- ***I.a.- Weight loss***

Weight loss should be recommended to all patients who are overweight or obese (*Epstein et al., 2009*). This is based on evidence that weight loss improves overall health, decreases the apnea hypopnea index, improves quality of life, and probably decreases daytime sleepiness (*Tuomilehto et al., 2009*).

The effects of weight loss on OSA were illustrated by a trial that enrolled 72 consecutive overweight patients (mean BMI 32 kg/m²) with mild OSA (mean AHI 10 events per hour of sleep). The patients were randomly assigned to receive a single session of general nutrition and exercise advice, or a more intensive program that included a low calorie diet for three months plus nutrition and exercise counseling for one year. Patients in the latter group had significantly greater weight loss, reduction

in the AHI, and improvement in quality of life compared to the control group. There was no difference in the degree of improvement in daytime sleepiness, but the relevance of this is uncertain since the degree of daytime sleepiness was hardly abnormal at baseline. Studies that included patients with more severe OSA and more daytime sleepiness at baseline suggest that weight loss also improves daytime sleepiness (*Tuomilehto et al., 2009*).

Several observational studies have found that the frequency of respiratory events (eg, apneas, hypopneas) during sleep decreases following bariatric surgery (ie, gastric banding, gastric bypass, gastroplasty, biliopancreatic switch, or duodenal switch) (*Rasheid et al., 2003*) (*Buchwald et al., 2004*) (*Grunstein et al., 2007*).

Most notably, a meta-analysis of 136 studies (22, 094 patients) found that OSA resolved in 86 percent of patients who underwent bariatric surgery. However, the meta-analysis was criticized because most of the studies were case series, the resolution of OSA following bariatric surgery was not defined by polysomnography, and patients with OSA, sleep-disordered breathing, or obesity hypoventilation syndrome were combined under the label OSA (*Buchwald et al., 2004*).

A subsequent meta-analysis of 12 studies (342 patients) was performed to address these limitations. Bariatric surgery was associated with a significant decrease in the BMI (from 55 to 38 kg/m²) and reduction in the mean AHI (from 55 to 16 events per hour of sleep). Although the improvement in the AHI was substantial, the final value was still abnormal. Clinical outcomes such as daytime sleepiness, quality

of life, and mortality were not measured in either meta-analysis(*Greenburg et al., 2009*).

Patients whose OSA improves or resolves after weight loss should strive to maintain their weight loss, since weight gain is associated with worsening of OSA (*Peppard et al., 2000*).

Such patients should also be followed closely because OSA may recur even in patients who maintain their weight loss. Counseling regarding ongoing diet modification and exercise, as well as referral to a nutritionist may be beneficial (*Newman et al., 2005*).

- ***I.b.-Sleep position***

Sleep position is important in treatment of OSA. During the diagnostic sleep study, some patients are observed to have OSA that develops or worsens during sleep in the supine position. These patients tend to have less severe OSA, to be less obese, and to be younger (*Morgenthaler et al., 2006*).

Sleeping in a non-supine position (eg, lateral recumbent) may correct or improve OSA in such patients and should be encouraged. However, sleeping in a non-supine position should not be used as the primary therapy unless normalization of the AHI when sleeping in a non-supine position has been confirmed by polysomnography (*Epstein et al., 2009*).

The benefit of sleeping in a non-supine position was demonstrated by a cross-over trial in which 13 patients with positional OSA (mean AHI 17 events per hour) were randomly assigned to sleep in a non-supine position or to receive continuous positive airway pressure (CPAP) for two

weeks. Both interventions significantly improved the AHI and oxyhemoglobin saturation compared to baseline, although CPAP was more effective in improving each of these parameters. There were no differences in the degree of improvement of clinical outcomes, such as daytime sleepiness (*Morgenthaler et al., 2006*).

Approximately one-half of patients who are encouraged to sleep in the lateral position will learn to do so and will maintain the behavior. A number of devices have been developed to reduce the likelihood of sleeping in the supine position, including posture alarms, special pillows (figures 11, 12), and modified nightshirts. A simple device that can be made at home is a snug-fitting T-shirt with a pocket sewn over the spine and tennis balls placed in the pocket. Obese patients may benefit from a harder type of ball, such as a baseball (figure 10). The discomfort associated with rolling into the supine position is generally enough to prompt the patient to roll back into the lateral position without awakening. This technique appears to be limited by poor adherence (*Bignold et al., 2009*).



Figure 10. Repositional sleep T-shirt used in treatment of OSAS (*Bignold et al., 2009*).

The Contour CPAP Pillow improves CPAP Compliance through making CPAP use more easier, providing more comfortable sleep for all CPAP users by supporting neck and alignment of spines and airway, so, decreasing the cause of snoring and sleep apnea and reducing lower back and neck pain. It Improves arm circulation, abolishing numbness and pain. It reduces mask leaks, pressure on mask and face and mask discomfort (*Bignold et al., 2009*).

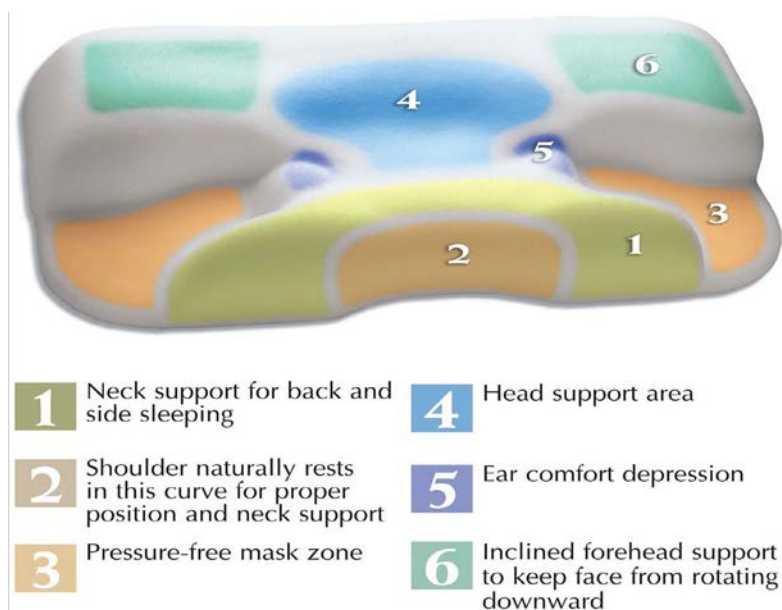


Figure 11. The Contour CPAP Pillow shape allows natural resting of shoulders, neck and head for contact free CPAP mask use. Each Contour CPAP pillow comes fitted with a custom, quilted pillow case with an attached hose tether to keep CPAP hose apparatus away from the patient (*Bignold et al., 2009*).



Figure 12. The Contour CPAP Pillow works with all major brands of CPAP masks (*Mador et al., 2005*).

A positional device that makes the patient sleep on his sides may be an alternative to CPAP in solving select sleep apnea cases. Apneic episodes caused by sleeping on back can be nearly eliminated by changing positions. The Zzoma Positional Sleeper is a large harness designed to prevent people from sleeping on their backs. It resembles a large backwards fanny pack (*Bignold et al., 2009*).



Figure 13. The Zzoma Positional Sleeper(*Bignold et al., 2009*).

- ***I.c.-Alcohol avoidance***

All patients with OSA should avoid alcohol, even during the daytime, because it can depress the central nervous system, exacerbate OSA, worsen sleepiness, and promote weight gain. The effect of alcohol consumption was illustrated by a series with seven patients who had varying degrees of upper airway obstruction during sleep, ranging from snoring alone to OSA. Following alcohol ingestion, the duration and frequency of obstructive respiratory events and the degree of oxyhemoglobin desaturation increased in five patients (71 percent). Two patients who had snoring alone at baseline developed frank OSA after alcohol ingestion (*Randerath et al., 2011*).

- ***I.d.-Medication selection***

The clinician of the patient should be informed that the patient has OSA, since certain medications should be avoided if reasonable alternatives exist. These include medications that inhibit the central nervous system, such as benzodiazepines and benzodiazepine receptor

agonists, as well as barbiturates, other anti-epileptic drugs, antidepressants, antihistamines, and opiates. When these medications are felt to be necessary despite the patient's OSA, their use should be monitored closely and the dose carefully titrated if possible (*Veasey et al., 2006*).

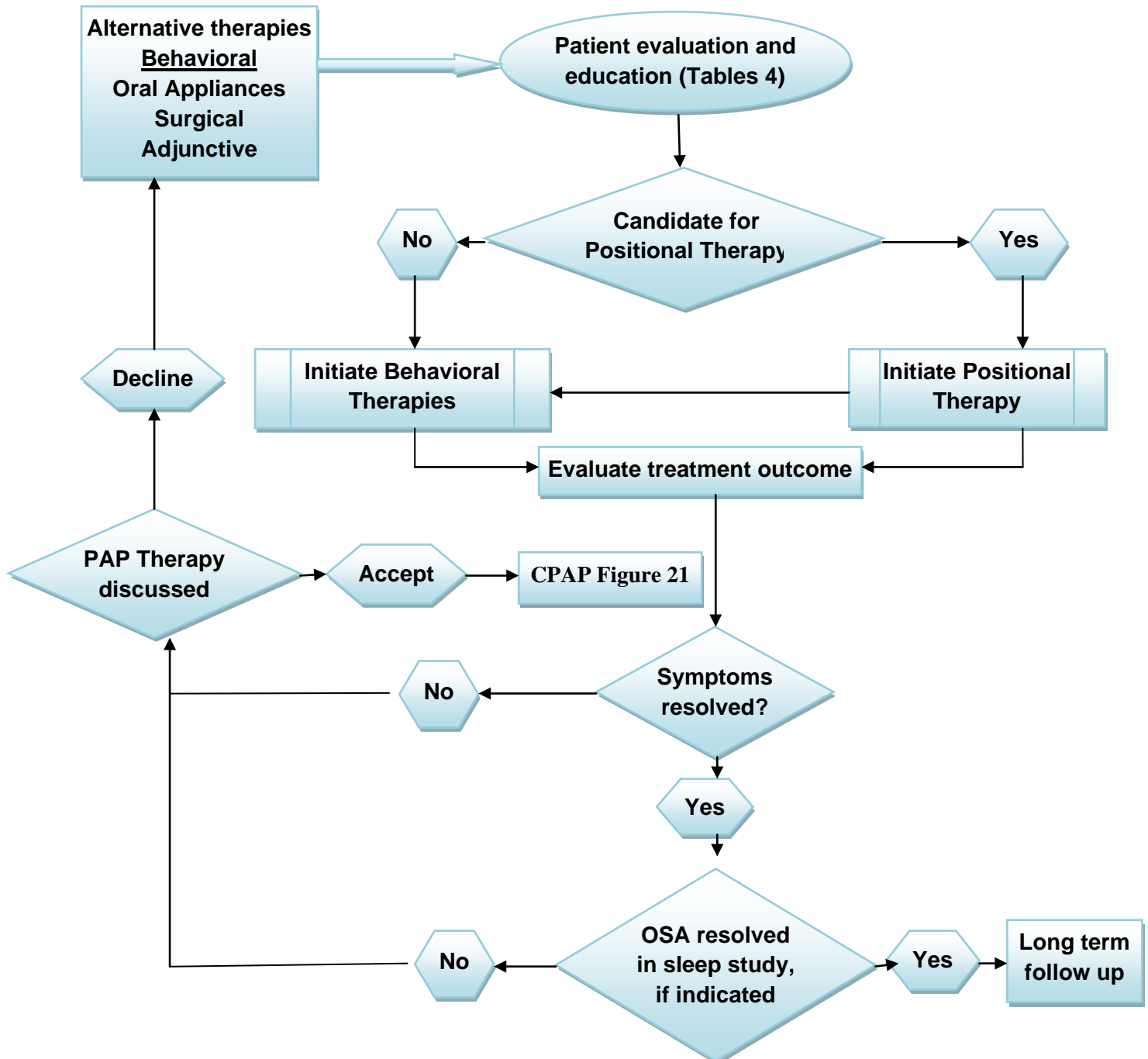


Figure 14. Behavioral treatment. Approach to initiation, management and follow up of behavioral treatment. (*Lawrence et al., 2009*).

- **II-OSA specific therapies**

OSA specific therapies include positive airway pressure, oral appliances, surgical treatment and pharmacological treatment (*Woodson et al., 2001*).

- ***II.a-Positive airway pressure***

It was first described by Sullivan in 1981. It provides pneumatic splinting of the upper airway as a result, respiratory events due to upper airway collapse (eg, apneas, hypopneas) are prevented (*Campos et al., 2005*).

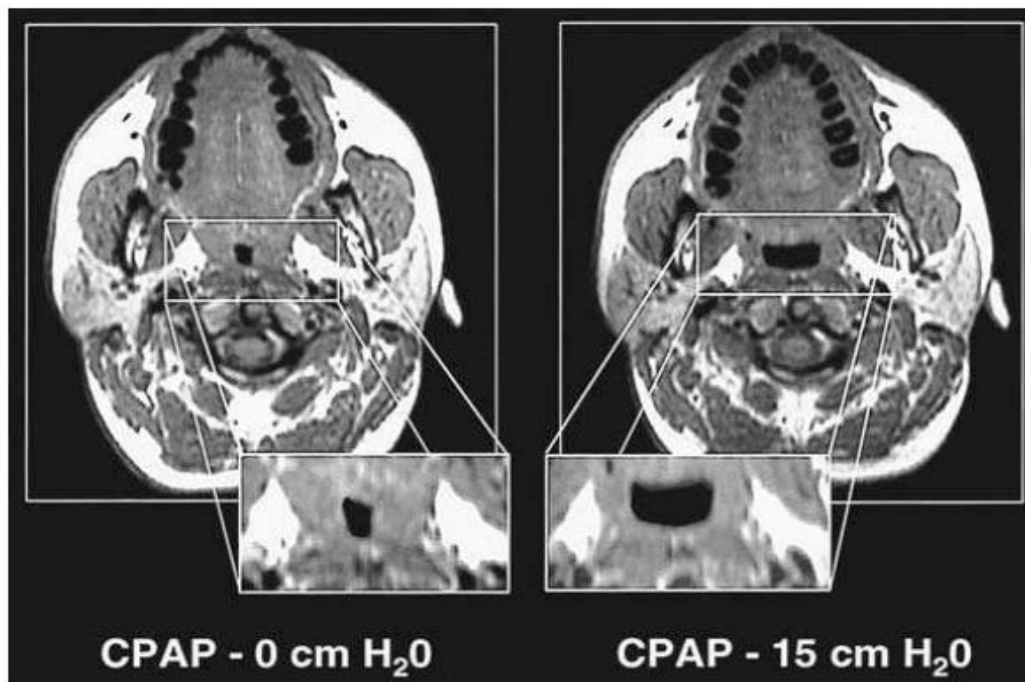


Figure 15. Axial MR image at the retropalatal region in a normal subject with 0 cmH₂O and CPAP of 15 cmH₂O. Airway enlargement is demonstrated predominantly in the lateral dimension with the application of 15 cmH₂O of CPAP. The anterior-posterior airway dimension is not significantly changed with CPAP at 15 cmH₂O (*Terri et al., 2010*).

Whenever positive airway pressure is the therapy chosen, available modes include continuous positive airway pressure (CPAP),

bilevel positive airway pressure (BPAP), and autotitrating positive airway pressure (APAP). Partial pressure reduction during expiration (pressure relief) can also be added. PAP applied through a nasal, oral or oro-nasal interface during sleep is the preferred treatment for OSA (*Epstein et al., 2009*).

Positive airway pressure therapy is generally considered first-line therapy for OSA. Generally, CPAP is favored because it is the most familiar and best studied. A trial of BPAP or APAP is appropriate for patients who do not tolerate CPAP. BPAP is appropriate initial therapy for patients with coexisting central sleep apnea or significant hypoventilation (*Marti et al., 2002*).



Figure16.Contineous positive airway pressure (*Marti et al., 2002*).

PAP is indicated for the treatment of moderate to severe OSA (standard) and mild OSA (option). CPAP is also indicated for improving self reported sleepiness (standard), improving quality of life (option), and

as adjunctive therapy to lower blood pressure in hypertensive patients with OSA (option) (*Fietze et al., 2007*).

CPAP delivers positive airway pressure at a level that remains constant throughout the respiratory cycle. It is used most often because it is the simplest, the most extensively studied, and associated with the most clinical experience (*Collen et al., 2010*).

BiPAP delivers a preset inspiratory positive airway pressure (IPAP) and expiratory positive airway pressure (EPAP). The tidal volume is related to the difference between the IPAP and EPAP. As an example, the tidal volume is greater using an IPAP of 15 cm H₂O and an EPAP of 5 cm H₂O (difference of 10 cm H₂O), than an IPAP of 10 cm H₂O and an EPAP of 5 cm H₂O (difference of 5 cm H₂O). There is no proven advantage to using BPAP instead of CPAP for the routine management of OSA (*Skomro et al., 2010*).

APAP increases or decreases the level of positive airway pressure in response to a change in airflow, a change in circuit pressure, or a vibratory snore (signs that generally indicate that upper airway resistance has changed). The degree of improvement of major outcomes conferred by APAP and CPAP is similar. However, APAP is preferred by more patients, although it has not been shown to improve adherence (*Fietze et al., 2007*).

Treatment with PAP should ideally be approached on case management basis utilizing a multidisciplinary care team that can include a sleep specialist, the referring physician, nursing personnel, respiratory therapist and sleep technologist (*Campos et al., 2005*).

Patients should be educated about the function, care and maintenance of their equipment, the benefits of PAP therapy and potential problems. Patients, in conjunction with their care team, should work together to select the appropriate PAP interface which may be full face mask, oral mask, nasal mask, total face mask, nasal pillow and nasal prongs. The addition of heated humidification and systematic educational program is indicated to improve CPAP utilization (standard). CPAP usage should be objectively monitored with time meters to help assure utilization (*Terri et al., 2010*).

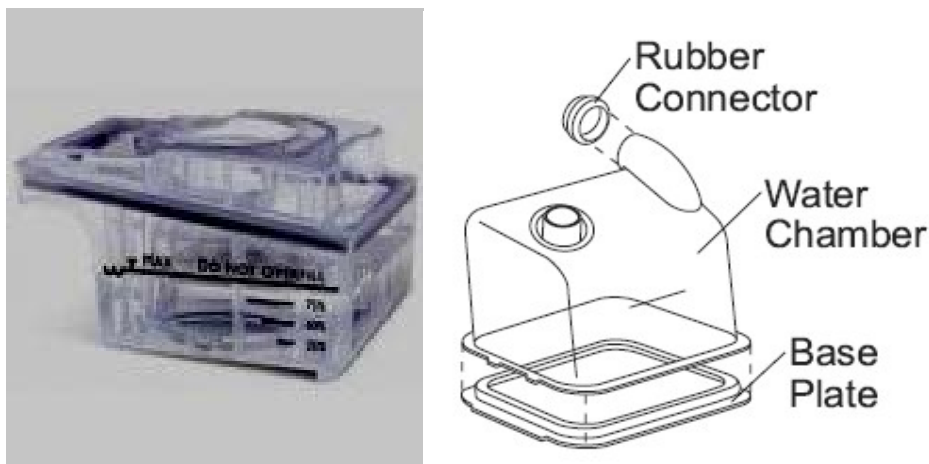


Figure17. humidifier of CPAP (*Terri et al., 2010*).



Figure18. Nasal prong and nasal pillow for delivery of Pressure generated by CPAP to patient (*Terri et al., 2010*).



Figure 19. Nasal and oral masks as part of CPAP interface (*Terri et al., 2010*).



Figure 20. Full face mask and total face mask as part of CPAP (*Fietze et al., 2007*).

There is high quality evidence from meta-analyses of randomized trials that positive airway pressure therapy reduces the frequency of respiratory events during sleep, decreases daytime sleepiness, and improves quality of life (*Phillips et al., 2005*).

A meta-analysis of 22 randomized trials (1160 patients) that compared nocturnal CPAP to a control was done. Controls included sham CPAP, placebo tablets, or conservative management, such as weight loss and sleep hygiene. Nocturnal CPAP significantly improved both subjective and objective sleepiness, quality of life, cognitive function, and depression. Mortality was not an outcome that was evaluated in this meta-analysis (*Giles et al., 2006*).

No randomized trial has found that positive airway pressure therapy improves mortality in patients with OSA. This may be because early randomized trials that compared positive airway pressure to either no therapy or a sham therapy usually measured outcomes other than mortality, such as the frequency of respiratory events during sleep and daytime sleepiness. Now that the beneficial effect of positive airway pressure on these outcomes is widely accepted, it is unlikely that similar trials will ever be performed to evaluate mortality because of concerns about whether it is appropriate to randomize a patient to no treatment (*Marti et al., 2002*).

However, an observational study of 385 patients who were being evaluated for OSA found an 8-year survival of 100 percent among patients who were treated with CPAP, compared to 63 percent among all patients, suggesting that treatment may improve mortality (*Patel et al., 2003*). Other observational studies similarly suggest that CPAP improves mortality (*Campos et al., 2005*).

Favorable outcomes likely depend on adherence to positive airway pressure therapy. However, it is estimated that 20 to 40 percent of patients do not use their positive airway pressure device and many others do not use it all night, every night (*Gay et al., 2006*).

Titration of CPAP pressure should be done either by full night or split night polysomnography or using Auto CPAP. An alternative way involves the use of predictive formula. Three predictive equations can be used for calculating the therapeutic pressure of CPAP including *Hoffstein et al, 1994* equation (therapeutic pressure= $(0.16 \times \text{BMI} + 0.13 \times \text{neck circumference (NC)} + 0.04 \times \text{AHI} - 5.12)$) which is most commonly used,

Seriês et al., 2000 equation ($0.193 \times \text{BMI} + 0.077 \times \text{NC} + 0.02 \times \text{AHI} - 0.611$) and *Stardling et al., 1994* equation ($0.048 \times \text{Oxygen Desaturation Index (ODI)} + 0.128 \times \text{NC} + 2.1$).

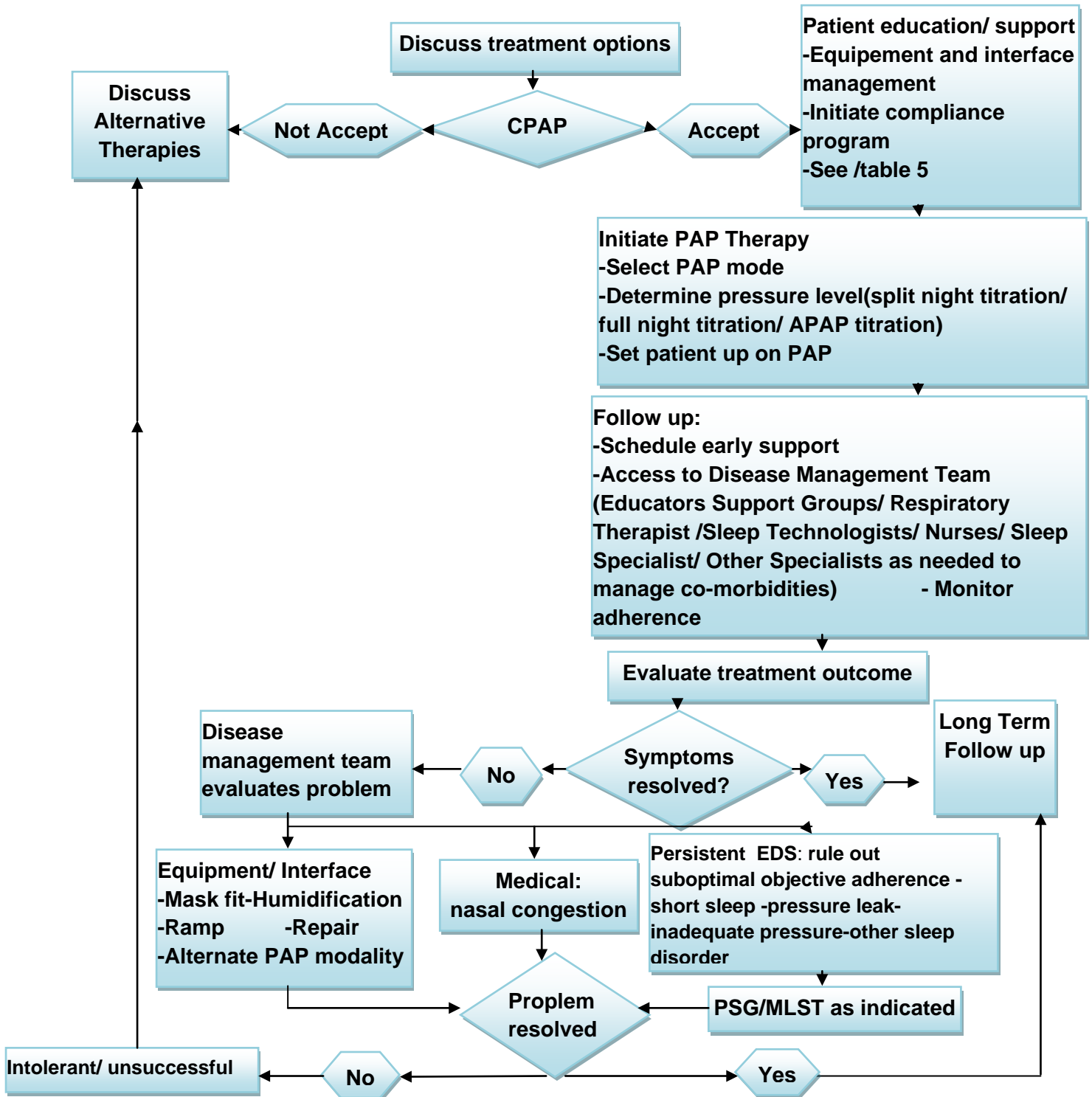


Figure 21. Approach to initiation, management and follow up of CPAP (*Lawrence et al., 2009*).

- ***II.b.-Oral appliances***

Oral appliances may improve upper airway patency during sleep by enlarging the upper airway and/or by decreasing upper airway collapsibility (e.g. improving upper airway muscle tone). Mandibular repositioning appliances (MRA) cover the upper and lower teeth and hold the mandible in advanced position with respect to the resting position. Tongue retaining devices (TRD) hold only the tongue in a forward position with respect to the resting position. Either design holds the soft tissues of the oropharynx away from the posterior pharyngeal wall, thereby maintaining upper airway patency (*Kushida et al., 2005*).

Klearway™ offers several advantages over other appliances. It works by keeping the teeth together and holding the lower jaw and tongue forward during sleep to open the airway. It possesses better retention characteristics designed to keep the appliance in the mouth during all the various complex jaw movements which can occur during sleep. Also, it provides full occlusal coverage of both arches and is very carefully designed not to encroach on tongue space. Furthermore, it facilitates the very slow and gradual movement of the mandible by permitting the patient to adjust the appliance according to his/her own comfort level with the guidance of the attending dentist. Klearway™ allows the patient to feel less restricted and thus less claustrophobic. Once warmed under hot water and inserted, the acrylic resin hardens as it cools to body temperature and firmly affixes itself to both arches. Lateral and vertical jaw movement is permitted which enables the patient to yawn, swallow, and drink water without dislodging the appliance (*Barnes et al., 2004*).



Figure 22. Klearway™ oral appliance used for treatment of OSAS(*Barnes et al., 2004*).

Oral appliances (OAs) decrease the frequency of respiratory events, arousals, and episodes of oxyhemoglobin desaturation, compared to no treatment or a sham intervention. They may also improve daytime sleepiness, quality of life, and neurocognitive function. Their impact on mortality is unknown (*Kathleen et al., 2006*).

Although not as effective as CPAP, OAs are indicated for use in patients with mild to moderate OSA who prefer OAs to CPAP, or who don't respond to CPAP, are not appropriate candidates for CPAP, or who fail CPAP or behavioral measures. OAs are appropriate for patients with primary snoring who don't respond to, or are not appropriate candidates for, treatment with behavioral measures (*Lawrence et al., 2009*).

The presence or absence of OSA must be determined before initiating treatment with OAs to identify those patients at risk due to complications of sleep apnea and to provide a baseline to establish the effectiveness of subsequent OAs treatment. The severity of OSA must be established. Patients should undergo a thorough dental examination to assess candidacy for an OA (*Kushida et al., 2005*).

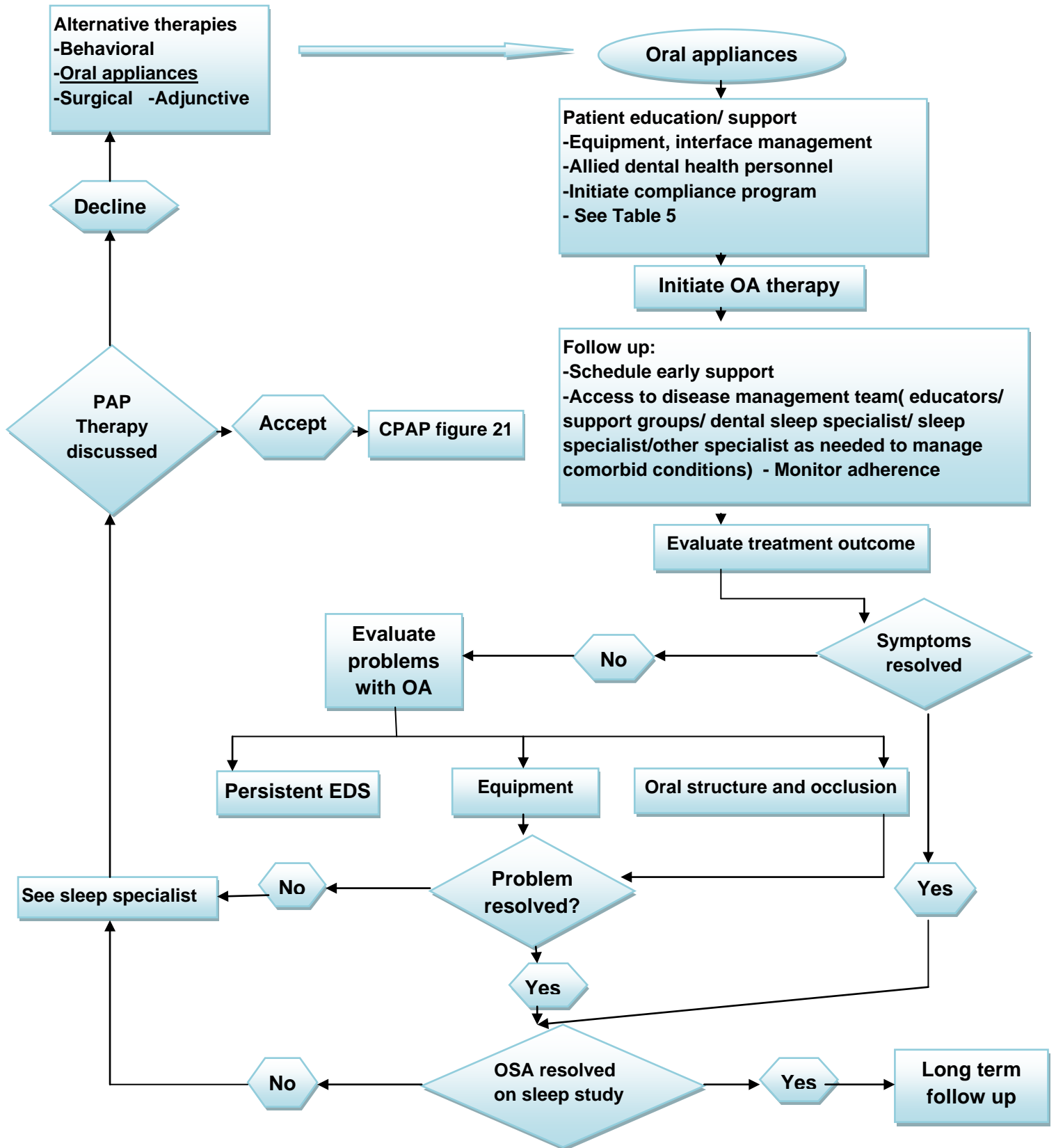


Figure 23. Oral appliances. Approach to initiation, management and follow up of patients using custom OA therapy (Lawrence et al., 2009).

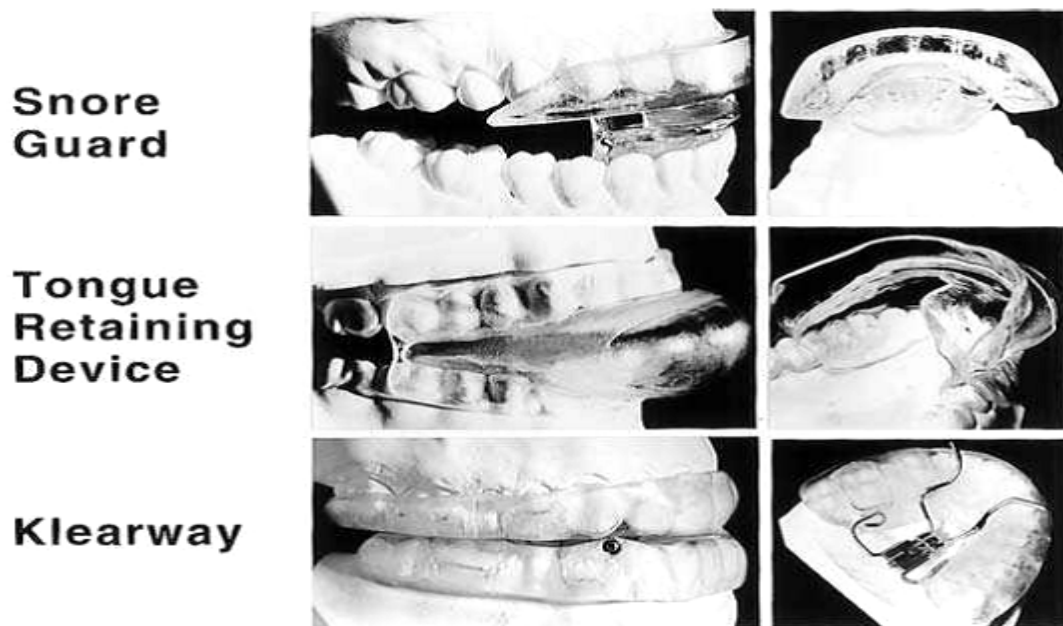


Figure 24. Lateral and single arch views of the Snore Guard, Tongue Retaining Device, and Klearway_ appliances (*Kushida et al., 2005*).

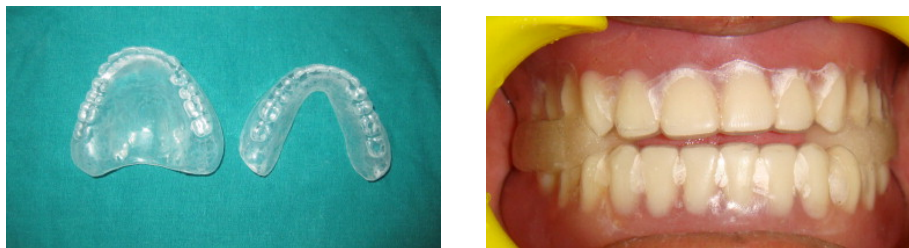


Figure 25. Frontal view with modified mandibular advancement appliance (*Chan et al., 2007*).

- ***II.c.- Surgical treatment***

It appears to be most effective in patients who have mild OSA due to a severe, surgically correctable obstructing lesion. As an example, surgery is a reasonable initial approach to treating a patient who has mild OSA that is likely due to tonsillar hypertrophy that is obstructing the pharyngeal airway (*Epstein et al., 2009*).

There is no consensus regarding the role of surgery in the absence of a strictly defined anatomic lesion (*Phillips, 2005*).

Uvulopalatopharyngoplasty (UPPP) is one of the most common surgical procedures that is performed in this context. It involves resection of the uvula, redundant retrolingual soft tissue, and palatine tonsillar tissue. Laser-assisted and radiofrequency ablation (RFA) are less invasive variants of UPPP. Other common surgical procedures for OSA include septoplasty, rhinoplasty, nasal turbinate reduction, nasal polypectomy, palatal advancement pharyngoplasty, tonsillectomy, adenoidectomy, palatal implants (ie, pillar procedure), tongue reduction (partial glossectomy, lingual tonsillectomy), genioglossus advancement, and maxillomandibular advancement (*Powell, 2005*).

Only a small number of trials have compared surgery to either conservative management or a nonsurgical therapy. Overall, the trials have failed to consistently demonstrate a benefit from surgical therapy. While this could be a true effect, it may also reflect the small sample sizes, the heterogeneous patient populations, or the use of short-term outcome measures (*Sundaram et al., 2005*).

A trial randomly assigned 32 patients with OSA and >50 percent obstruction at the palatal level to receive conservative management or to undergo UPPP, with or without mandibular osteotomy. At one year, there was improvement of subjective daytime sleepiness with UPPP compared to conservative treatment, according to an unvalidated sleepiness scale. In addition, more patients in the UPPP group had a normal oxygen desaturation index. However, the surgical complication rate was 22 percent (*Pazos et al., 2001*).

UPPP appears to achieve a surgical cure (defined as a postoperative AHI of <5 events per hour of sleep) in only a minority of

patients (*Khan et al., 2009*), and may compromise subsequent CPAP therapy by promoting mouth leaking and reducing the maximal level of pressure tolerated by many patients treated with CPAP (*Terris et al., 2001*).

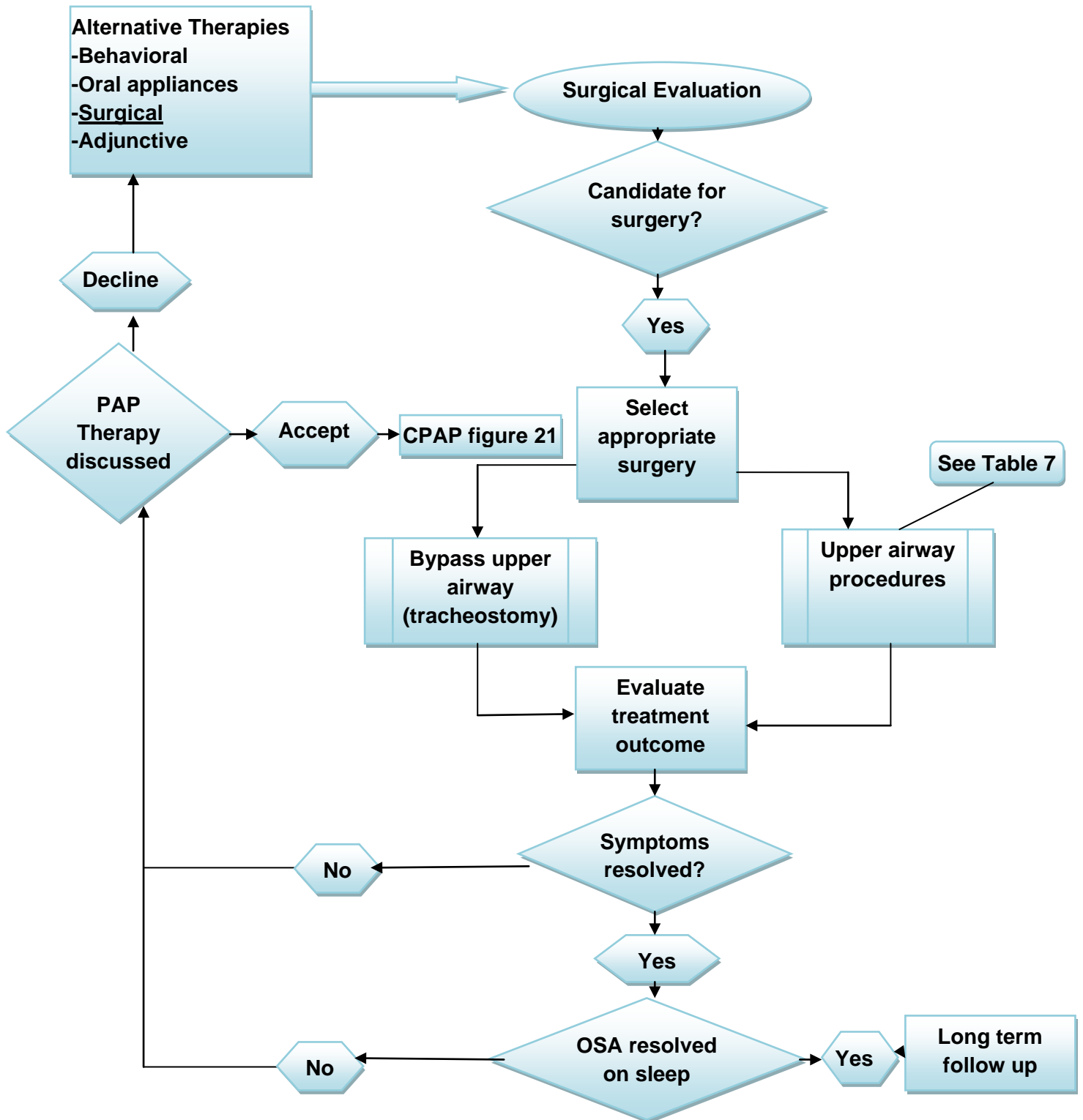


Figure 26. Approach to management of OSA with surgical therapy (*Lawrence et al., 2009*).

Table 8. Common surgical procedures for OSA (*Lawrence et al., 2009*).

Upper airway bypass procedure	Tracheostomy
Nasal procedure	Septoplasty
	Functional rhinoplasty
	Nasal valve surgery
	Turbinate reduction
	Nasal polypectomy
	Endoscopic procedures
Oral, Oropharyngeal and Nasopharyngeal procedures	Uvulopalatopharyngoplasty and variations
	Palatal advancement
	Pharyngoplasty
	Tonsillectomy and/or adenoidectomy
	Excision of tori mandibularis
	Palatal implants
Retroglossal procedures	Tongue reduction <ul style="list-style-type: none"> • Partial glossectomy • Tongue ablation • Lingual tonsillectomy
	Tongue advancement/ stabilization <ul style="list-style-type: none"> • Genioglossus advancement • Hyoid suspension • Mandibular advancement • Tongue suspension
Laryngeal procedures	Epiglottoplasty
	Hyoid suspension
Global airway procedures	Maxillomandibular advancement
	Bariatric surgery

- ***II.d.-Pharmacologic***

Pharmacological agents have been investigated as potential primary therapies for patients with OSA. However, no agent has been

identified that prevents or overcomes upper airway obstruction enough to justify pharmacologic therapy as a primary therapy in the routine management of patients with OSA. It is unknown whether pharmacologic agents used concurrently with positive airway pressure therapy lowers the amount of airway pressure required to treat the disorder (*Randerath et al., 2011*).

Acetazolamide stimulates the ventilatory drive by inducing metabolic acidosis. However, there is a paucity of high quality evidence regarding its impact on patients with OSA. Acetazolamide may decrease the frequency of obstructive events, but may not eliminate OSA or its symptoms. Until the effects of acetazolamide are better characterized cannot be recommended for the routine care of patients with OSA (*Yvonne et al., 2011*).

Donepezil is a cholinesterase inhibitor that may improve OSA in patients with Alzheimer disease. Donepezil significantly decreased the frequency of obstructive apneas and hypopneas (from 19 to 9 events per hour of sleep) and the duration with an oxyhemoglobin saturation below 90 percent (from 13.5 to 4 percent of the total sleep time). It also blunted the severity of oxyhemoglobin desaturation and improved cognitive performance (*Moraes et al., 2008*).

Theophylline increases ventilatory drive, likely through its inhibition of adenosine. It has been used to treat patients with periodic breathing or central sleep apnea due to left ventricular systolic dysfunction. In patients with OSA, however, theophylline further disrupts sleep and only minimally reduces (ie, does not normalize) the frequency of obstructive events. Theophylline therapy requires that serum levels be

monitored closely because of its narrow therapeutic index and potential for significant toxicity (*Hein et al., 2000*).

Progestational agents are respiratory stimulants whose effect on patients with OSA is uncertain. Some studies have reported a net beneficial effect (*Manber et al., 2003*), while others have not (*Saletu et al., 2003*).

Hormone replacement therapy in post-menopausal women may cause adverse health outcomes including breast cancer, venous thromboembolism, and symptomatic coronary heart disease (*Rossouw et al., 2002*). In light of the risks of hormonal therapy and the absence of consistent clinical benefit, the routine use of progestational agents in patients with OSA is not warranted (*Epstein et al., 2009*).

The prevalence of OSA in patients with hypothyroidism may be greater than 25 percent (*Skjodt et al., 1999*). Conversely, the prevalence of hypothyroidism in patients with OSA is less than 3 percent. **Thyroxine therapy** in patients with coexisting hypothyroidism and OSA has eliminated the OSA in some studies, even in the absence of weight loss. The resolution of macroglossia may play a role (*Kapur et al., 1998*).

Patients with OSA who have hypothyroidism should be treated with replacement therapy. However, there is no role for thyroid replacement in euthyroid patients with OSA (*Skjodt et al., 1999*).

Serotonergic agents Patients with OSA may have a central deficiency of serotonin activity, which may be partially explained by obesity and insulin resistance. Therefore, serotonin reuptake inhibitors have been tested as a therapy for OSA, but their effectiveness varies.

Paroxetine increased muscle inspiratory activity, but did not alter the duration or number of events (*Hudgel et al., 1995*).

Thus, while serotonergic reuptake inhibitors may decrease the frequency of obstructive events, they do not eliminate OSA or its symptoms and should not be used in the routine management of OSA (*Epstein et al., 2009*).

Protriptyline is a tricyclic antidepressant that potently suppresses REM sleep. In theory, decreasing REM sleep may decrease the frequency or severity of obstructive events because OSA is often most severe during this stage of sleep. Protriptyline is highly anticholinergic and is associated with dry mouth, constipation, and urinary retention. Protriptyline should not be used in the routine management of patients with OSA because side effects are likely and efficacy unproven (*Epstein et al., 2009*).

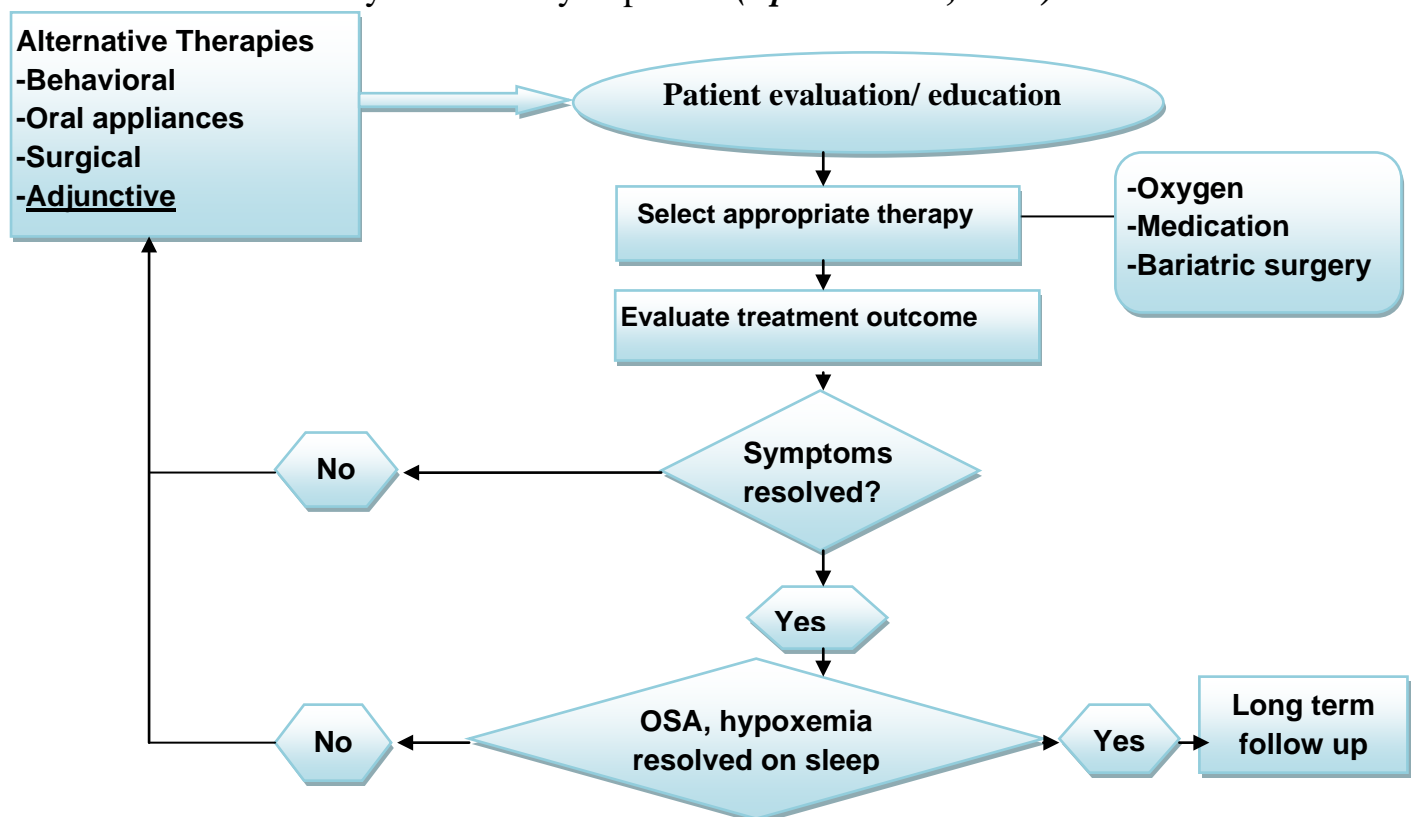


Figure 27. Approach to management of OSA with adjunctive therapies (*Lawrence et al., 2009*).

The three major OSA-specific therapies (positive airway pressure, oral appliances, surgery) have been directly compared. Direct comparisons of positive airway pressure to oral appliances have used CPAP as the mode of positive airway pressure. Trials indicated that CPAP is more effective at reducing the frequency and severity of both respiratory events and oxyhemoglobin desaturation episodes during sleep, but not symptoms. Patients generally prefer oral appliances. Direct comparisons of oral appliances to surgery are scarce, but appear to favor oral appliances. Direct comparisons of surgery to positive airway pressure are also scarce (*Schwartz et al., 2003*).

One trial randomly assigned 90 patients with OSA (mean AHI of about 20 events per hour) to receive temperature controlled radiofrequency tissue ablation (TCRFA), CPAP, or a sham intervention. Patients who received CPAP had a significantly greater reduction in the AHI when compared to patients who received TCRFA. However, there was no difference in daytime sleepiness or quality of life (*Schwartz et al., 2003*).

Follow up

Follow up for patients who receive OSA-specific therapy varies according to the chosen therapy (*Epstein et al., 2009*).

- **Airway pressure**

Patients who elect to be treated with positive airway pressure should be evaluated frequently, especially during the first few weeks of therapy (after one week; then at 3, 6, 12 months interval or when needed). This may include frequent telephone calls and as-needed opportunities to meet face to face with a clinician. The purpose of frequent evaluations is

to quickly identify and manage any side effects that develop, since this may affect long-term adherence with the therapy (*Epstein et al., 2009*).

Once any side effects of the positive airway pressure are successfully managed and the patient is adhering to the therapy, the patient should be asked whether the symptoms of OSA have resolved. An objective sleep evaluation is generally unnecessary if the symptoms of OSA have resolved, but repeat testing is indicated for patients who do not improve or who have recurrent or persistent symptoms such as daytime sleepiness. Objective testing may consist of polysomnography or portable monitoring (*Epstein et al., 2009*).

The purpose of such testing is to help the clinician determine the reason for the treatment failure. Possible causes of treatment failure include nonadherence, weight gain, an inappropriate level of prescribed positive pressure, or an additional disorder causing sleepiness (eg, narcolepsy) that may require alterations in the therapeutic regimen. A review of medications should also be undertaken since many drugs may lead to sleepiness. Sleep deprivation related to lifestyle may also negate the expected effects from treatment of apnea (*Kuna et al., 2011*).

Once the patient's positive airway pressure therapy has been optimized and symptoms resolved, a regimen of long-term follow-up should be established. Annual visits are reasonable, with more frequent visits in between if new issues arise. The purpose of long-term follow-up is to assess usage and monitor for recurrent OSA, new side effects, an air leak, and fluctuations in body weight (*Epstein et al., 2009*).

- **Oral appliance**

Patients treated with an oral appliance should be re-evaluated soon after the initiation of treatment. The purpose of the initial reassessment is to confirm efficacy. This is done by asking whether the symptoms of OSA have resolved and by performing full polysomnography or portable monitoring with the oral appliance in place (*Epstein et al., 2009*).

- **Surgery**

Patients who undergo surgery for OSA should receive surgical and medical follow-up. The frequency of surgical follow-up is determined by the type of surgery, but should include assessment of wound healing, the anatomical result, side effects, and complications. Once the surgical team determines that healing is complete (usually one to three months after the procedure), the efficacy of the procedure should be assessed by asking whether the symptoms of OSA have resolved and by performing full polysomnography or portable monitoring (*Epstein et al., 2009*).

Reassessment is generally coordinated by the sleep specialist. Long-term follow-up by the sleep specialist is indicated to monitor for recurrent OSA. This approach is applicable to patients who underwent surgery because of an obstructing lesion or because an alternative therapy was declined, not an option, or ineffective (*Schumann et al., 2009*).

- **Pharmacologic therapy**

Pharmacologic therapy may be beneficial as adjunctive therapy for excessive daytime sleepiness that persists despite adequate conventional therapy (eg, positive airway pressure, oral appliances). Prior to the initiating pharmacologic therapy, adherence with conventional therapy should be confirmed and alternative causes of daytime sleepiness should be excluded (*Epstein et al., 2009*).

- **Comorbidities**

OSA can worsen other medical conditions, such as hypertension, heart failure, or ischemic heart disease. Any comorbid condition that may be impacted by OSA should be monitored closely following the initiation of OSA-specific therapy. Therapy directed at such comorbidities may need to be modified once therapy for OSA is instituted. As an example, dosages of antihypertensive medications may need to be reduced after successful treatment of OSA (*Somers et al., 2005*).

Adherence to CPAP in OSAS

Continuous positive airway pressure (CPAP) therapy is a highly effective treatment for OSAS, eradicating the airway closure during sleep and thereby reversing the daytime effects of OSAS. Yet, patients' use of CPAP is often less than optimal. Non adherence to the treatment is a significant problem (*Weaver, 2006*).

Definition of adherence

The World Health Organization defines adherence as the “extent to which a person’s behaviour- taking medications, following a diet, and/or executing lifestyle changes- corresponds with agreed recommendations from a health care provider”. The term has come to replace the term compliance, as the use of adherence more explicitly emphasizes the patient as an autonomously acting individual (*Sabate, 2003*).

As the exact amount of CPAP usage necessary to obtain positive health effects is debated, it is difficult to operationalise adherence to CPAP use in a way that is universally agreed upon (*Haniffa et al., 2004*).

Adherence to CPAP is considered as regular use of the CPAP machine; however, the precise frequency of use to attain therapeutic effect is unknown. Recommended use is between 6-8 h per night, but researchers have defined CPAP adherence as anywhere from an average of 4 h a night for 70% of nights. It is not surprising that reports of CPAP adherence range from as low as 28% to more than 83% (*Weaver et al., 2008*).

While it has been common to define adherence as using CPAP for at least 4 h per night (*Weaver, 2002*), there seem to be a dose-response relationship between CPAP usage and reductions in severity indices. Adherence failure is defined as use of CPAP for less than 4 hours/ night in at least 70 percent of nights and /or lack of symptomatic improvement. (*Stepnowsky and Dimsdale, 2002*).

Adherence to CPAP treatment has been described as a significant problem, despite the fact that the treatment improves the life situation for patients with OSAS in many different ways (*Gilles et al., 2006*).

Prevalence

Several studies have reported relatively low CPAP adherence, with reported usage ranging from 65% to 80% and an initial refusal to engage in treatment of 8–15%. It was reported that only 46% of patients were considered adherent when the 4 hours per night rule was applied (*Haniffa et al., 2004*).

Empiric studies have suggested that rates for CPAP use range from 30-60 percent (*Engleman et al., 1994*).

Although the average daily use of those who use CPAP every night is approximately 6 hours, those who routinely skip nights use it on average 3 hours. Moreover, those who use CPAP for shorter durations also skip nights of treatment and this pattern is established early, within the first week of treatment (*Weaver et al., 1997*).

More alarming is the fact that patients who become non adherent in the first few days of CPAP treatment generally remain non adherent (*Aloia et al., 2007*).

Measurement of adherence to CPAP

The earliest studies examining adherence to CPAP therapy used self-report measures, including diaries and verbal recall (*Kribbs et al., 1993*).

Since the publication of these papers, several studies have identified the self report measure of CPAP adherence as unreliable, with reported overestimates of CPAP use by one hour. Subjective reports of CPAP use, measured by self-reported diary records in follow up research visits, consistently overestimated CPAP use (69 ± 110 minutes / day) as compared with microprocessor recordings (*Engleman et al., 1996*).

In a prospective cohort study comparing subjectively reported CPAP use (questionnaires) with an objective measure of CPAP use (by calculating the daily hours of use based on the formula (hours unit powered on/days CPAP use). Objective measured use time was reported as 4.9 ± 0.3 hour/night compared with self-reported use time as 6.1 ± 0.3 hour/night. The study also found that subjects with poor adherence most frequently “misestimated” their CPAP use time (*Rauscher et al., 1993*).

It is necessary to distinguish subjective compliance, defined as the compliance that the patient declares (self-report, questionnaires and phone calls), from objective compliance, which is defined as the average number of hours of running of the machine per 24 hour period, calculated from the built-in time counter of the device. These two concepts have to be distinguished from effective compliance, which is the time spent at the prescribed effective pressure per night (*Marin et al., 2005*).

I-Subjective compliance

I.a. Self-reported compliance

To determine subjective compliance, the patient can be asked simple questions such as how many hours per night and nights per week nasal CPAP is used, usual time of sleeping in the evening, usual time of putting away mask in the morning and usual time of getting up in the morning. The answers to these questions enable the definition of three parameters indicating subjective compliance which include the estimated usage time per night (ie, the reported hours of use per night), reported mean usage time per night (ie, the interval between the reported time of usually going to sleep and the reported time of putting away the mask) and calculated subjective time of use per night (ie, the reported hours of use per night multiplied by the reported nights on CPAP per week divided by 7) (*Marin et al., 2005*).

Raucher et al. 1993 found that subjects with poor adherence most frequently estimated their CPAP use time inaccurately. Self-reports are an inaccurate tool to determine compliance with nasal CPAP therapy for obstructive sleep apnea.

Trying to estimate daily use time by simply asking how many hours a night the patient uses the device generally results in a considerably inaccurate estimation of actual mean treatment time per night because the patient is likely to provide the number of hours of CPAP use solely for the nights it was worn. Including the question about the number of nights on CPAP per week makes self-reports of nasal CPAP use a bit more accurate (*Weaver et al., 2007*).

Part of this discrepancy may be attributed to not using the machine when travelling, during episodes of upper airway infection or during self-prescribed treatment vacancies from time to time. Another explanation is that most patients do not re-establish CPAP after the first awakening (e.g. going to the bathroom) and spend the rest of the night without CPAP. Subjective compliance tells us more about human psychology, social beliefs and hopes, or the patient–doctor relationship than about acting in accordance with a medical request (*Delguste and Rodenstein, 2000*).

I.b. Questionnaires

Questionnaires and rating scales can be useful for measuring a patient's transition from diagnosis to treatment, and for tracking long-term results. As compliant patients are more prone to return a questionnaire, self-selection may be a problem (*Bazzani, 2007*).

Compliance in a study by *Hoffstein et al., 1992* ranged from 82% in repliers to 62% in non repliers who were contacted by telephone.

I.c. Clinical and patient satisfaction

Clinician and patient satisfaction can be assessed using questionnaires. Patients are asked to rate their overall satisfaction with care (1: poor; 5: excellent), their likelihood of continuing to use CPAP (1: not likely; 5: highly likely) and their concern about being monitored (1: not concerned; 5: highly concerned) (*Stepnowsky et al., 2002*).

- ***Self-efficacy***

CPAP self-efficacy refers to OSA patients' confidence or belief that they can adhere to the regimen necessary to manage their OSA with CPAP. The CPAP self-efficacy scale is a five-item self-report scale. This scale assesses the strength of an individual's belief in their ability to

respond to novel or difficult situations and to deal with any associated obstacles or setbacks. Patients were asked to indicate how true they believed 10 generalized self-efficacy statements to be, in relation to themselves, along a scale of 1 (not at all true) to 4 (exactly true) (*Stepnowsky et al., 2002*).

- ***Epworth sleepiness scale***

The Epworth sleepiness scale (ESS) is a very simple, self-administered questionnaire used to assess a patient's own perception of sleepiness and chance of falling asleep. The ESS was developed by *Johns, 1991* from the Epworth Hospital in Melbourne, VIC, Australia.

- ***Functional outcomes of sleep questionnaire***

OSA-specific health-related quality of life can be assessed using the functional outcomes of sleep questionnaire (FOSQ). This is a 32-item, clinically validated self-report measure that assesses the impact of disorders of excessive sleepiness on multiple activities of daily living. The FOSQ is available as an option with selected Philips-Respironics (Murryville, PA, USA) CPAP machines (*Weaver et al., 2007*).

I.d. Phone calls

Patients can be contacted via telephone within the first few weeks of CPAP set-up in order to discuss any concerns they may be having regarding to air pressure, mask fitting, leaks and other issues as they arise. They can also be contacted at the end of months 1, 2 and 3, and then quarterly thereafter, in order to receive any updates regarding pressure, leaks and mask fittings, and answer a list of questions to ensure they are using their equipment properly. Regular telephone support over a 2–3-month period in new or continuing CPAP users resulted in a 1.0–1.3-

hour /night higher utilization compared with a usual-care group (*Chervin et al., 1997*).

Other authors found no increase in CPAP utilization after 12 weeks following an augmented support protocol (educational video, educational session and six calls from a respiratory therapist) (*Hui et al., 2000*).

However, communication between the patient and practitioner is at times delayed when they are unable to connect by telephone (*Taylor et al., 2006*). Use of a video teleconference system for offering specific advice about CPAP has led to a greater rate of compliance compared with a control group (*Smith et al., 2006*).

II. Objective compliance

Objective monitoring of CPAP use has become the standard of care for managing patients with sleep apnea. The tracking systems are not limited to conventional CPAP alone, but also can be utilized in patients being treated with auto-CPAP, bilevel, auto-bilevel, or adaptive servo-ventilation (*Bazzani, 2007*).

II. a. Built-in time counters, real-time clocks and pressure monitors

Objective compliance can be assessed by reading the built-in time counter of the CPAP machine or the effective use recorded using a pressure monitor coupled with a microprocessor (*Nosedá et al., 2000*).

- ***Built-in time counter***

The built-in time counter of the CPAP device measures the cumulative time that the apparatus is turned on (“machine run time” or “CPAP run time”). However, the running time of the machine can be

significantly different from the actual therapeutic mask pressure delivery. Furthermore, the run-time clock provides an average measurement of use between two readings, but no information on the pattern of use during each 24 hour episode (*Nosedá et al., 2000*).

There is an estimated 10 percent difference between machine-on recorded adherence (hour-meter) and mask-on, at effective pressure recorded adherence. By measuring mask-on time at effective pressure, which can be accessed by a card containing a microprocessor chip (smart card-SD cards), memory stick, USB, modem, download cable, wireless transmission or web-based server, this objective measure of CPAP treatment adherence affords new opportunities for insight into CPAP adherence behavior (*Aloia et al., 2007*).

- *Effective compliance*

Effective compliance is based on effective mask pressure measurements. Microprocessors use an algorithm for the detection of “mask-on” pressure. The variable component of the pressure signal given by the pressure transducer is analyzed in order to determine whether the patient is breathing into the mask. Drops in therapeutic pressure greater than 2–5 cm and lasting > 10 seconds can be documented as “mask-off” events. Thus, the duration of therapeutic pressure delivery per day (also referred to as mask time) and number of missed days of use can be determined. From these data, a number of parameters can be calculated (*Reeves-Hoche et al., 1994*).

Table 9. Parameters calculated from the built-in pressure monitor and real-time clock (*Reeves-Hoche et al., 1994*).

Parameter	Definition
% days when CPAP was used	(Number of days when ≥ 1 hour of use was recorded/ total number of follow up days) $\times 100$
% days when CPAP used >4 hour	(Number of days when >4 hour of use was recorded/ total number of follow up days) $\times 100$
Mean daily use	Total hours of CPAP used / total number of follow up days
Mean daily use on days CPAP was used	Total hours of CPAP used/ total number of follow up days when >1 hour of use was recorded
Mean delivered pressure	Arithmetic mean of the pressures delivered in all sessions, weighted by duration of each session
Residual apnea index	Sum of all apneas identified by the monitor, divided by the cumulative time of effective use

A printout can be obtained showing the pattern of CPAP use at home, including the time at which CPAP treatment was started, number of episodes when the pressure was no longer applied during the night, time of quitting CPAP and presence or not of naps with CPAP (figures 28-30) (*Nosedá et al., 2000*).

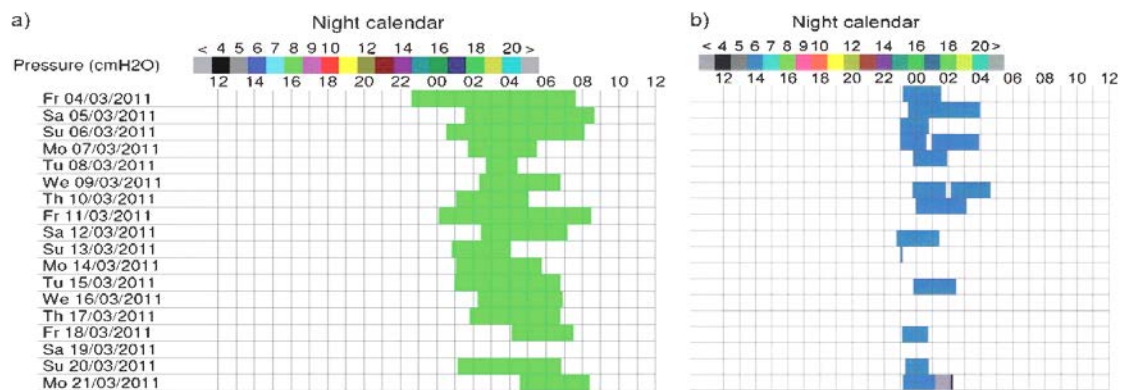


Figure 28. Printouts from a memory card showing the pattern of continuous positive airway pressure (CPAP) use at home, including the times of starting and ending CPAP treatment. a) Moderate CPAP compliance (3–6 hours/night). b) Poor CPAP compliance (<3 hours/night) (*Nosedá et al., 2000*).

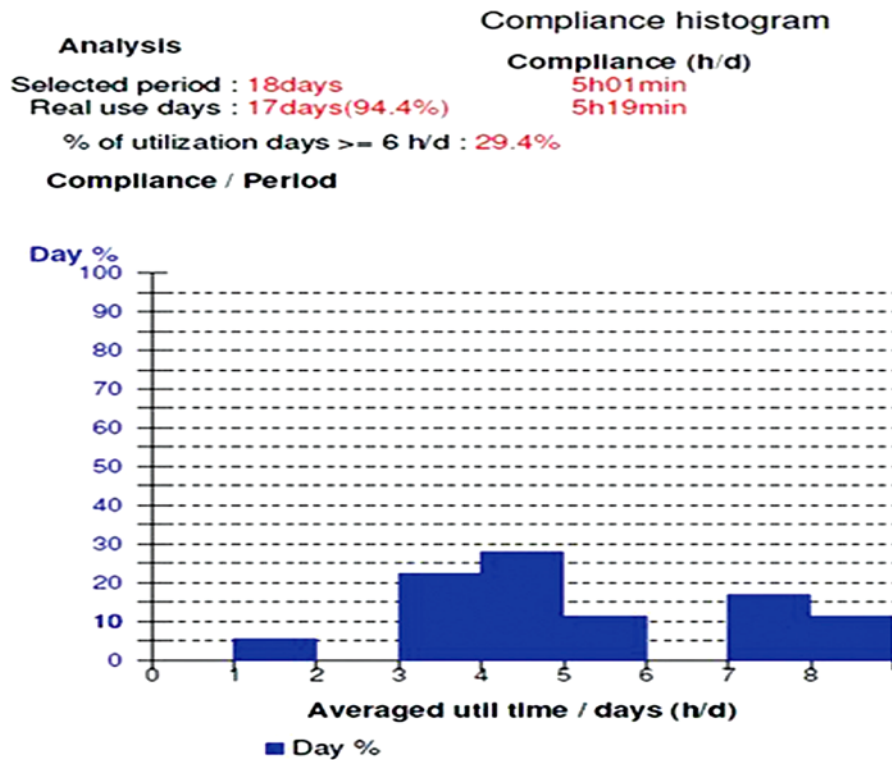


Figure 29. Compliance histogram, showing distribution of compliance based on averaged utilisation time per night (Nosedá *et al.*, 2000).

CPAP Compliance Report can be done by checking the nightly usage hours and checking leak data which is usually shown as Liter/second. Values significantly greater than 0.4 Liter/second are an indication that the patient is using an inappropriate or poorly-fitting CPAP mask. Also, apnea events must be reviewed. Apnea events indicates the number of times the patient has stopped breathing and is shown as events per hour. These values should be at or near zero if the patient is receiving sufficient airway pressure; multiple apnea events per hour are an indication that the patient's CPAP pressure needs to be adjusted (Nosedá *et al.*, 2000).

Compliance report		
Analysis period		
Start date : 04/03/2011	Period : 18days	
End date : 21/03/2011	Total util time : 90h24min	
days		
	Utilization	No utilization
Number of days :	17	1
Average of days / week :	5.7	0.3
% average / Period :	94.4%	5.6%
Util time		
	Utilization	Total period
Compliance (h/d) :	5h19min	5h01min
Min compliance (h/d) :	1h43min	0h00min
Max compliance (h/d) :	9h00min	9h00min
Sessions and ramps		
Average of sessions / use days : 1.06		
Average of ramps / use days : 0.18		
Abstract		
Compliance (h/d.Period) : 5h01min		
real utilization days (d/w) : 5.7 (94.4%)		
% of utilization days >= 6 h/d : 29.4%		

Figure 30. Detailed compliance report(Noseda et al., 2000).

The discrepancy between mean use assessed from the time counter and mean effective use measured with a pressure monitor may vary between 0.4 and 0.5 hour, which is 89% of the run time (Reeves-Hoche et al., 1994). Without knowing actual sleep time, which may vary considerably between patients, studies of mask pressure may still not provide fully accurate data on true effective compliance (Engleman et al., 1994).

- **Modern technology**

Modern technology has provided a variety of tools (compliance meters and data-card tracking devices) and options that may measure, assist, track and enhance compliance, and expedite the collection and analysis of these data (Bazzani, 2007).

Current CPAP devices can be interfaced with a computer in the office or home setting to download data. Newer modems can interface with the CPAP unit and the integrated chip to facilitate the reporting of remote data and reduce the need for face-to-face visits (Lankford, 2004).

These may be used as augmentation to therapy for a compliant patient or to identify and assist a non-compliant patient. Some patients feel that they are using the device each night, while upon reviewing the compliance meter, the usage is recorded as poor. This could be related to unknowingly removing the CPAP mask early in the night, while believing it is near morning when this happens. Other patients believe that they have not been using their device as much as expected, while the compliance meter confirms adequate use. In such cases, the patient can be reassured about their accomplishments and praised for their efforts (*Bazzani, 2007*).

Also, smart cards can be inserted into slots in the CPAP units to imprint the data (figure 31). These cards are then transported to the appropriate professional in a variety of ways, including the use of mail, carriers or other modes, to reduce the need for direct patient travel for regular compliance reporting. Two popular devices are the ResScan TM Data Card (ResMed, Bella Vista, NSW, Australia) and Respironics Encore_ Pro Smart Card (Philips, Murryville, PA, USA) (*Lankford, 2004*).



Figure 31. Memory card for recording compliance data(*Lankford, 2004*).

Data cards fits into CPAP machine to collect sleep data. These cards can be taken out and read by a card reader that hooks up to

computer. Software is still necessary for data to be downloaded (*Galetke et al., 2008*).



Figure 32. Data card used to download tracking information from CPAP for assessment of adherence to it (*Galetke et al., 2008*).

These cards track usage hours, pressure, mask leak, snoring and apnea–hypopnea index (AHI) on selected machines, and may assist sleep technicians, physicians and home care providers by providing feedback on efficacy data to determine effective therapy and to measure outcomes. If enabled, these cards allow patients to monitor their own treatment, in terms of usage time, set pressures and mask leaks (*Bazzani, 2007*).

AHI measurements by some machines have been shown to be highly correlated with the measures recorded by polysomnography (*Gugger, 1997*).

A further innovative development in the collection and reporting of compliance data is the use of wireless networking and internet technologies or modem to transmit clinical data to a remote site. Provider-specific reports can then be generated, including graphic displays. Such advances in data collection and transmission should enable the provider to routinely monitor CPAP use and to intervene when use is suboptimal (*Lankford, 2004*).

Download cables are used to connect CPAP machine to computer so that sleep metrics may be downloaded. Software is needed for reviewing this data. Card reader may be used instead if moving machine close to computer is not wanted (*Galetke et al., 2008*).



reader Download cable

Figure 33. Download cables and reader used for assessment of adherence to CPAP (*Galetke et al., 2008*).

USB Smart Stick Memory Card is designed for use with all Fisher & Paykel Sleep Style 242 (HC242), Sleep Style 244 (HC244) and Sleep Style 254 (HC254) CPAP Machines. The Smart Stick works as a miniature USB drive capable of transferring therapy data from a Smart Stick enabled Sleep Style CPAP to a computer without the need for a separate card reader. To review data on the card software Performance Maximizer Software is required (*Galetke et al., 2008*).



Figure 34. USB smart stick memory card used for transferring sleep data from CPAP to computer (*Galetke et al., 2008*).

II.b. Frequency of routine monitoring of CPAP compliance

Compliance should be checked approximately at 7, 30, 60 days and approximately 12 months after the initial CPAP setup. The patient should be seen by a qualified sleep professional in order to assess usage of the equipment (hours of use and hours of application), check the machine settings and ensure the interface (mask, pillows, etc.) is in good condition. Immediate and long term follow-up are indeed crucial to compliance, but monitoring efficacy is also critical to compliance and successful therapy (*Richards et al., 2007*).

The results of the time/usage monitor should be discussed and the need to be faithful to the prescribed treatment reinforced. An annual office visit should be scheduled to check all the equipment and the time/usage. Masks wear out and break, so an annual replacement should be included in the care plan. Changes in the patient's condition may warrant a change in CPAP pressure (i.e. weight loss may allow for a lower CPAP setting or vice versa) (*Rose, 2006*).

Sin et al., 2002 studied compliance monitoring, including consistent follow-up, “troubleshooting” and regular feedback to both

patients and physicians, achieved CPAP compliance rates >85% over 6 months. There was a decrease in ESS score of 44% by 2 weeks of therapy and the patients continued to improve over the follow-up period, with the lowest mean ESS score observed at 6 months.

Tele-monitoring

Telemedicine involves the provision or support of direct clinical care via the application of electronic and communications technology, including the remote monitoring of health status. By providing patient data early in the course of CPAP prescription, it is believed that this technology would be immensely useful in improving compliance and acceptance of the device in patients with sleep apnea (*Smith et al., 2002*).

Telemedicine allows tailored management of OSA CPAP patients through monitoring of “time at prescribed pressure” and transmission of those CPAP compliance- and efficacy-relevant data to care providers in 24 hour cycles. All that is needed is a personal computer, an Internet connection and the proprietary software (figure 35) (*Sparrow et al., 2010*).



Figure 35. Equipment for continuous positive airway pressure telemonitoring based on modem technology (Encore Anywhere™; Philips Respironics, Murrysville, PA, USA) (*Sparrow et al., 2010*).

Reports can be generated to show usage information and then forwarded electronically to referral labs or physicians without generating additional paperwork. A modem connected to a CPAP unit in the patient's home automatically dials into the server each evening. The compliance server analyses the data and notifies the physician of any patients with low CPAP usage. Patient confidentiality is protected through a username/password-protected system that provides security for the patients' information (*Kwiatkowska and Ayas, 2010*).

Such systems provide an accurate, thorough and advanced method of insuring that each patient is receiving the maximum benefit from their CPAP therapy. Wireless applications have become available, which transmit compliance data every day to a central server, eliminating the need for data cards or home telephone lines and frequent patient visits (figures 36 and 37) (*DeMolles et al., 2004*).

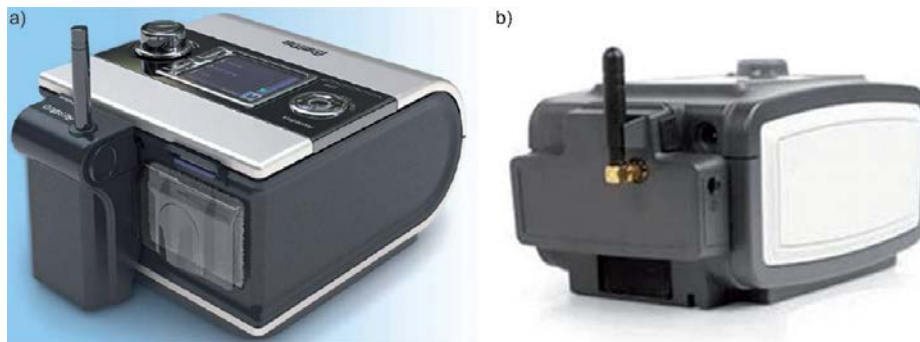


Figure 36. a) Continuous positive airway pressure unit with wireless networking module (ResTraxxTM; ResMed, Bella Vista, NSW, Australia), which transmits daily compliance data to a server, providing timely access to the most up-to-date patient data. b) Respironics EncoreAnywhereTM wireless networking module for (Philips, Murrysville, PA, USA) (*Kwiatkowska and Ayas, 2010*).



Figure 37. Screenshot of software program for wireless monitoring of continuous positive airway pressure compliance. Visual colour-coding allows easy identification of patients that require attention. Remote settings changes are available to fine-tune therapy (*Kwiatkowska and Ayas, 2010*).

One's own patients can be checked at a glance with physician summary reports. Visual colour coding allows the easy identification of patients that require attention. Remote settings changes are available to fine tune therapy and streamline patient management. Historical data can be requested at any time to retrieve data that was not transmitted via the monitoring schedule. As a provider-extender, telemedicine support for patients initiating use of CPAP may allow for greater practice efficiency while maintaining quality of care. Better CPAP compliance and clinical efficacy can be achieved by combining and integrating the most promising elements of both psycho-educational and technological innovations. Study results suggest that use of telemonitored CPAP compliance and efficacy data appears to be as good as usual care in its effect on compliance rates and outcomes in new CPAP users (*Stepnowsky et al., 2007*).

De Molles et al., 2004 used a daily computer-based telephone system to monitor patients' self-reported compliance behavior and

provide automated counseling through a structured dialogue. The impact of the intervention was not significant in comparison with usual care; however, the findings suggested that concurrent education and reinforcement during the initial and early treatment periods are effective countermeasures to patient-reported attenuated compliance.

Taylor et al., 2006 used computers to provide daily Internet-based informational support and feedback for problems experienced with CPAP use. Questions related to CPAP use, hours of sleep and quality of sleep were sent to patients via a computer. The patient's responses were monitored by the sleep medicine practitioner and the patient telephoned if deemed necessary. They found no significant differences in patient functional status and satisfaction with CPAP between the telemedicine intervention and usual-care groups at 30 days. This approach only provided self-reported data to the healthcare provider, and objective compliance and detailed physiological information may have been more useful in effectively troubleshooting problems and may have improved CPAP compliance.

Stepnowsky, et al., 2007 demonstrated that a telemonitored clinical care group had a compliance rate of 4.1 hour/night after the 2-month period, which represents a 46% increase in compliance over the mean compliance level of the usual clinical care group (2.8 hour/night). There were some concerns over the potential for data loss via wireless transmission. However, the loss was negligible and once the wireless unit is properly attached, data from previous nights that are stored in the flow generator device can be re-transmitted and obtained wirelessly.

Sparrow et al., 2010 applied an automated telemedicine intervention system, based on a theory-driven interactive voice response system designed to improve CPAP adherence. The system monitors patients' self-reported behaviour and CPAP-related symptoms, and provides feedback and counselling through a structured dialogue to enhance motivation to use CPAP. The monitor used digitized human speech to speak to the patients and the patients communicate via the touch-tone keypad of their telephones. Each call began with an assessment of the self-reported frequency and duration of CPAP usage during the previous week, followed by one of several motivational counseling modules. If the participant reported excessive side-effects or OSA syndrome symptoms, the system then recommended that the patient contact their physician to discuss the problems. The computer system called the participants if they did not place a call at the expected times. Routine printed reports were sent to the participants' physician biweekly during the first month and every month thereafter. It was found that this telemedicine intervention resulted in a median CPAP usage that was 0.9 hour/night (at 6 months) and 2.0 hour/night (at 12 months) higher than that of an attention control group.

Adherence tracking systems can collect data that measures the date ranges of CPAP usage and the total number of nights the CPAP was utilized (and not utilized); the systems also can manipulate and further sort the data to present the percent of nights CPAP was utilized, percent of nights CPAP was used > 4 hours/night, in general, the CPAP adherence-tracking systems are accurate in objectively determining CPAP use (*Taylor et al., 2006*).

CPAP tracking systems provide averaged data (over many nights, so these data may not reflect the last week or month) for the residual AHI while using CPAP. Currently, CPAP devices use a reduction in airflow (measured with a pneumotachograph) to estimate the residual AHI. In contrast, during polysomnography, apnea or hypopnea determination is based on more robust data, including respiratory flow patterns (nasal pressure and a thermistor), EEG arousal, thoracoabdominal effort, and oxyhemoglobin desaturations. Thus, residual AHI measured from a CPAP download is not a true surrogate of the AHI measured during a sleep study. Caution therefore must be used in interpreting OSA resolution or persistence from CPAP adherence data reports (*Stepnowsky, et al, 2007*).

Event detection data should be used in the management of OSA patients if the data are at either end of the spectrum [normal AHI (< 5 events/hour) or very high AHI (> 30 events/hour)] (*De Molles et al., 2004*).

Intermediate residual AHI data can be difficult to interpret and should be examined within the clinical context of the patient. Reduction in CPAP mask leak can improve adherence and improved adherence can improve OSA outcomes, Mask leaks depend on both the mask (nasal pillows, or full face) and the pressure being delivered. There may be no leak threshold that is clinically acceptable as even a small leak directed into a patient's eyes can be a problem(*Sparrow et al., 2010*).

Mask leak data are averaged measurements and may not reflect recent changes in the CPAP interface. Mask leak may be secondary to leaking through the mouth or around the mask. If the CPAP unit is

running when a patient goes to the bathroom, this may appear as large leak in the download even though there is not a true mask leak. Leak data, like event detection data, must be examined within the clinical context of a patient; extreme measurements on the spectrum are more likely to be valid than middle of the road numbers. If the patient's mask leak is significantly greater than the leak threshold specified by the specific CPAP manufacturer, the interface could be changed (*Kwiatkowska and Ayas, 2010*).

The new CPAP adherence tracking devices measure many other respiratory signals data, including periodic breathing (Cheyne-Stokes pattern), vibratory snoring, flow limitation, clear airway apnea (central sleep apnea). Unfortunately there are essentially no examining for the validity, reliability, reproducibility, or utility of these signals(*Taylor et al., 2006*).

Optimization of Adherence

The ability to identify determinants of adherence is essential for improving patient outcome. Studies have focused on several variables. For example, there is no evidence that positive pressure modalities influence adherence to CPAP as there is no evidence of improved compliance with bi-level devices. In contrast, patients receiving 'intensive support' (included the standard support, with CPAP education provided in the participants' homes with partners, 2 additional nights of CPAP titration in the sleep center for CPAP troubleshooting during initial CPAP exposure, and home visits by sleep nurses after 7, 14, and 28 days as well as after 4 months) have a higher likelihood of CPAP usage than standard support patients do (based on their usual care for newly diagnosed OSA patients and included verbal explanation for CPAP

treatment, a 20- min educational video, a 20-min acclimatization to CPAP during waking hours, one-night CPAP titration in the laboratory, and telephone follow up on days 7 and 21 followed by clinical visits at 1, 3, and 6 months) (*Chervin et al., 2000*).

Likewise, patients who are self-referred have greater CPAP usage than those whose referral is initiated by bed partner. Similarly, patients with subjective complaints of excessive daytime sleepiness, however, show greater hours of CPAP use versus those with minimal daytime complaints (*Chervin et al., 2000*).

Conclusive evidence of improved compliance with auto-CPAP, bi-level, self-titration, and humidification is also lacking. There is some evidence that psychological/educational interventions improve CPAP usage (*Zozula and Rosen, 2001*).

Approach to poorly compliant patients

Poorly compliant patients could be separated in two groups including those with a low percentage of use and those with only short use (*Shivalkar et al., 2006*).

Irregular users of the machine are candidates for some educational intervention or weekly follow-up by phone calls, which have been shown to be effective in improving compliance (*Smith et al., 2006*).

However, subjects who fall asleep every night on CPAP but use it only for a short time, because they cannot tolerate it for a longer period or their sleep time is very short, may require a change to some other CPAP modality, such as auto-CPAP, bilevel CPAP, EPR (ResMed) or C-Flex

(Philips Respironics), or may benefit from some pharmacological therapy to lengthen or stabilise sleep (*Nosedá et al., 2000*).

Optimal CPAP adherence rate to improve health Outcome

Several studies have attempted to define optimal use relative to health outcomes. In a placebo-controlled trial, subjective sleepiness measures, objective sleepiness measures, and energy/fatigue measures demonstrated greater improvement with more CPAP use. The investigators identified that at least 5 hours/night of CPAP treatment at effective pressure was necessary to restore sleepiness to normal levels (*Stradling and Davies, 2000*).

Other studies examined outcomes relative to CPAP use in mild OSA subjects using two different, but relatively low amounts of nightly CPAP use, to define adherence (2.5 and 4 hours), identified that even with low usage levels, improvements in the outcomes of respiratory disturbance, subjective sleepiness, and symptoms improved but more hours of use per night was consistent with greater improvements in these outcomes. A limitation of these studies was the inclusion of all participants, regardless of whether they exhibited abnormal values prior to treatment, in examining the relationship between CPAP adherence and the recovery of normal functioning, thus potentially blunting the treatment effect (*Barnes et al., 2002*).

In a study that examined the effect of adherence to CPAP on recovery of memory in those participants who had abnormal values on a memory test (delayed recall) prior to treatment found after 3 months of treatment that those who had normal values on the delayed recall test

used their devices significantly longer than those who did not (5.21 versus 3.42 hour/night) (*Zimmerman et al., 2006*).

Participants who used CPAP greater than 6 hours/night were 7.9 times more likely to have normal values on the memory task than those who used their CPAP less than 2 hours per night(*Galetke et al., 2008*).

A prospective cohort study of 149 newly diagnosed OSA participants with severe disease were followed for 3 months on treatment to determine the estimated likelihood of returning to normal levels of subjective sleepiness, objective sleepiness, and daily functioning relative to the nightly duration of CPAP use. This study showed that the greatest proportion of participants with abnormal values on these metrics had a positive response to treatment demonstrated by normal values with increased use (*Weaver et al., 2007*).

The greatest gain in improvement in the Epworth Sleepiness Scale to a value less than 11 was with 4 hours use/night; while 6 hours nightly use produced the largest proportion of individuals who had a value greater than 7.5 minutes on the Multiple Sleep Latency Test, and 7.5 hours use resulted in the highest number of participants with normal values on the Functional Outcomes of Sleep Questionnaire, a measure of daily functioning. These reliable relationships were linear for the Epworth Sleepiness Scale score and Multiple Latency Test, but not for the Functional Outcomes of Sleep Questionnaire, principally because there were so few participants who used CPAP beyond 7.5 hours per night to ascertain whether the slope continued in a progressive fashion (*Weaver et al., 2007*).

A study of CPAP dose-response provided new evidence that the amount of CPAP use (*i.e.*, adherence) to produce “normal functioning” is not only related to how long CPAP is applied nightly, but is also dependent on the outcome selected to define normalcy. The question of “how much CPAP use equates to adherence, ” is critically important as empiric studies of CPAP adherence have variably defined adherence (*Weaver et al., 2007*).

When definitions of CPAP adherence outcomes differ across studies, it becomes increasingly difficult to translate the findings of CPAP adherence studies to clinical practice and possibly more important, to understand the effect of CPAP on clinical outcomes of importance (*Smith et al., 2007*).

Factors that influence the complex nature of CPAP Adherence

There is not any single factor that has been identified as consistently predictive of CPAP adherence. They include patient characteristics, disease characteristics, technological factors, initial CPAP exposure factors and psychosocial factors. Yet, the findings from the studies suggest that a multiplicity of factors that are highly variable between individuals, are predictive of CPAP adherence (*Smith et al., 2009*).

Age, sex, marital status, and socio-economic status have been examined as possible predictors of CPAP adherence without consistent findings. Also, race has been examined as a predictor of CPAP adherence (*Budhiraja et al. 2007*).

▪ **Race**

Although only African, American and Caucasian race has been examined, there is some evidence to suggest that African Americans use CPAP for less time, on average, than Caucasians (*Joo et al., 2007*).

▪ **Gender**

Lichuan et al., 2009 found that female gender was significantly associated with non-compliance with CPAP therapy. In contrast, *Sin et al., 2002* described that women, on average, used CPAP more frequently than men. *Ye et al., 2009* observed no gender differences in CPAP adherence.

▪ **Disease-specific characteristics**

Numerous studies have examined disease-specific characteristics that may predict subsequent CPAP adherence. Disease severity, as measured by AHI and nocturnal hypoxaemia has been shown to have a weak predictive relationship with CPAP adherence, yet these findings have not been consistent. Daytime sleepiness has been associated with CPAP adherence in some studies (*Janson et al., 2000*).

However, post-treatment perception of somatic benefit has a stronger relationships with CPAP use, but has limited usefulness in the identification of who will likely be non adherent prior to the initiation of therapy (*Engleman, Wild, 2003*).

▪ **Technological advancements**

Since the first description of CPAP by *Sullivan et al., 1981* there have been many technological advancements in the delivery of positive airway pressure. Many of these technology improvements have evolved as a result of patients' difficulties using and adhering to the treatment,

although the impact of these improvements on improving adherence remains unclear. Approximately two thirds of patients will experience side effects from CPAP such as skin irritation, nasal stuffiness, eye puffiness, or gastric fullness (*Engleman and Wild, 2003*).



ResMed S8 AutoSet™ II Sleep One CPAP

Figure 38. CPAP used for treatment of OSAS(*Galetke et al., 2008*).

The association of the delivery of positive airway pressure with auto-titrating devices with CPAP adherence has also been examined. The particular indication of auto-titrating CPAP in OSAS is its use in patients requiring higher pressure settings. There is no consistent relationship between the use of auto-titrating CPAP and adherence in heterogeneous groups of CPAP-treated OSA persons (*Galetke et al., 2008*).

Pressure relief CPAP was developed to address pressure-related side effects, although these adverse events have not been shown to deter use (*Gay et al., 2006*).

C-Flex is a pressure relief feature that makes breathing back against CPAP pressure at the beginning of exhalation and returning to therapeutic pressure just before inhalation (*Bazzani, 2007*).

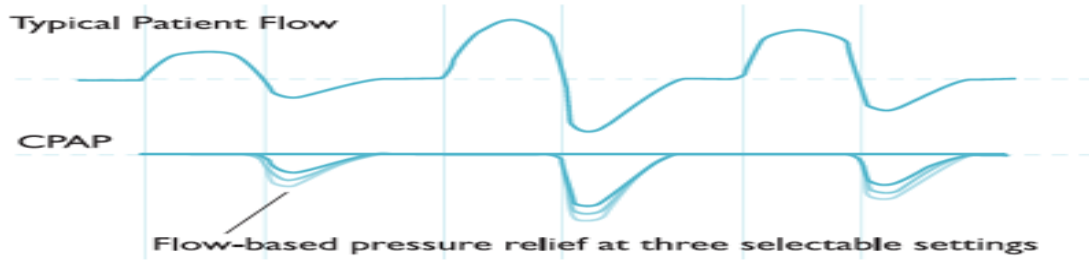


Figure 39. flow based pressure relief by C-Flex (Bazzani, 2007).

Like C-Flex, C-Flex+ reduces the pressure at the beginning of exhalation. Like A-Flex, C-Flex+ softens the pressure transition from inhalation to exhalation to provide additional comfort in fixed CPAP mode (Bazzani, 2007).

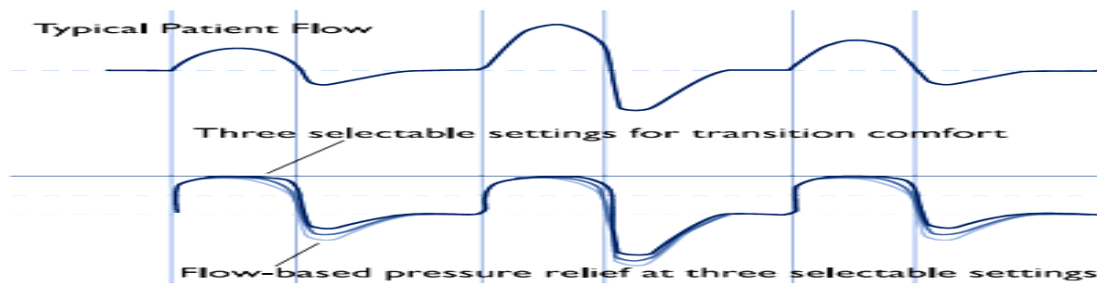


Figure 40. soft pressure transition between inhalation and expiration and flow based pressure relief during early expiration by C-Flex+ (Bazzani, 2007).

Bi-flex delivers pressure relief at three critical points in breathing cycle including the transition from exhalation to inhalation, the transition from inhalation to exhalation and during early exhalation (Bazzani, 2007).

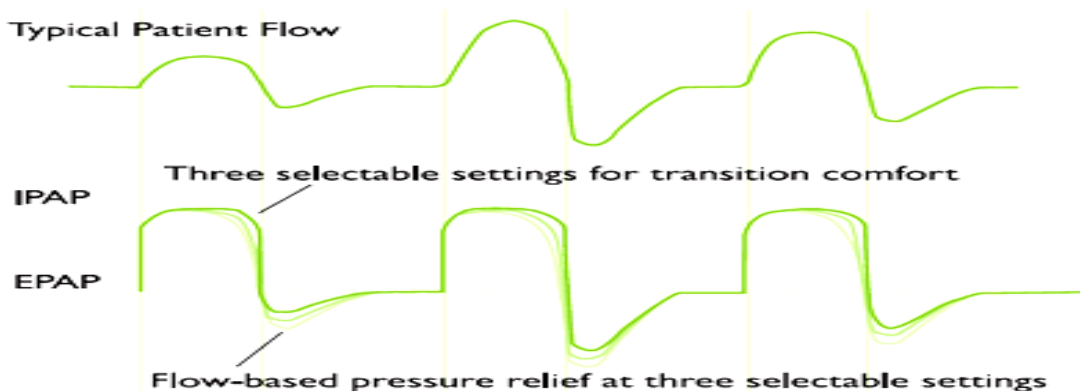


Figure 41. flow based pressure relief at transition from expiration to inspiration, from inspiration to expiration and at beginning of expiration by Bi-Flex (Bazzani, 2007).

Like C-Flex, A-Flex provides flow-based pressure relief at the beginning of exhalation. Like Bi-Flex, A-Flex softens the pressure transition from inhalation to exhalation to provide additional comfort in an auto-CPAP mode (*Bazzani, 2007*).

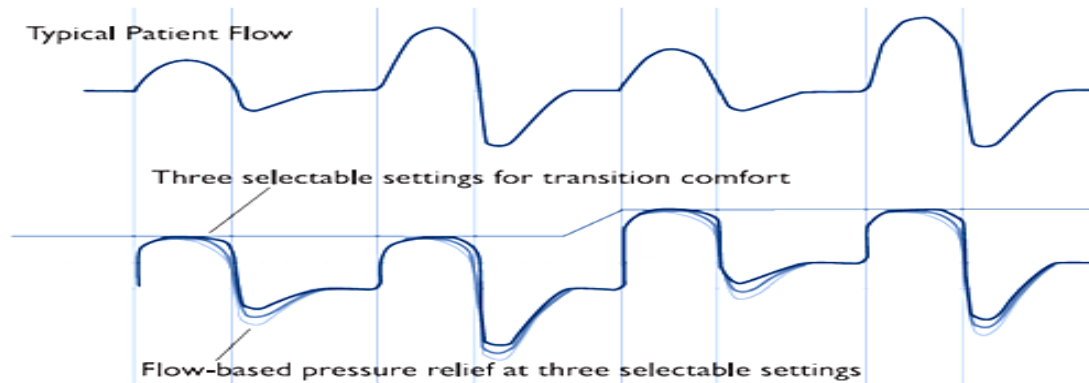


Figure 42. flow based pressure relief at transition from inspiration to expiration and beginning of expiration by A-Flex in auto-CPAP (*Bazzani, 2007*).

The positive effect of pressure relief CPAP has not been clearly established (*Aloia et al., 2005*).

In a small prospective, randomized cross-over study conducted in Germany, there was no difference in CPAP adherence at seven weeks among the pressure relief CPAP group versus conventional CPAP group (*Nilius et al., 2006*).

Similarly, in a larger randomized, controlled trial which included four sites in the U.S. and Germany identified no difference in CPAP adherence outcome among pressure relief CPAP subjects compared with standard CPAP participants at 30, 90, and 180 days (*Dolan et al., 2009*).

Pressure relief CPAP was rated on visual analogue scale as more comfortable than standard CPAP. Data may indicate that this technological feature may be beneficial for only a select group of patients who are adversely affected by pressure (*Marshall et al., 2008*).

▪ **Side effects of CPAP**

Side effects of the treatment have not been shown to be predictive of adherence to CPAP. It has been demonstrated that those who reported mask-side effects were in fact those patients who used CPAP regularly (*Sugiura et al., 2007*).

There have been a few studies that have indicated that nasal resistance affects CPAP use and the initial acceptance of the device (*Nakata et al., 2005*).

Smaller nasal cross-sectional area and reduced volume, measured with acoustic rhinometry, were associated with non adherence. Self-reported nasal stuffiness was not related to nasal dimensions (*Li et al., 2005*).

Surgery, shown to improve tolerance to CPAP, may be warranted for patients presenting with either total nasal resistance of more than 0.38 Pa/cm³ per second, nasal obstruction that does not resolve with medical treatment, nasal septum deviation, or inferior turbinate hypertrophy. Self-reported claustrophobic tendencies, evident in 15 percent of patients, have been associated with more variability in CPAP use and lower overall adherence (*Chasens et al., 2005*).

▪ **The effect of diagnostic procedures and method of CPAP delivery**

Important considerations in understanding factors affecting CPAP are the effect of diagnostic procedures and method of CPAP delivery. Two-night, in-laboratory polysomnogram (*i.e.*, diagnostic followed by CPAP titration) as compared with split-night polysomnogram (*i.e.*, diagnostic and CPAP titration combined in one night study) does not influence overall CPAP adherence rates (*Gay et al., 2006*).

With the introduction of auto-titrating CPAP and unattended diagnostic polysomnography equipment, an empiric study examined how attended polysomnography and CPAP titration versus unattended diagnostic study and initial CPAP exposure in the home affect CPAP adherence. Although there was no difference in the number of nights of use between the groups, patients who underwent attended diagnostic and titration study procedures used their CPAP for more hours per night, on average, than those patients who had unattended studies and no supervised initial CPAP exposure (4.1 vs. 2.9 hour; $P < 0.05$). These differences in CPAP adherence suggest that a supervised, initial exposure to CPAP is an important factor with regard to CPAP use. However, the benefit of more than one-night of supervised CPAP titration has not been shown to further improve CPAP adherence rates (*Kaplan et al., 2007*).

Based on interviews with adherers (continuers of CPAP treatment) and nonadherers (discontinuers of CPAP treatment), it was found that adherence to CPAP (*e.g.*, decision to “continue using CPAP”) was common among users who subjectively experienced initial benefit from the treatment, had positive experiences during the titration, and perceived that they received thorough, necessary information from their provider. In contrast, the investigators suggest that the non adherent group experienced no subjective improvement with CPAP treatment, were less satisfied with the polysomnogram experience, and reported a lack of anticipatory guidance with regard to the polysomnogram experience (*Terri et al., 2010*).

By examining adherence to CPAP after one month, problems identified on the first night of CPAP use was consistent with lower CPAP adherence (*Lewis et al., 2004*).

It has been shown that not only the initial CPAP experience is important to adherence, but also the benefit perceived on the first night of treatment. The evidence suggests that the technological aspects associated with polysomnography and treatment delivery is less important in promoting adherence than a supportive environment and first impressions of ease of use and benefit of therapy (*Drake et al., 2003*).

▪ **Education and cognitive behavioural therapy**

Group education for 2 hours every 6 months improved CPAP use significantly, and a multidisciplinary program based on group education with six workshops related to OSAS, different aspects of CPAP treatment, as well as suitable self-care activities improved excessive daytime sleepiness (*Golay et al., 2006*).

A study using verbal education in small groups improved knowledge about sleep apnea (*Smith et al., 2009*).

Video education showed limited effects on knowledge and adherence, even when written information about OSAS and CPAP, as well as telephone support was added (*Hui et al., 2000*).

On the other hand, a 15 min educational video during the initial visit improved the return rate to a CPAP clinic after 1 month (*Wiese et al., 2005*).

An intervention based on two sessions with cognitive behavioural therapy including educational video resulted in increased adherence to CPAP treatment (*Richards et al., 2007*).

An extensive home-based individual education on four occasions during 3 months did not significantly improve adherence when compared to standard education with verbal information about OSA and the CPAP device. The reason for the conflicting results in these studies might be small sample sizes, lack of power in the educational intervention, or lack of validated and reliable instruments (*Meurice et al., 2007*).

▪ **Psychological and social variables**

There has been increased interest in considering the influences of psychological and social variables on CPAP adherence. Studies of psychological factors have applied a number of health promotion models including Bandura's social cognitive theory (*Bandura, 1977*), Prochaska and DiClementes' transtheoretical model (*Prochaska and DiClemente, 1983*), and Lazarus and Folkman's stress and coping model (*Lazarus and Folkman, 1984*).

Collectively, these studies suggest that psychological correlates of adherence behaviour are important to understand adherence to CPAP and suggest important opportunities for adherence interventions (*Aloia et al., 2005*).

Psychological factors such as depression, anxiety, stress, and social desirability have not been shown to predict CPAP use (*Terri, et al., 2010*).

Yet, how individuals cope with challenging situations (active versus passive) has been shown to be associated with CPAP adherence (*Stepnowsky et al., 2002*).

Patients who experience difficulties and proactively seek solutions to resolve problems (active coping) are more likely to be adherent than those who are less inclined to troubleshoot difficulties with the treatment (passive coping). Whether an individual is motivated internally or externally (locus of control) to engage in healthy behaviours has been examined as a predictor of CPAP adherence. Although there were no pre-treatment differences in degree of internal locus of control, those who discontinued treatment were less externally motivated suggesting that they would be less receptive to admonitions by others to apply the treatment (*Terri, et al., 2010*).

With treatment exposure, perceptions regarding CPAP therapy affect both short- and long-term CPAP adherence. Components of social cognitive theory, risk perception, treatment outcome expectations, and self-efficacy (*i.e.*, belief in own ability to perform the desired behaviour), and tenets of the transtheoretical model have been shown to be significant predictors of CPAP adherence (*Aloia et al., 2005*).

As patients gained experience with CPAP, the strength of the association between these psychological variables and adherence increased, explaining more than 30 percent of the variance (*Stepnowsky et al., 2002*).

When *Tyrrell et al., 2006* employed a semi-structured interview based on the health belief model, they found that those who discontinued treatment after 6 months identified few benefits of using CPAP, could not articulate treatment expectations, indicated there were many drawbacks, and did not view OSA as a health problem.

These statements are consistent with previous research done by *Stepnowsky et al., 2002* utilizing other health promotion models that indicate the critical role of perceptions in acceptance of CPAP treatment

▪ **Social factors**

Social factors have also been shown to influence CPAP adherence, including social support, partner involvement in treatment, and partner sleep quality (*Zoidis, 2008*).

CPAP users who live alone have been found to be significantly less likely to use their CPAP than those who live with someone. Although partner-referred patients are less likely to be adherent to CPAP. Spouse or bed partner sleep disturbance and sleep quality are important to patients' CPAP adherence behaviours. Patients who were more adherent to treatment had spouses or bed partners who had better sleep quality (*McArdle et al., 2001*).

Sleeping with a spouse or partner who may provide feedback regarding the elimination of symptoms such as snoring, may also contribute to higher CPAP adherence. These studies indicate the importance of immediate sources of social support in promoting CPAP use and the contribution of CPAP use to positive outcomes for the bed partner (*Cartwright, 2008*).

Predictors of non adherence to CPAP

Table 10. Potential predictors of non-adherence are listed in the table (*Weaver and Grunstein, 2008*).

Patient related factors	Therapy and medication related factors	Health professional related factors
Failure to understand the importance of the therapy.	Complexity of therapy, in device use, or medication dosing.	Poor relationship with patient.
Failure to understand instructions concerning the therapy.	Increased rate of adverse reactions. (Device use has complications, and the provider needs to meet with the patient periodically to determine adverse events and help address these issues).	Expression of doubt concerning therapeutic potential.
Concomitant self-administration of prescription or non-prescription medications or alcohol.	Characteristics of illness; long-term or chronic illnesses are a problem, as compliance decreases over time.	Unwillingness to educate patients.
Social Isolation, thus lack of social support. (Patients with supportive families have been shown to be more compliant with prescription drugs - data not available for CPAP use).	Expensive therapy (Only a problem when a patient must pay out of pocket or has not met the deductible.)	Lack of knowledge of medications the patient is taking or has access to. (Sedatives and alcohol can compound OSA, and their use should be evaluated).
Feeling ill, or being too tired to use the therapy.	Lack of efficacy.	
Physical limitations, including vision, hearing, hand coordination.		

The following factors have been associated with better long-term adherence with CPAP therapy. They include adherence with CPAP during the first week of therapy (*Rosenthal et al., 2000*), increased self-

reported daytime sleepiness (ie, an Epworth Sleepiness Scale score >10) plus moderate to severe OSA (ie, an apnea hypopnea index ≥ 30 events per hour of sleep) (*Morris et al., 2005*), low nasal resistance (*Sugiura et al., 2007*), CPAP titration via attended polysomnography (*Means et al., 2004*), a nasal pillows interface (*Massie et al., 2003*), certain psychological traits, including lack of claustrophobic tendencies (*Chasens et al., 2005*), presence of problem solving skills (*Stepnowsky et al., 2002*), optimism regarding the benefit of CPAP therapy (*Aloia et al., 2005*), and self-efficacy (defined as a positive subjective assessment of one's motivation, volition, and confidence to engage in a healthy behavior) (*Aloia et al., 2005*), the patient made the decision to seek medical attention (*Weaver et al., 2008*) and greater severity of OSA (a weak relationship) (*Gay et al., 2006*).

Interventions which improve CPAP adherence

There is increasing number of intervention studies aimed at promoting CPAP adherence. These investigations can be categorized as supportive, educational, cognitive behavioural, or mixed strategy based on their reported content, methods, and theoretical framework (*Engleman and Wild, 2003*).

Supportive interventions are described as “reinforcement”, support, and/or enhanced access to sleep-specific, healthcare resources (*Hui et al., 2000*).

Educational interventions focus on enhancing patient knowledge relative to the diagnosis and treatment of OSA (*Hui et al., 2000*).

Cognitive behavioural intervention strategies are explicitly described as such, theoretically-derived, and delivered by expert interventionists.

Finally, mixed strategy describes a combination of support and education (*Hui et al., 2000*).

▪ **Supportive interventions**

The majority of published intervention studies can be categorized as supportive interventions. Early studies reporting supportive interventions aiming to promote CPAP adherence were based on comparison between positive reinforcement and usual care (*Hui et al., 2000*).

The mechanisms of support varied across studies (*i.e.*, phone call, print documents, clinical follow up). However, no differences in CPAP adherence between the experimental and control groups were observed. Several investigators have applied telecommunications methods such as a computerized telephone system or wireless telemonitoring as supportive interventions. Additionally, CPAP-naïve participants received feedback (reinforcement) and supportive information in response to the objective telemonitored pattern of CPAP use (*Stepnowsky et al., 2007*).

In a placebo-controlled study which examined whether CPAP adherence was improved in those with a well-established pattern of non-adherence at 12 week, those exposed to a telecommunications-supported intervention had significantly greater use compared to a control group (*Smith et al., 2007*).

From the intervention studies that are categorized as supportive, simplistic unidirectional (provider to patient) reinforcement of CPAP use is not adequate to improve overall adherence rates to CPAP. However, when combined with real-time assessment of CPAP use (CPAP adherence records as in telecommunications studies) and support for problem-solving or troubleshooting difficulties with CPAP, supportive interventions may be useful in promoting adherence to CPAP. This might be especially applicable to those without existing sources of social support (*i.e.*, spouse, bed partner) and/or those lacking confidence in their own ability to apply the treatment (*Rose et al., 2006*).

▪ **Educational intervention**

Interventions based on education to promote adherence have been examined. Three clinical trials applying three different educational strategies have been published, each of which reported no significant effect on adherence (*Wiese et al., 2005*).

Possibly the most widely recognized CPAP adherence intervention study compared standard support with intensive support. Standard support was based on their usual care for newly diagnosed OSA patients and included verbal explanation for CPAP treatment, a 20-min educational video, a 20-min acclimatization to CPAP during waking hours, one-night CPAP titration in the laboratory, and telephone follow up on days 2 and 21 followed by clinical visits at 1, 3, and 6 months (*Smith et al., 2007*).

Intensive support included the standard support, with CPAP education provided in the participants' homes with partners, 2 additional nights of CPAP titration in the sleep center for CPAP troubleshooting

during initial CPAP exposure, and home visits by sleep nurses after 7, 14, and 28 days as well as after 4 months. The intervention strategy combined support, education, and the concept of self-efficacy promotion through the initial CPAP exposure under supervised conditions (*Smith et al., 2007*).

Although significant improvement in CPAP adherence was identified at 6 months (5.4 ± 0.3 vs. 3.8 ± 0.4 hours/night, intensive versus standard), the applicability of the intervention to clinical practice is limited as the intervention is labour-intensive and time-intensive (*Smith et al., 2009*).

Furthermore, in the current climate of limited sleep healthcare resources this intervention strategy is not cost-effective nor does it promote access to sleep services. Yet, the study does point to the importance of addressing adherence from a multidimensional perspective. The study also highlights the importance of initial exposure to CPAP experiences and social support (*i.e.*, partner or spouse) in patients' decisions to use CPAP and persist with the treatment. Emphasizing the multidimensional nature of adherence to CPAP, a study combined education and supportive techniques in a music and habit forming intervention designed to promote relaxation, CPAP instruction, and habitual application of CPAP (*Smith et al., 2009*).

A randomized controlled trial of newly-diagnosed, CPAP-naive patients assigned to the habit-promoting experimental audio intervention or the placebo “get in habit of daily vitamins in your diet” audio intervention identified more adherers (*i.e.*, > 4hours use/night and at least 9/14 nights) in the experimental group than the placebo group at 1 month

but not at 3 or 6 months. Early patterns of CPAP application and use are important to long-term CPAP adherence. Although this intervention addressed the demands for early habit-formation, relaxation, and positive reinforcement, additional interventions may be necessary to sustain good CPAP habits. This may be particularly true among early persistent CPAP users who experience difficult ties with CPAP (*Aloia et al., 2007*).

A study conducted in France, compared four types of educational interventions including reinforced education by both prescriber and homecare provider; reinforced education by prescriber and standard care by the homecare provider; standard education by prescriber and reinforced education by homecare provider; and standard education by both the prescriber and the homecare provider, which served as the control. Compared to standard education, reinforced educational interventions were delivered with increased frequency (reinforced education) with expanded explanation and demonstration. CPAP adherence was measured at 3, 6, and 12 months without statistically significant differences between intervention groups compared to the control group. The overall, average adherence for all groups at three and six months was 5.6 and 5.8 hours/night at twelve months (*Meurice et al., 2007*).

In a smaller study of 35 severe OSA subjects, a newly developed interdisciplinary, educational intervention for CPAP users was tested. Applying a variety of educational strategies (*i.e.*, video, demonstration, discussion), some of which were based on the Health Belief Model, subjects and their spouses participated in a one-day program followed by a single-night of in-hospital CPAP exposure. After 1 year of use, on

average, baseline adherence was 4.4 ± 0.3 hours/night (*Golay et al., 2006*).

Following participation in the educational program, CPAP adherence, measured 3 months after intervention, was 5.1 ± 0.4 hours/night. There was no reported measure of knowledge before or after the educational intervention. The educational intervention was extensive, theoretically-based, and labour-intensive. In this pilot study, likely underpowered to detect differences in adherence to CPAP, there was a trend toward higher CPAP adherence after the intervention. The cost-effectiveness of the intervention, however, must be addressed, as the utility of this intervention may be limited by personnel, time, and patient burden costs (*Golay et al., 2006*).

The inclusion of relatively few nonadherers, indicated by the high level of adherence, may have contributed to the absence of an intervention effect. It is also not known whether the educational intervention enhanced subjects' knowledge of their diagnosis and treatment as no direct measure of knowledge was reported (*Golay et al., 2006*).

A more simplistic education intervention, a 15-minute video program, included content addressing the definition of OSA, symptoms of OSA, information about the device, the sensation of wearing CPAP, and benefits of using CPAP. After randomization, the experimental group was exposed to the video education intervention after their initial clinical visit with a sleep provider and the control group completed the initial clinical visit and a set of questionnaires. The sample had relatively mild OSA (AHI for experimental group 9.6 events/hour, 8.9 events/hour for

the control group). CPAP use, measured as machine-on time, for participants who returned for a 4-week follow up visit, was reportedly not associated with treatment effect. Rate of follow up, however, was associated with video education, with 72.9 percent of experimental group versus 48.9 percent of control group returning for follow up. The simple video education program tested in this study may reduce attrition at clinical follow up, yet it is not clear that CPAP adherence improves with this educational strategy (*Wiese et al., 2005*).

Collectively, educational interventions alone do not influence future use of CPAP among OSA patients. From this group of studies, it is not clear that the educational interventions influenced the mediating variable of interest, knowledge, as none of the studies measured this variable. Instead, the studies examined the outcome of CPAP adherence, or return to clinic, as a surrogate outcome, with the underlying assumption that CPAP adherence is amenable to influence through the process of knowledge acquisition. Knowledge is a pre-condition for health behaviour or change in health behaviour; yet, knowledge alone is unlikely to be a sufficient influence for exacting healthful behaviours (*Bandura, 2004*).

▪ **Cognitive behavioural variables**

Over the past several years, several prediction studies have examined cognitive behavioural variables as predictors of CPAP adherence (*Stepnowsky et al., 2002*).

The intervention studies provide some consistency with regard to influencing actual acceptance and persistence with CPAP treatment (*Wild et al., 2004*).

The earliest study to examine cognitive behavioural intervention was a randomized clinical pilot trial in older adults with OSA, naïve to CPAP. The intervention group received 2-45 minute sessions, one-on-one, that provided participant-specific information about OSA, symptoms, performance on cognitive tests, treatment relevance, goal development, symptom change with CPAP, troubleshooting advice, treatment expectations, and treatment goal refinement. The investigators suggested that providing individualized education and information influences self-efficacy and decisional balance and thereby enhances CPAP adherence (*Aloia et al., 2001*).

The control group received a placebo intervention consisting of 2-45 minute sessions of general information about sleep, sleep architecture, and patient opinions regarding the sleep clinic experience. No difference in CPAP use was observed at 1 and 4 week. However, at 12 week, the experimental group used CPAP for 3.2 hours more than the control group with a large effect size. Although the investigators did not measure the cognitive behavioural constructs of interest (*i.e.*, self-efficacy, decisional balance), this small pilot study suggests that an intervention based on cognitive behavioural constructs potentially influences CPAP adherence behaviours over time (*Aloia et al., 2001*).

In a larger, randomized controlled trial, the same intervention strategy was applied focusing on education to promote self-efficacy and decisional balance compared with motivational enhancement therapy and standard care. Interventions were delivered after one week of CPAP use. Both motivational enhancement therapy and education groups had lower discontinuation rates over the 13 week protocol than the standard of care group. Together with the investigators' earlier work, these cognitive

behavioural interventions may influence the overall risk of very poor adherence (*i.e.*, < 1 hour/night) and abandonment of the treatment all together (*Aloia et al., 2007*).

Acceptance or “uptake” of CPAP treatment was greater among a group who received two 1-hour cognitive behavioural therapy sessions at baseline (*i.e.* prior to CPAP titration in the sleep center) compared with usual care in this large randomized study of moderately severe OSA subjects. The intervention group also exhibited higher CPAP adherence both at 1 week and at 1 month than the control group (5.90 versus. 2.97 hours/night, 5.38 versus. 2.51 hours/night, respectively). The study also demonstrated that the specific cognitive behavioural variables of interest, self-efficacy and social support, but not outcome expectations, also differed robustly between the groups, suggesting that adherence to CPAP increased as a result of the cognitive behavioural intervention (*Richards et al., 2007*).

▪ **Sedatives-hypnotics**

The observation that adherence with CPAP during the first week of therapy predicts long-term adherence led to the hypothesis that early treatment with a sedative-hypnotic may improve long-term adherence with CPAP therapy (*Rosenthal et al., 2000*).

This was tested in a trial that randomly assigned 160 patients with newly diagnosed OSA (mean AHI 37 events per hour) to receive eszopiclone (3 mg) or placebo during the initial 14 nights of CPAP therapy. After six months of follow-up, the eszopiclone group used their CPAP device for more nights (64 versus 45 percent of nights) and for a longer duration (4 versus 3 hours) than the placebo group. The

eszopiclone group was also less likely to discontinue CPAP therapy (42 versus 58 percent) (*Lettieri et al., 2009*).

▪ **Mixed strategy**

Although not explicitly described as such, intervention studies that are categorized as mixed strategy incorporate more than one intervention (composite intervention or multidimensional intervention) to affect CPAP adherence rates. Interventions to promote adherence likely need to address the complex nature of this behavioural outcome, consistent with the belief that behaviours are multidimensional and contextually dependent (*Hoy et al., 1999*).

Multidisciplinary approach to managing CPAP-related problems

Pre-CPAP interventional level

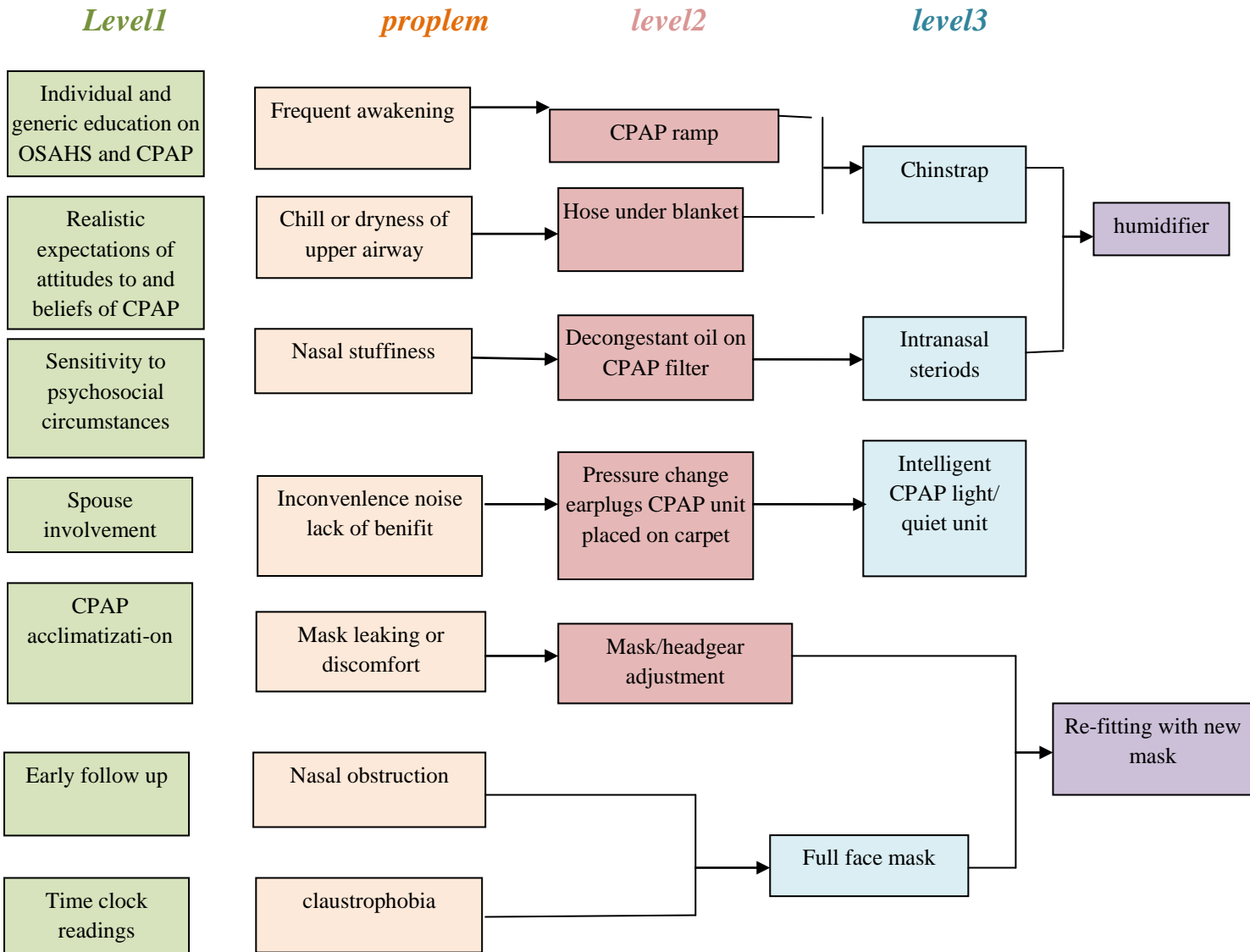


Figure 43. approach to CPAP-related problems (*Engleman et al., 2003*).

Cognitive and performance impairments in OSA

Cognition is the ability to know and think using memory, logic reasoning and all of the higher cortical function. It allows people to appreciate their inner and outer worlds and to interact with others (*Blomberg, 2011*).

OSA patients complain of various neuropsychiatric symptoms. Cognitive impairment and affective disorders such as depression are frequently encountered in OSA (*Kezirian et al., 2007*). In addition, a high prevalence of other psychiatric symptoms such as anxiety, somatization, Attention Deficit Hyperactivity Disorder (ADHD-type), and obsessive-compulsive symptoms have been reported in these patients (*Borak et al., 1996*) (*Yue et al., 2003*). Nocturnal panic attacks, diverse parasomnias, delirium, psychosis, personality change, and violent outbursts can be also reported in some OSA patients (*Sharafkhaneh et al., 2005*).

Considerable research has examined neuropsychological deficits associated with OSA. A meta-analysis of 25 studies (involving >2000 patients) was conducted to examine patterns of neuropsychological deficits in OSA. They found impairments in vigilance (sustained attention), executive function (a set of higher cognitive abilities that control and regulate other basic abilities like attention, memory, and motor skills), and motor coordination but no effect of OSA on general intelligence and verbal ability. The effects of OSA on visual and motor skill and memory functioning were inconsistent (*Beebe et al., 2003*).

On the other hand, *Aloia et al., 2004* reported that 60% of reviewed studies found deficits in attention and vigilance, executive functioning, and memory impairment, and 80% of reviewed studies found visuo-construction and psychomotor functioning impairments.

▪ **Sustained attention or vigilance**

Vigilance is defined as the process of paying close and continuous attention; wakefulness, watchfulness. Sustained attention is one of the most commonly affected cognitive problems for OSA patients (*Feuerstein et al., 1997*). It is assessed by tests such as the Conner's Continuous Performance Test, Psychomotor vigilance task (PVT), Walter Reed palm-held psychomotor vigilance test, reaction time and Oxford university test. Conner's Continuous Performance Test is used to test vigilance in all subjects before and after 12 weeks after of CPAP use. In this test letters are flashed on a computer screen in rapid succession, Subjects are asked to press a response key when they see the letter X, but only when it is preceded by the letter A. This AX condition is thought to maximize the cognitive load of vigilance over and above that of simple reaction time. The test lasts about 12 minutes, and provides measures of accuracy and speed of target detection. Dependent measures include the total number of hits, average reaction time to targets, d' (a measure of signal sensitivity), and the total number of target omissions (*Chugh and Dinges, 2002*).

OSA patients initially perform comparably to normal controls during short-duration tasks, but performance degrades with longer duration tasks, where one sees increased response time, lapses or failure to respond, and false responses (*Weaver, 2001*). Operating a motor vehicle requires sustained attention, and impairment in this cognitive

function may contribute to elevated rate of car accidents. OSA patients with excessive daytime sleepiness are 6 to 10 times more likely to have an accident than non-sleepy controls (*George, 2003*).

The reaction time is defined as the lapse of time between stimulation and the beginning of response. It is done by clicking the large button on the right to begin and waiting for the stoplight to turn green. When the stoplight turns green, the large button is clicked quickly then the large button is clicked again to continue. The stoplight may take up to seven seconds to change. The amount of time is random. Any key may be used instead of clicking the mouse button. The test is repeated five times, and the average reaction time is calculated (*Redline et al., 1997*).

The reaction time of patients with mild to moderate sleep disordered breathing is worse than that found in healthy, non-sleepy subjects with blood alcohol levels of 0.080 g/dL (the typical legal limit for intoxicated driving in the USA) (*Powell et al., 1999*).

The Psychomotor Vigilance Task is a sustained-attention, reaction-timed task that measures the speed with which subjects respond to a visual stimulus. It indicates increased sleep deficit correlates with deteriorated alertness, slower problem-solving, declined psycho-motor skills, and increased rate of false responding. The PVT was championed by David. Dinges and popularized by its ease of scoring, simple metrics, and convergent validity (*Dinges, Powell, 1985*). However, it was shown that motivation can counteract the detrimental effects of sleep loss for up to 36 hours (*Loh et al., 2011*).

The PVT is a simple task where the subject presses a button as soon as the light appears. The light will turn on randomly every few

seconds for 5–10 minutes. The main measurement of this task is not to assess the reaction time, but to see how many times the button is not pressed when the light is on. The purpose of the PVT is to measure sustained attention, and give a numerical measure of sleepiness by counting the number of lapses in attention of the tested subject (*Walker, 2009*).

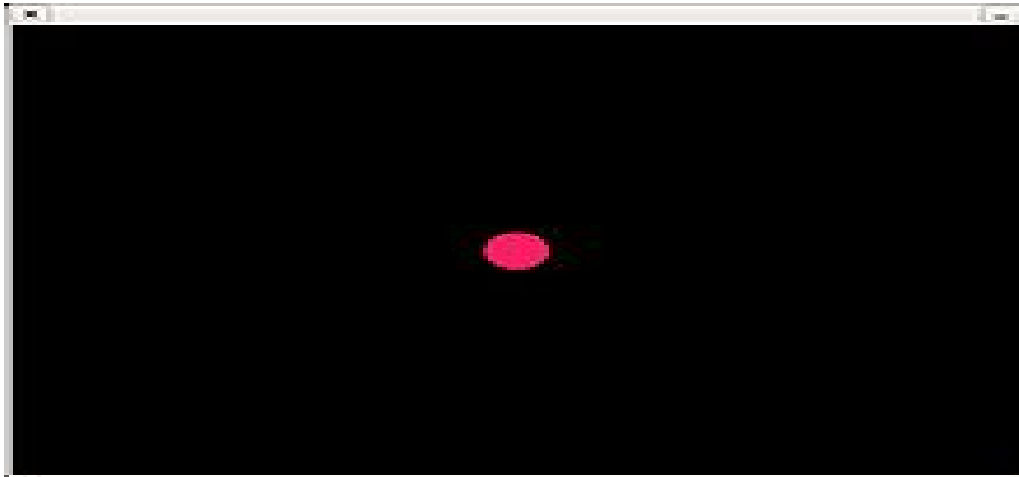


Figure 44. Screen shot of Perceptual Vigilance Test (*Walker, 2009*).



Figure 45. Psychomotor vigilance testing of professional drivers in the occupational health clinic (*Wichniak, et al., 2011*).

The Walter Reed palm-held psychomotor vigilance test is a field-portable reaction time test and analysis software run on devices using the Palm operating system. It is designed to emulate a test and commercial device widely used in sleep deprivation, shift work, fatigue, and stimulant

drug research but provides additional capabilities. Experimental comparisons with the standard commercial device in a 40-hour total sleep deprivation study show it to be comparably sensitive to selected experimental variables (*Rollnick et al., 2008*).

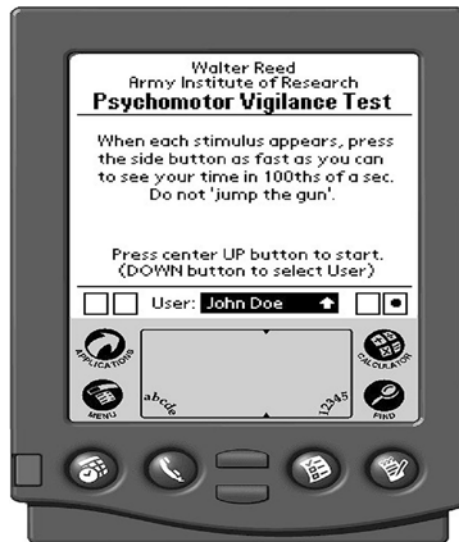


Figure 46. Walter Reed Psychomotor vigilance test (*Rollnick et al., 2008*).

▪ **Memory**

It should be noted that among OSA patients, only 30% of cases present memory deficits in an initial assessment (*Kloepfer et al., 2009*). There is less consistency in studies of short-term and long-term memory functioning in OSA patients (*Engleman et al., 2000*). Diminished performance was described on short-term memory in patients with moderate and severe sleep apnea, although only the severe group demonstrated evidence of impairment in delayed recall. These memory disturbances were associated with a decrease in vigilance (*Rouleau et al., 2002*). Others have reported short- and long-term memory problems (*Salorio et al., 2002*). On the other hand, other study found no differences between patients with OSA and controls on subscales of the Wechsler memory scale (in either immediate or delayed conditions) (*Beebe, et al., 2003*).

Differences in samples such as level of disease severity, clinic versus population-based studies, or the use of normal controls versus norm-referenced comparisons may account for the inconsistencies. These inconsistencies may also reflect impairment in organization and retrieval caused by different levels of executive function deficits in OSA patients (*Beebe et al., 2003*).

▪ **Executive functions**

Executive function refers to a set of higher cognitive abilities that control and regulate other basic abilities like attention, memory, and motor skills. Executive functions may involve the ability to engage in goal directed behaviors and abstract thinking. Executive function impairments can be measured by assessing diverse domains such as planning, sequencing, self monitoring, set-shifting, verbal fluency, abstract reasoning, working memory, visual–spatial organization, and memory (*Goldberg, 2001*).

Many studies have examined disturbances in executive function in OSA, demonstrating impairment in several domains. For example, widespread deficits were reported in various executive functions (verbal fluency, planning, sequential thinking, and constructional ability), with extent of impairment related to severity of the breathing abnormality (*Sateia, 2003*).

Etiology and mechanism of cognitive and performance impairments

The pathogenesis of cognitive deficits in OSA is controversial and most likely multifactorial. The two most commonly implicated etiological mechanisms are repetitive sleep fragmentation and nocturnal hypoxemia.

However, the evidence is as yet tenuous linking either measure of OSA severity and any cognitive domain (*Aloia et al., 2004*).

Researchers have assumed that neuropsychological tests reflect abnormalities in specific brain regions. The evidence of disturbance in executive functions has led to the suggestion that OSA may be associated with frontal lobe dysfunction (*Aloia et al., 2004*) (*Feuerstein et al., 1997*).

The prefrontal model has been proposed as a conceptual framework for the relationship between sleep disruption and nocturnal hypoxemia and primarily frontal deficits in OSA. This model hypothesizes that OSA-related sleep disruption and intermittent hypoxemia as well as hypercapnia alter the brain's restorative processes, thereby inducing a variety of cellular and biochemical stresses that disrupt functional homeostasis as well as neuronal and glial viability within the prefrontal cortex (Figure 47) (*Beebe, Gozal, 2002*).

In this model, OSA related sleep disruption and intermittent hypoxemia and hypercapnia alter the efficacy of restorative processes occurring during sleep, and disrupt the functional homeostasis and neuronal and glial viability within particular brain regions. Subsequent dysfunction of prefrontal cortical regions is manifested by dysfunction of the cognitive "executive system," which alters cognitive abilities, thereby resulting in maladaptive daytime behaviors (*Beebe and Gozal, 2002*).

Volumetric studies in OSA patients also show diminished gray matter in the hippocampus, nearby cerebral cortex, and cerebellar cortex and deep nuclei (*Macey et al., 2002*). The cerebellar damage in OSA may contribute to loss of coordination of upper airway muscle activity

(hypotonia of upper airway muscles), failure to regulate sympathetic tone, and further disruption of higher-order cognitive processes (*Row et al., 2002*).

Functional neuro-imaging studies have begun to investigate the cerebral substrates of cognitive function in OSA. In an functional magnetic resonance imaging (fMRI) study, it was reported that untreated OSA patients showed reduced performance on a back working memory task, as well as reduced activation within anterior cingulate, dorsolateral prefrontal, and posterior parietal cortices. It was suggested that the fragmented sleep contributed to these deficits more than the nocturnal hypoxia(*Rollnick et al., 2008*).

Chai et al., 2006 examined the cerebral response to a verbal learning task in OSA patients by fMRI and found that verbal learning performance was similar in both the OSA and control groups, but OSA patients showed increased brain activation in several brain regions (bilateral inferior frontal and middle frontal gyri, cingulate gyrus, areas at the junction of the inferior parietal and superior temporal lobes, thalamus, and cerebellum). The recruitment of additional brain areas during tasks in OSA patients was felt to reflect an adaptive compensatory recruitment response.

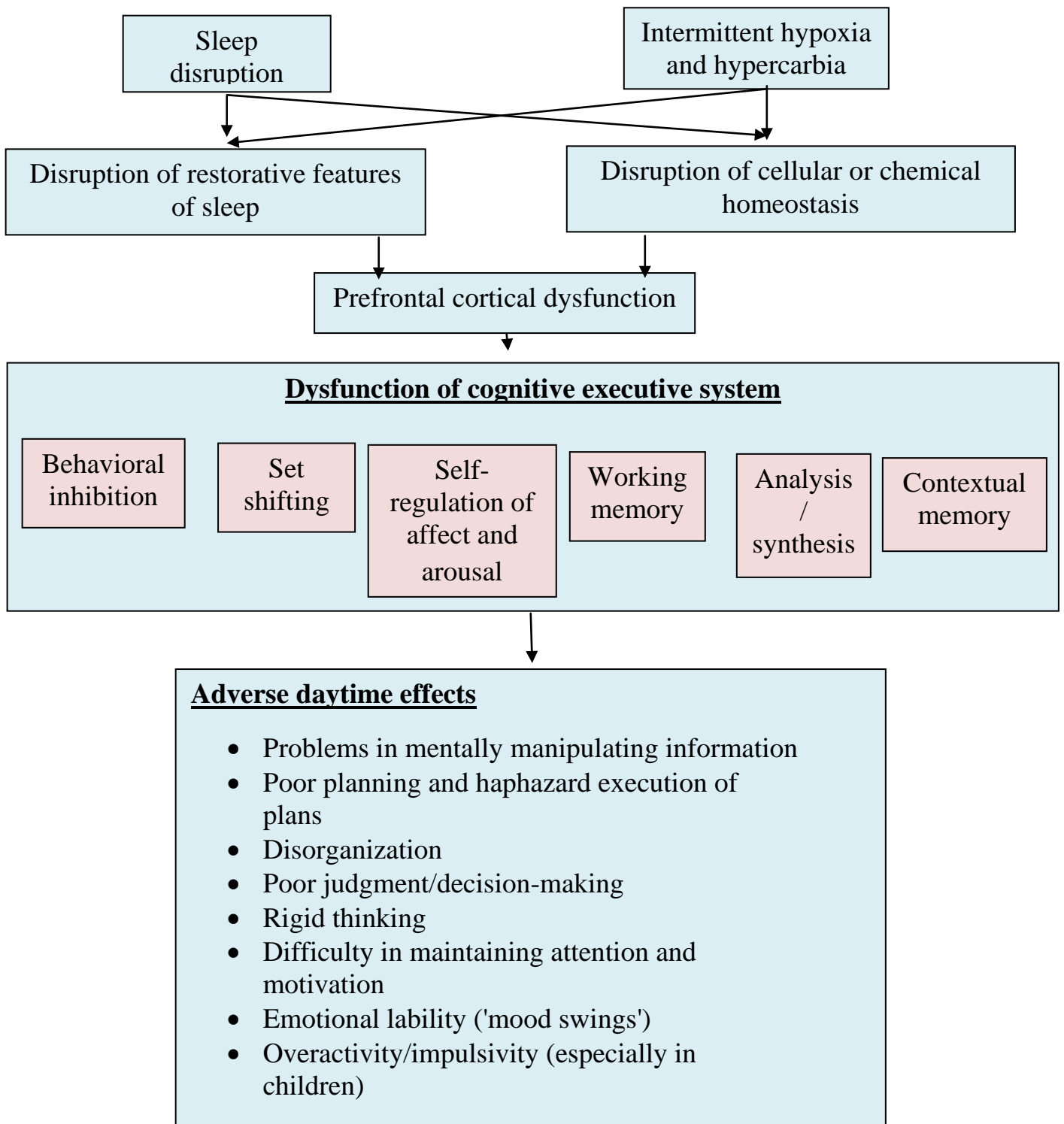


Figure47. The proposed prefrontal model. (Beebe and Gozal, 2002).

Behavioral therapy

Several behavioral interventions have been shown to improve CPAP adherence such as frequent contact and follow-up with the health care provider, intensive patient support, and cognitive behavior therapy (CBT) plus education (*Richards,et al., 2007*).

Frequent contact and follow-up with the health care provider who has expertise in treating sleep disorders is especially important during the first week of therapy. The interaction should focus on determining whether the CPAP settings need to be adjusted and whether the patient is tolerating CPAP therapy. There should be troubleshooting of any side effects and the level of adherence and associated outcomes should be determined. General encouragement should be offered and previously provided education should be reinforced (*Gay et al., 2006*).

Intensive patient support improves adherence to CPAP. A trial that randomly assigned 80 patients with OSA to receive intensive or usual support. Intensive support consisted of a three night trial of CPAP in a sleep center, CPAP education at home (including the partner), and ongoing home visits once CPAP therapy had begun. Intensive support improved CPAP adherence (5.4 versus 3.9 hours per night), symptoms, mood, and cognitive performance at six months (*Hoy,et al.,1999*).

CBT is a structured psychotherapeutic method used to alter attitudes and behaviors. The effect of CBT plus education on adherence with CPAP is likely related to improving self-efficacy(*Richards et al., 2007*).

Twelve patients with OSA were randomly assigned to receive CBT plus education or the same amount of therapist contact without information about OSA or CPAP (*Aloia et al., 2001*). CBT plus education consisted of two 45 minute sessions during which subjects were informed about the consequences of OSA and the beneficial outcomes associated with CPAP therapy. Patients who received CBT plus education were more adherent with CPAP therapy (approximately 3 hours longer per night) and more alert at 12 weeks than patients in the control group, even after controlling for age, education, disease severity, and vigilance (*Jean, et al., 2005*).

The benefits of CBT were confirmed by a meta-analysis of 17 randomized trials (1070 patients). Adherence was significantly higher among patients who received CBT compared to those who did not (85 versus 46 percent) (*Smith et al., 2009*).



Figure48. Education of the patient about different CPAP interfaces, how to use them. (*Richards, et al., 2007*).

I-Motivational enhancement therapy for CPAP

Motivational enhancement therapy (MET) is a line for treatment of OSA patients that aims at motivating adherence to Positive Airway Pressure (PAP) in obstructive sleep apnea (*Golay et al., 2006*).

I.1-Rationale for MET and approach

MET is an intervention that directly targets the constructs of readiness, importance, and confidence. It has been used successfully to alter behaviors such as excessive alcohol use and smoking. More recently, it's application has been broadened to the maintenance of positive health behaviors. Studies show that MET has been successfully used in increasing adherence to CPAP therapy (*Aloia, et al., 2005*).

MET is based on the core principles and therapeutic process of Motivational Interviewing (*Aloia, et al., 2005*).

Miller and Rollnick, 2002 developed Motivational Interviewing (MI) in which Motivational Enhancement is a patient-centered counseling approach that focuses on the concerns and perspectives of the individual and explores the individual's ambivalence about behavior change in a supportive and non-confrontational manner. Patients are encouraged to think about the benefits and barriers to behavior change (i.e., regularly using PAP therapy). Patients then need to incorporate their feelings of confidence and recognition of the importance of change into their consideration of the identified benefits of, and barriers to, PAP treatment. A key goal in motivational enhancement is to increase the amount of importance which the patient attaches to changing his/her behavior, while maintaining an empathic, supportive, and non-judgmental atmosphere.

The provider does not directly advocate for behavior change (i.e., using CPAP as prescribed), but rather asks key questions to help the patient explore his/her conflicted feelings about change, weigh the pros and cons of change, and allow the patient to realize the discrepancy between the present risky behavior (i.e., not using CPAP as prescribed) and the patient's self-identified goals and values. This lack of direct advocacy is important when it comes to CPAP adherence. Patients will often speak of roadblocks to using CPAP. Commonly cited roadblocks to CPAP use include discomfort, disturbance for bed partner, and travel. With MET the provider may use different methods such as open-ended questions and reflections to clarify the patient's concerns or strategic reflective listening (*Haynes et al., 2008*).

1.2-Indications

This treatment modality has been tested with patients who have been diagnosed recently with Obstructive Sleep Apnea (OSA) and who are judged to be good responders to PAP. A good responder is someone who exhibits AHI less than 10; remission of snoring; arousal index of less than 10 and PLMS index of less than 15 on a PAP titration study (*Golay et al., 2006*).

Adapted MET is particularly useful to a home care provider who has limited time to interact with patients when trying to ascertain and promote compliance with CPAP therapy. MET is adapted for medical settings which often have time-limited patient encounters and involve some form of feedback on the individual's health or behavior compared with normative data (*Aloia, et al., 2005*).

1.3-Contraindications

Weaver and Grunstein, 2008 found that MET may be less beneficial for patients with serious medical condition as end stage renal failure, severe COPD or severe asthma as these conditions may contribute to daytime sleepiness that does not improve with PAP treatment. Continued daytime sleepiness, despite adherence to PAP, could contribute to non-adherence to the device.

History of current diagnosis of a major psychiatric illness including current substance abuse with the exception of depression is a contraindication for MET. Certain psychiatric illnesses may interfere with a patient's ability to effectively participate in treatment (*Bardwell et al., 2001*).

Also, MET is contraindicated in notable cognitive impairment due to dementia or other causes that may interfere with the ability to engage effectively in the intervention (*Nae'gele' et al.,1995*).

1.4-Using theory to develop treatment strategies

Theories of behavior change have been used to guide the development of more effective interventions to improve adherence in other medical populations. Behavior change involves three specific constructs which are *readiness to change* that refers to an individual's motivation to change his/her behavior (e.g., begin to use PAP nightly), *perceived importance of change* which refers to an individual's belief that the benefits of changing behavior outweigh the costs and are relatively important in his/her life. This thought process is often referred to as the decisional balance, suggesting that the decision to change relies on weighing the benefits of change against its costs, and *Confidence in one's ability to change* which refers to an individual's perception of that

individual's ability to change his/her behavior under difficult circumstances. Self-confidence appeared to be the strongest single psychological predictor of long-term adherence (*Miller, Rollnick, 2002*).

1.5-Key concepts of MET

a) Developing discrepancy

This refers to the discrepancy reflecting the patient's ambivalence to making the change from not using CPAP to using CPAP. Most patients will have some idea that changing their behavior and using CPAP as prescribed is positive, but they will also see the barriers to use and, thus, will be ambivalent about whether to make the change. The provider tries, in a supportive manner, to help the patient see the discrepancy between the patient's current risky behavior and the patient's self identified goals and values. The patient's recognition that their behavior is hindering attainment of their goals or is not consistent with their values may make the patient feel some anxiety as they realize they are not meeting their goals. The desire to reduce that anxiety and to meet their goals becomes the impetus of change for the patient (*Prochaska, et al., 1997*).

b) Expressing empathy and avoiding argumentation

Cartwright, 2008 found that expressing empathy and avoiding argumentation can help the provider avoid being pulled into a debate over PAP use. Change does not come from making a person feel bad about their behavior. The goal is not to play the expert but to provide useful information the patient wants, allowing the patient to feel comfortable exploring the patient's conflicts about change. Information is never provided without consent from the patient. Be careful to express understanding of the patient's difficulty with the behavior change at hand (e.g., increasing CPAP use).

The goal is alignment with the patient's approach to change, not to confront the patient on the patient's poor adherence. Argument for change will, paradoxically, decrease the likelihood of patient change. The ambivalent patient will want to assert autonomy, especially within the context of not feeling in control of his/her health (*Sin et al., 2002*).

c) Roll with resistance

Resistance is expected when an ambivalent person is approached with information pointing to the need to change. The counselor must not resist in kind, but must roll with this resistance, supporting the patient's autonomy by emphasizing that it is the patient's choice as to whether or not the patient wants to change. Change can never be forced (*Prochaska, et al., 1997*).

d) Support self-confidence

Self-confidence is the patient's perceived ability to change a particular area of behavior. Patients are bound to have some successes in their past that will point to their ability to change. Promoting self-confidence involves highlighting those moments of past success and having the patient set small but achievable goals which will motivate the patient to achieve future success (*Prochaska, et al., 1997*). Together these concepts help guide the provider in what is often a difficult task – changing behavior (*Haynes et al., 2008*).

1.6-Guiding principles of MET and their application to OSA patients

Aloia et al., 2004 reported that there are six guiding principles in MET therapies. The provision of feedback is a key factor in MET therapy as it distinguishes MET from other educational therapies. Responsibility is given to the patient to change or not to change. The role of the provider of MET therapy is to assist as needed. This can easily be accomplished

during set-up or follow-up calls with the patient. One method of assisting is by providing advice when asked by the patient. The provider's role is always to empathize with the patient's barriers to change. The expression of empathy is key to MET. The poor self-confidence is among the greatest limitations to behavior change.

Table 11. Guiding principles of MET and their application to OSA patients (*Aloia et al., 2004*).

Guiding principle	Example
I. Feedback	Conduct a thorough assessment and offer personalized feedback about changes in clinical measures of OSA between the diagnostic PSG and titration PSG.
II. Responsibility	Emphasize that it is the patient's personal choice/responsibility to decide whether or not to use the CPAP machine.
III. Advice	Let patients know that, for health reasons, you recommend using CPAP, but that the decision is ultimately theirs.
IV. Menu	Patient chooses two different strategies that the patient can use to attempt to improve compliance (e.g., record changes in mood and sleepiness every day).
V. Empathy	Empathize with the patient's stated barriers to CPAP use and reinforce that these are common among other patients with OSA.
VI. Self Confidence	Highlight the statements of self confidence that the patient expresses during the session and the success the patient has had thus far.

1.7-Areas of MET questioning when the patient is going home with his/her CPAP device

It includes assessing the patient's readiness and confidence to use CPAP as recommended, determining which aspects of health are important to the patient providing information to the patient regarding the

aspects of sleep apnea that are related to the patient's health concerns. Information is only provided if the patient agrees to receive it, using feedback from the patient's own sleep study to place the severity of the patient's apnea in context for the patient, using the patient's titration study to demonstrate the stark difference between the patient's baseline apnea and the patient's apnea when treated with CPAP, assessing where the patient is in his/her preparedness to use CPAP regularly and developing goals for CPAP use over the next week (*Aloia et al., 2004*).

1.8-Implementation of MET

The timing of MET is not necessarily crucial. However, there is significant data suggesting that early intervention with PAP therapy may be best and that long-term adherence to PAP is often established early in the course of treatment. Timing MET to capitalize on the earliest teachable moment and the critical period of the development of adherence patterns can maximize use of the intervention. Perhaps the best time to start MET is when one begins discussing therapy itself (*Cartwright, 2008*).

Much of the intervention, educating the patient on how the patient can benefit from treatment and learning what is important to the patient, can begin when treatment is being discussed. It should be noted, however, that MET is designed to be applicable at any point during treatment or even after treatment has been refused (*Joo, Herdegen, 2007*).

A study showed that skipping CPAP for two or more nights within the first week of treatment signals potential for non-adherence and emphasizes the need for close follow-up during this period of time. The first week to month of home therapy appears to be the most Critical phase for intervention and securing long-term compliance (*Weaver et al., 1997*).

I.9-Step by step description of procedures

There are two face-to-face sessions, 1 week apart, and a follow-up phone call at 1 month (*Bardwell et al., 2001*).

a) Session 1: Patient assessment of PAP during titration night

Patients are asked about the experience of using PAP during the sleep study. This provides a starting point for learning about potential benefits and specific challenges experienced by them. The therapist reflects positive statements about PAP and empathizes when challenges are articulated, noting said challenges and positive statements to be used later in therapy (*Tyrrell et al., 2006*).

o *Assessment of motivation to use PAP*

The therapist asks patients to rate their motivation to use PAP on a scale of 1 to 10 in which 1 indicated that he doesn't want to use it at all and 10 indicated that he wants to use it very much. It is imperative that the therapist uses a dispassionate tone so that accurate information is shared. The therapist tailors the visit based on the patient's stated level of motivation (*Bardwell et al., 2001*).

Patients may also be asked why they feel their rating is not higher or lower. The question of why the rating is not higher should be asked first. The response represents the stated barriers to using PAP, and these can be used later in therapy. Perhaps more importantly, patients are asked why their rating is not lower. This is often more difficult for them to answer. Answers to this question represent the patient's own conceptualization of the benefits of treatment. These can also be used to support positive statements about PAP, and carefully challenge negative statements in future sessions (*Bardwell et al., 2001*).

○ ***Information exchange: video clip of OSA patient***

A video clip of somebody experiencing apnea is shown to patients to illustrate what happens during episodes of apnea. After the video clip, they are asked for thoughts and feelings about the video and how the video might relate to them. This allows them to consider how apnea episodes may impact them personally. A personal focus is applied whenever possible (*Seneviratne, 2004*).

○ ***Review of patient's pre-treatment polysomnography (PSG)***

The therapist shares the patient's diagnostic PSG with him or her. The severity of apnea is made clear by placing the patient's AHI on a graph representing normal, mild, moderate, and severe ranges of apnea. The same type of graphic representation is provided for the patient's oxygen desaturation index (outlining normal and abnormal levels). Feedback is provided using the Elicit–Provide–Elicit strategy (The therapist asks an open-ended question “Elicit”, shares information “Provide”, and follows up with another open-ended question “Elicit” to learn the patient's reaction.). Immediately after reviewing the information, patients are asked to share their thoughts and feelings about the graphs and how the results might relate to them (*Morgenthaler et al., 2006*).

○ ***Review of symptoms***

Patients are asked to specify the primary symptoms that led them to seek treatment. Typically, symptoms include mood, concentration, fatigue, and daytime sleepiness. In addition, medical and cognitive correlates of OSA that may not be apparent to patients are discussed (e.g., hypertension, cardiovascular risk, etc.). The tone of the exchange is neutral in order to provide the patients with relevant information while

allowing them to weigh the importance of the information to themselves personally (Gay *et al.*, 2006).

○ **Review of mortality graph**

A graph that shows cardiovascular morbidity and mortality rates according to PAP use (<1 hour; 1–6 hours;>6 hours) over a period of 0–120 months is shown to the patient (Figure 49). The graph illustrates the significantly higher cumulative survival rates for those who use PAP >6 hours and those who use it 1–6 hours as compared with those who use it <1 hour. Patients are asked for thoughts and feelings about the graph and the degree to which they perceive it as applying to them. This exchange offers an opportunity for patients to reflect upon the consequences of using or not using PAP over the long-term. As with all feedback, the Elicit–Provide–Elicit process is used (Campos *et al.*, 2005).

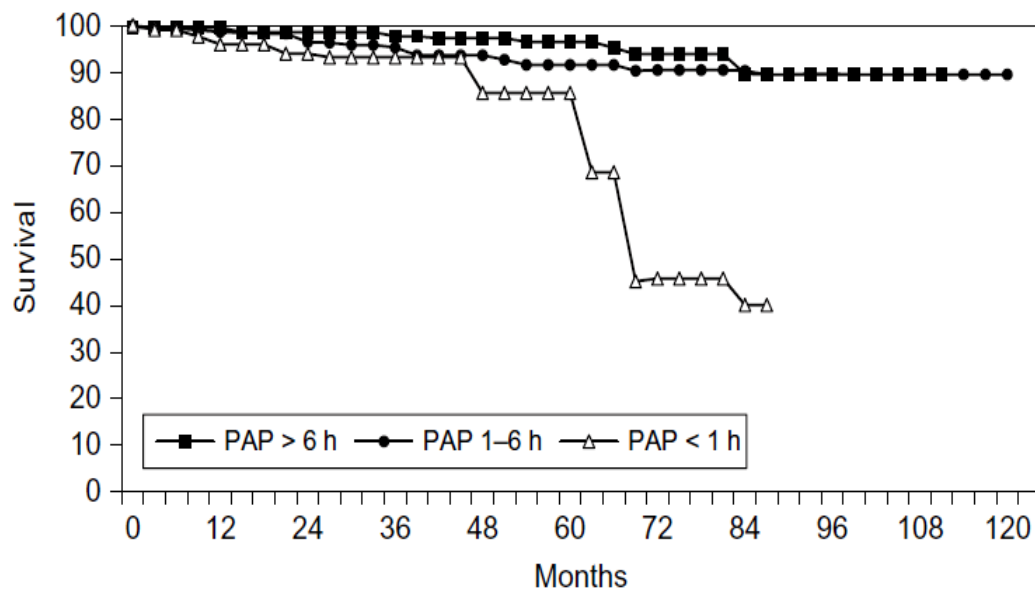


Figure 49. cumulative survival rates according to categories of PAP compliance in OSAS patients treated with positive airway pressure (Campos *et al.*, 2005).

Cumulative survival rates in the PAP > 6 h group were significantly higher than in the PAP < 1 h group ($p < 0.00005$). Cumulative survival rates in the PAP 1–6 h group were significantly

higher than in the PAP < 1 h group ($p = 0.01$). Cumulative survival rates were not different in the PAP > 6 h group and the PAP 1–6 h group ($p = 0.11$) (*Campos et al., 2005*).

○ *Review of titration PSG and comparison to diagnostic PSG*

The patient has a personalized review of the effectiveness of PAP based on his or her PSG data (diagnostic and titration). Special attention is given to the degree to which AHI and oxygen desaturation are improved with the use of PAP. As in the previous feedback section, normative values are used for comparison. The primary goal is to enhance patient self-efficacy by demonstrating that patients are capable of treating their OSA with PAP (*Weaver et al., 2007*).

○ *Negotiate a plan based on the patient's readiness and confidence*

To evaluate patients' confidence and readiness to initiate treatment, they are asked the extent to which they will be able to use PAP for 5+hours/night. Subsequently, patients are asked to set achievable, specific goals by identifying steps related to PAP use. The goals should be based on their readiness and confidence at the time of this visit. Goals are better set low than high, to allow them to be reachable and to enhance self-efficacy. Patients are asked to note daily improvements in patient-specific areas of concern so that any changes that result from PAP use are duly noted (*Saunama" ki et al., 2007*).

○ *Summary*

The therapist provides a summary of the session with highlights of the take-home message, including patient concerns about health related to having untreated OSA; patient reaction to feedback on the PSG; medical conditions the patient may be at risk for with untreated OSA; benefits the

patient experienced after using PAP; motivation to use PAP; and patient goals. The importance of Session 2 is emphasized, and is subsequently scheduled for 1 week after the mask fitting (*Rollnick et al., 2008*).

b) Session 2: patient's subjective appraisal of adherence to PAP

The patient is asked to provide an estimate of the frequency and duration of PAP use over the previous week. This provides a starting point for understanding the patient's experience of using the device during the first week of treatment. The therapist empathizes with difficulties and reinforces positive aspects of PAP. If the patient denies any changes due to PAP use, the therapist normalizes the fact that changes and benefits may be noticed over a period of time (*Bardwell et al., 2001*).

○ *Values assessment*

To begin the values assessment exercise, patients are asked to share tangible goals and activities related to daily living, and to share what is most important to them (e.g., having a nice place to live, spending time with family, being more active). The therapist encourages the patient to discuss three or more areas in order to select a suitable area for discussion. The therapist initiates a discussion of values in order to build a discrepancy between the patient's current status and aspired status with regards to the stated goal and its relation to apnea. The patient is asked to address the extent to which PAP might help with achieving the goal; the extent to which PAP might hinder the goal; the chances (scale 0–100) that the goal will be achieved if PAP is not used or is used less than 5 hours/night; the chances (scale 0–100) that the goal will be achieved if PAP is used 5+ hours/night. After a short summary, the therapist reflects

the patient's goals and highlights the discrepancy between current sleep apnea and the patient's broader values/goals (*Lim et al., 2007*).

○ *Decisional balance exercise*

This section includes a motivational enhancement technique designed to explore pros and cons of adherence to PAP. The therapist asks the patient to provide the downsides of using PAP, followed by the upside of using it. After summarizing, the therapist engages in a volley of reflections in an effort to explore the patient's ambivalence about using PAP. Overall, the therapist highlights ambivalence, normalizes this ambivalence as a natural part of this process, and expresses empathy. The therapist guides the patient through any ambivalence about using PAP, with the goal of its ultimate resolution (*Beebe et al., 2003*).

○ *Review of feedback on reaction time*

The patient is given a graph illustrating his or her reaction time off-treatment, which is based on AHI off-treatment. This is done in cases where reaction time is assessed before and after treatment. AHI pre- and post-treatment is covered in a previous assessment session. The patient is told how this reaction time equates to reaction times calibrated to alcohol use and an inferred risk for motor vehicle accidents. The Elicit–Provide–Elicit process is used when exploring the patient's thoughts about the graph. The therapist communicates with a non-judgmental tone and empathizes throughout the exchange. Ongoing reflections assist the patient with processing the information (*Morgenthaler et al., 2006*).

○ *Information exchange: PAP benefits for health and functioning*

The therapist shares information about the medical benefits of sleep apnea treatment (e.g., lower blood pressure, decreased risk of heart

attack) and how treatment is associated with improvements in daily functioning (e.g., increased alertness/productivity). The therapist provides a menu of options to the patient, who is asked to select the areas that are most relevant for him or her. A discussion of these areas then ensues. In addition, the therapist reviews medical related concerns previously identified in Session 1 to help the patient link stated concerns with medical benefits identified in this section. Reflections and empathy are used throughout this section (*Lam et al., 2005*).

○ *Information exchange: cognitive benefits of Sleep Apnea treatment*

The therapist shows the patient a graph which illustrates the relative increase in vigilance performance for those who use PAP ≥ 4 hours a night compared with those who use PAP < 4 hours a night, over a 6-month period of time (Figure 50). The graphs indicate that greater use of PAP increases vigilance from initial use to 3 months, with continued increases from 3 to 6 months. The patient is asked to share any thoughts and concerns. The therapist reinforces insight about the importance of PAP use, and empathizes with stated patient concerns (*Ferini-Strambi et al., 2003*).

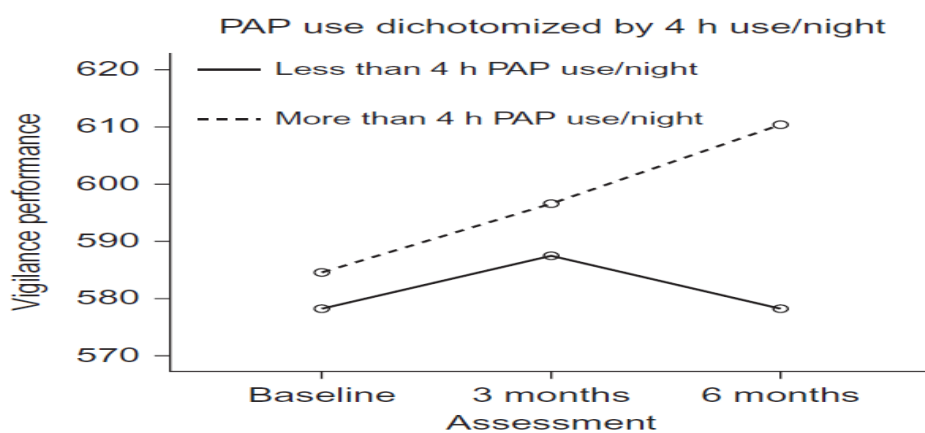


Figure 50. Relative increases in vigilance performance for Good (≥ 4 h/night) versus Poor (< 4 h/night) CPAP users over time (*Ferini-Strambi et al., 2003*).

○ *Assess patient motivation and confidence*

The patient is asked to rate, on a 10-point scale the motivation for treatment, the motivation to use PAP 5+ hours/night, and the confidence to implement treatment. The patient is asked why each rating was chosen, why a lower rating was not chosen, and what will need to occur for a higher rating to be given. This helps to clarify the patient's level of motivation and confidence, and potential factors that may interfere with PAP use (*Beebe et al., 2002*).

○ *Explore and identify experienced or anticipated barriers to PAP use*

Montserrat et al., 2011 reported that the therapist tailors this section to the patient's stated motivation from the previous section. The therapist explores experienced and anticipated barriers the patient raised during the session (e.g., decisional balance sheet) in an effort to highlight any remaining areas of concern. The patient is asked to consider creative ways to problem solve areas of concern and to identify steps to support the routine use of PAP.

○ *Renegotiate a plan based on readiness and confidence*

The therapist helps the patient to renegotiate goals based on his or her stated readiness and confidence to use PAP. Patient goals from Session 1 are reviewed, barriers are identified, and positive steps to remediate difficulties are discussed. The patient is encouraged to notice all changes, even subtle ones, that result from PAP use. In addition, the patient is asked to consider additional resources to help achieve the stated goals (*Budhiraja et al., 2007*).

○ ***Wrap up and results***

The therapist summarizes the session including patient concerns about symptoms of untreated OSA, potential barriers to PAP use, patient benefits from PAP use, current patient motivation and confidence, specific steps the patient will take with regards to PAP use, the patient's ultimate goal, and additional resources the patient has identified that can be helpful (*Morgenthaler et al., 2006*).

c) Phone call

○ ***Patient self-report of PAP use***

The therapist summarizes major points from Session 2, including the patient's stated goals. The patient is asked about the frequency of PAP use for the previous week, the greatest number of hours of use, and the lowest number of hours of use. The therapist asks the patient to reflect on the nights in which he or she was able to use PAP 5+ hours (and PAP <5 hours/night) and to identify supports and barriers to PAP use. In addition, the patient is asked to reflect on both scenarios (PAP use ≥ 5 hours, PAP use <5 hours) and to share the presence or absence of symptoms the next day. The therapist maintains a nonjudgmental attitude and a dispassionate tone, empathizing with the patient's difficulties and reflecting the patient's motivational statements (*Barnes et al., 2002*).

○ ***Building confidence to use PAP***

The therapist asks the patient to rate confidence on a 10-point scale, queries why a higher number was not chosen, and asks what needs to happen for confidence to increase. The therapist lists various situations and asks the patient to rate the confidence level in using PAP regularly ("not at all", "somewhat", "very"). The situations are ones typically faced by PAP users (increased time getting ready for bed; mask discomfort; side effects; feeling closed in; feeling embarrassed; traveling; concern

about disrupting a bed partner). The patient's unique concern is included in the list, as well. The patient is asked to develop a concrete plan to address each item to which the reply was "not at all confident". Upon completing the section, the patient is asked to identify a concern or challenge, other than sleep apnea, that he or she was able to overcome successfully. The therapist facilitates the patient's application of strategies used for the other problem to the issue of adherence in the use of PAP (e.g., internal motivation, external support) (*Faccenda et al., 2001*).

○ *Summarize session*

The therapist summarizes the patient's motivation and strategies to use PAP regularly, and how he or she intends to build confidence in using PAP (*Becker et al., 2004*).

d) Possible modifications with variants

Oksenberg et al., 2006 reported some modifications which can be made with regard to the specific information discussed with the patients, and any personal feedback can be employed and tied to potential PAP use. One such modification presented above involves performance testing before and after treatment. This is a powerful way of demonstrating real changes with treatment on a personal level. The tenets of MET, however, must remain constant. Among them, the Elicit–Provide–Elicit process must be used when dealing with situations in which defenses might be raised by the information provided to the patient.

II-Exposure therapy for claustrophobic reactions to Continuous Positive Airway Pressure

II.1-Indications

Exposure therapy is indicated for individuals with sleep apnea who are unable to tolerate CPAP devices due to anxiety reactions. Some patients with prescribed CPAP therapy for sleep-related breathing disorders experience claustrophobia, anxiety, or panic symptoms related to wearing the mask (feeling restricted) and/or tolerating the air pressure (feeling suffocated) (*McCrae and Ingmundson, 2006*).

II.2-Contraindications

Absolute contraindications for CPAP exposure therapy are unknown. It is reasonable to presume that this intervention would hold similar contraindications as exposure therapy for other anxiety disorders. Such contraindications may include unstable psychiatric symptoms (e.g., substance use, post-traumatic stress disorder, suicidal/homicidal ideation, psychosis), inability to maintain a therapeutic relationship, or economic/domiciliary instability (*Flack et al., 1998*).

II.3-Rationale for intervention

Claustrophobia is a form of specific phobia that entails extreme anxiety and panic elicited by situations such as tunnels, elevators, or other settings in which the individual experiences a sense of being closed in or entrapped. Claustrophobia is composed of two “core” fears including fear of restriction, and fear of suffocation (*Rachman and Taylor, 1993*).

Traditionally, the development of such fears has been explained by the two-factor model described by *Mowrer, 1960*. This model, which

evolves from the early work of *Pavlov, 1928* proposes that fear reactions such as claustrophobia are initially acquired by classical conditioning and then are maintained by operant conditioning. The classical conditioning involves the learning of associations between an unconditioned stimulus (UCS) and a conditioned stimulus (CS). Typically, the UCS is a stimulus that evokes danger or discomfort reactions that are called unconditioned responses (UCRs). A conditioning occurs when the CS is paired with the UCS over one or more trials, and through this association the CS comes to produce conditioned responses (CRs) that mimic the UCRs. Under proper circumstances, the CS–UCS link tends to decay over time as the CS is presented in the absence of the UCS (extinction) (*Foa and Kozak, 1986*).

However, when a fear such as claustrophobia develops, this process of extinction is blocked because the person quickly learns the fear can be reduced or prevented by avoiding or escaping the CS that causes the CR. This avoidance behavior reduces anxiety in the short-term, but prevents extinction from occurring thereby maintaining the phobia over time. Of course, learning history, personal belief systems, emotional processing, and other cognitive factors may be involved in the classical and operant conditioning of a phenomenon such as claustrophobia and contribute to the fear response (*Cox and Taylor, 1999*).

Because CPAP requires the patient to breathe pressurized air through a nasal or full-face mask strapped to the head, it is not difficult to understand how this treatment can tap into fears of suffocation and restriction. In some patients, this therapy may elicit memories of the original UCS or set of circumstances that elicited the claustrophobic response to CPAP (*Melanie and Edinger, 2011*).

Some patients may awaken from sleep feeling as though they are not getting enough air from CPAP, and experience frightening feelings of suffocation. This anxiety reaction may be exacerbated by nasal congestion experienced either as a side effect of the CPAP or due to other causes (sinus problems, respiratory infections, etc.). Such experiences may serve as a “one-trial” classical conditioning paradigm that sets the stage for CPAP avoidance and consequent perpetuation of the anxiety via operant conditioning. In either case, such difficulties tend not to remit spontaneously, and require targeted intervention (*Rachman and Taylor, 1993*).

The treatment of choice for specific phobias, including claustrophobia, is exposure therapy which describes a variety of techniques wherein the phobic individual confronts the feared object or situation either imaginally or in real life (in vivo). Typically, a hierarchy of fearful situations ranging from least to most anxiety-provoking is generated by the individual. Under the guidance of a therapist, the individual is supported in experiencing these feared situations in a gradual manner, and over time the anxiety decreases. The effectiveness of exposure therapy stems from learning to tolerate and manage anxiety without the need to escape or avoid the phobic stimulus, thereby permitting extinction to occur. The emotional processing of the fear is facilitated by fear activation (exposure to the phobic stimulus) in the context of incompatible information that there is no negative outcome and the individual is safe (*Wolitzky-Taylor et al., 2008*).

In addition to reducing fear, exposure therapy increases the individual’s perception of control over fear. Exposure-based therapies,

particularly in vivo exposure, produce robust and durable treatment effects for specific phobias (*Zayfert and Becker, 2007*).

Exposure therapy for CPAP emerged as a means of breaking the link between anxiety (triggered by CPAP as the CS) and the avoidance response. A deconditioning process based on those used for specific phobias is employed so that CPAP loses its value as a CS for anxiety and avoidance. This goal is achieved through the gradual re-exposure of the patient to CPAP in a structured manner so as to extinguish the link between CPAP as the CS, and the UCS that led to the initial problematic response (*Choy et al., 2007*).

Admittedly, this link is often a symbolic one in that CPAP was never associated with the original UCS but merely mimics it and elicits memories of it. Nonetheless, graded exposure to CPAP under therapeutic guidance helps eliminate this link and foster CPAP tolerance. Most likely, exposure therapy results in both a classical deconditioning of CPAP-related anxiety as well as significant subtle cognitive processing or reframing such that the CPAP device comes to be viewed as a safe, anxiety-free, and potentially rewarding activity (*Edinger and Radtke, 1993*).

II.4-Step by step description of procedures

Exposure therapy for CPAP-related claustrophobia is a short-term behavioral intervention that typically can be delivered effectively in one to six sessions over 1–3 months. There is no scientific evidence delineating specific treatment components that yield the most effective outcomes. This section describes typical clinical protocol which was found to be successful with military veterans. The components of each therapy session are outlined in table 12 (*Means and Edinger, 2007*).

The purpose of the first session is not only to implement the exposure intervention, but also to conduct an assessment and clinical history, evaluate the patient's knowledge of sleep apnea and CPAP therapy, and cultivate the therapeutic relationship. The session typically begins with asking patients to describe their experiences with CPAP thus far, which renders information about their perception of the problem. Obtaining information on which elements of CPAP therapy (e.g., tolerating air pressure, having the mask on the face, having the mask strapped over the head) the patient finds most distressing is informative (*McCrae and Ingmundson, 2006*).

An assessment of claustrophobia in other situations and the presence of other anxiety disorders assists in conceptualizing the problem. As part of the assessment, it may be useful to collect baseline measures of variables such as claustrophobia like Chasens questionnaire for adaptation of a claustrophobia for use with apnea patients or daytime sleep propensity (e.g., the Epworth Sleepiness Scale) that can be used to monitor treatment progress. It is often helpful to assess patients' knowledge and understanding of both sleep apnea and CPAP therapy. This information can be used to correct any misunderstandings and foster motivation to engage in CPAP therapy (*Chasens et al., 2005*).

Table 12. Exposure therapy session components. (*Means and Edinger, 2007*).

Table 12. Exposure therapy session components

Initial session (session 1)

- Assessment and history
 - Claustrophobia
 - CPAP therapy
- Patient education on sleep apnea and CPAP therapy
- Build therapeutic rapport and trust
- Implementation of exposure therapy
 - Presentation of treatment rationale
 - Establish exposure hierarchy
 - Goal setting/homework

Follow up sessions (sessions 2–6)

- Assess adherence to homework
Monitor progress
 - Patient self-report
 - Objective CPAP data
 - Problem-solve obstacles
 - Conduct in-session exposure trial (if indicated)
- Provide feedback and support regarding CPAP use

The first step in implementing the exposure protocol is presenting the treatment rationale, which is arguably the most important step in ensuring the success of the exposure intervention. Most patients will present to treatment having already developed a strong association between the CPAP device and emotional distress (anxiety, claustrophobia), such that they are avoiding CPAP entirely and are reluctant to try the device again. The therapist typically explain to patients that the purpose of treatment is to help them adapt to CPAP gradually through a series of “small steps” and practice. In this way, they can learn to overcome their discomfort with the device and use it

successfully. Patients may benefit from both an understanding of how their CPAP intolerance developed and a “normalization” of their problem through an explanation that claustrophobic reactions to CPAP are common. They may be reassured to learn that their problem is treatable, and that they can reap the rewards of sleep apnea treatment (*Koontz et al., 2003*).

Once the patient understands the treatment rationale and accepts the exposure intervention, the CPAP exposure steps are presented. A standard exposure therapy patient handout presents a hierarchy of steps from least anxiety-provoking to most. Although this hierarchy was found to be sufficient for many patients, individualizing the protocol for some patients is indicated. To break the association between night-time attempts at using CPAP and claustrophobic reactions, the therapist typically instruct patients to discontinue CPAP at bedtime during the initial stages of exposure treatment. Many patients are relieved by this instruction. In most cases, the exposure intervention itself can be enacted at home by the patient, per the patient handout. Regular daily CPAP practices in the home environment is emphasized, starting with short periods of time (5–10 minutes) and gradually increasing length of practice (up to 20–30 minutes) (*Zayfert and Becker, 2007*).

Table 13. Sample patient handout describing exposure steps for home practice. (*Zayfert and Becker, 2007*).

The goal of these steps is to help you to become more comfortable with CPAP while you are awake so that you can learn how to sleep easily with CPAP. For now, do not try wearing CPAP during sleep until you are comfortable with it during the daytime. If your machine has a RAMP button, you may use this function to keep the pressure at a low level during practices.

1. Turn the CPAP airflow ON. Hold mask over your nose, and practice breathing with machine on while awake. While you are doing this, keep your mouth closed and breathe regularly through your nose. Start with short periods of time (1–5 min) and gradually build up to longer periods of time.

2. Turn the CPAP airflow ON and wear the mask over your nose with the straps on your head. Practice breathing with CPAP on while awake. Wear CPAP for longer periods of time until you can have it on for 15–20 min comfortably.

3. Take a nap during the day with CPAP machine and mask on. It is not important whether you fall asleep or not – the goal is to rest comfortably in your bed with the CPAP on.

4. Wear CPAP at night when you go to sleep.

If you experience claustrophobia or uncomfortable feelings, go to previous step until comfortable. Then proceed to next step.

Patients are instructed to cease practice if anxiety rises to an uncomfortable level. It may be helpful for them to self-monitor their level of anxiety before and after practice sessions (*Zayfert and Becker, 2007*).

They are encouraged to proceed at their own pace and to reintroduce CPAP at bedtime only when they have increased their comfort with this device. Thus, the session concludes with a discussion of homework and goals regarding the home CPAP practice, along with an assessment of any obstacles or barriers to enacting the treatment recommendations at home. A follow-up session is scheduled for

approximately 2 weeks to evaluate progress. CPAP machines are equipped with internal software that records CPAP use on a removable card, and patients are asked to bring this card to their next session in order to monitor progress (*Koontz et al., 2003*).

Follow-up sessions provide an opportunity to evaluate progress, address problems, and conduct additional exposure therapy if needed. The session begins with a patient report of progress. Successes are reinforced through supportive comments, and obstacles are addressed as needed. The CPAP card is read during the session, which permits the patient to receive immediate feedback regarding treatment progress. Because the CPAP card displays the time of day and length of time CPAP was used, this information provides a direct and objective measurement of adherence to homework. Many patients who are practicing diligently with CPAP respond positively to seeing their efforts displayed on the CPAP report. When the CPAP report indicates that the patient engaged in CPAP exposure practices infrequently or not at all, the focus of the session becomes obstacles towards homework adherence. In some cases, the hierarchy may need to be modified or re-negotiated. Other individuals respond well to setting goals and rewards to improve adherence to home practice. For example, one patient set a goal of practicing with CPAP at home 5 days a week for 3 weeks. When this goal was met, he rewarded himself by dining at his favorite steakhouse (*Means and Edinger, 2007*).

If the patient continues to report claustrophobic reactions while using CPAP at home, or does not seem to be making progress through home practice, more intensive therapeutic guidance and an in-session exposure trial are indicated. The patient is asked to bring his or her CPAP equipment to the session. Ask patients to apply their CPAP as they do at

home. This request evolved from observations that, for some patients, “claustrophobia” is caused by an incorrectly applied or fitted mask. Some patients who, despite receiving CPAP training from nursing staff, therapist and a home care company, were applying the mask upside down or adjusting the straps incorrectly. Correcting these errors in mask application resolved the claustrophobia. Along these same lines, claustrophobia can sometime be ameliorated by trying an alternative mask style, and this observation bespeaks the importance of close follow-up by an experienced treatment team to resolve such problems expediently(*McCrae and Ingmundson, 2006*).

The therapist begins the in-session exposure trial with the patient seated in a chair. Many patients report increased feelings of claustrophobia while reclined in bed compared to sitting, probably in part due to obesity-related breathing restriction in a supine position. By explaining each step of the procedure at the outset, the therapist engenders the patient’s trust and confidence. When exposure therapy is used for other anxiety disorders, the importance of the therapeutic relationship is well-recognized. It is also critical that the patient maintain a sense of control during the exposure process. To this end, the patient is permitted to hold and remove the mask during the entire procedure and can remove the mask quickly if needed. Adjustments to mask fit are made only after the patient gives permission to be touched (*Noyes and Hoehn-Saric, 1998*).

Depending on the degree of CPAP-related claustrophobia, the patient will be asked to start at a level that induces anxiety at a tolerable level. For some individuals, this may be as brief as holding the mask over their nose for a few seconds at a time at the lowest pressure of 4 cm/H₂O

(as per manufacturers' guidelines, patients are never asked to wear the CPAP mask unless the air pressure is on). The patient is encouraged to keep the mask in place until the anxiety subsides. Asking the patient to rate his or her anxiety level on a scale of 0–100 provides a method of measuring anxiety levels during the session. The patient sets the pace and progresses through the additional hierarchy steps during the same or subsequent sessions. Because the exposure therapy is provided in the context of a sleep laboratory, the advantage of observing the patient using CPAP while reclined on a bed is available. With sufficient exposure, it is not unusual for the patient to fall asleep during a session. As the patient becomes increasingly comfortable with CPAP, it is important to increase tolerance of the CPAP pressure to the therapeutic level. In-session successes are strongly reinforced through verbal feedback from the therapist. Patients are often surprised at their progress, and develop a sense of confidence, mastery, and self-efficacy (*Zayfert and Becker, 2007*).

One of the risks of exposure therapy is creating an increase in anxiety symptoms if the exposure proceeds too quickly. Additionally, it is possible that patients for whom the anxiety level was too uncomfortable dropped out of treatment altogether. Exposure therapy requires patients to be motivated and committed (*Jaycox and Foa, 1996*).

Once patients complete the exposure protocol and are using CPAP at home successfully, they may find it easier to maintain successful CPAP use with ongoing support and feedback about their increasing CPAP use provided by the device's internal adherence monitoring software. Follow-up visits may be spaced at increasing intervals (e.g., 3 months, 6 months, 12 months), or as needed (*McCrae and Ingmundson, 2006*).

II.4-Possible modifications/variants

Koontz and his colleagues, 2003 found that there is a variety of modifications and variants that may increase treatment success for certain individuals. Alternative exposure protocols for adults and children have been published. The CPAP exposure protocol also can be modified and implemented prophylactically to prevent anticipated claustrophobia. For example, prior to the diagnosis of sleep apnea, some patients express a concern about being able to tolerate CPAP on the night of their sleep study. These individuals often benefit from the opportunity to try CPAP gradually before their sleep study. In addition, the exposure treatment have been employed successfully with other types of positive airway pressure delivery systems (e.g., auto-CPAP, BiPAP, etc.). Although home CPAP practice is routinely prescribed, this may not be necessary for treatment success if exposure is conducted in session (*McCrae and Ingmundson, 2006*).

The implementation of relaxation training may be indicated for patients who are unable to reduce their level of anxiety sufficiently during the exposure protocol. In such cases, it may be beneficial to cultivate relaxation through therapeutic techniques such as relaxation training, visualization, or deep breathing prior to initiating the exposure therapy. Once the patient becomes adept at relaxing, the exposure therapy can be initiated. This technique can help patients learn how to manage anxiety and use CPAP while in a relaxed state (*Rains, 1995*).

A number of additional therapeutic strategies may enhance the exposure treatment. As an adjunctive intervention, cognitive-behavioral therapy techniques can be useful both in challenging patient beliefs or thoughts that may be interfering with the exposure therapy and in helping

the patient develop positive coping statements. As an example, many claustrophobic patients, upon applying CPAP, think, “I can’t breathe. I am suffocating.” Helping the patient recognize this automatic thought and substitute it with a helpful thought (such as, “I can breathe easily and freely with CPAP”) can reduce anxiety (*Zayfert and Becker, 2007*).

Because exposure therapy involves discomfort to the patient, difficulties with adherence, attendance, and motivation should be anticipated. Such problems can be addressed through direct therapeutic discussion, or other techniques such as behavioral contracts, goal setting, or the use of rewards (*McCrae and Ingmundson, 2006*).

II.5-Proof of concept supporting data/evidence base

Claustrophobia is a commonly reported side effect of CPAP therapy, and may lead to treatment abandonment. Almost one-third of sleep apnea patients endorse CPAP-related claustrophobia (*Chasens et al., 2005*). In a large sample of newly diagnosed sleep apnea patients, CPAP-related claustrophobia was perceived as one of the largest deterrents to CPAP therapy, with less than half of patients reporting that they would use CPAP if they felt claustrophobic (*Weaver et al., 2003*).

Chasens and his colleagues, 2005 found that sleep apnea patients recruited from multiple North American sleep centers were more than twice as likely to have low CPAP adherence if they scored high on a claustrophobia questionnaire. Interestingly, claustrophobia scores decreased over the 3-month treatment period, which may reflect a naturalistic exposure to CPAP.

In a retrospective case series study, patients with CPAP-related claustrophobia attended between one and six exposure sessions with a

behavioral sleep psychologist. At post-treatment (an average of 15 weeks after the final therapy session), patients used CPAP on a greater percentage of nights and for more hours per night compared to pre-treatment. Effect size calculations for CPAP adherence variables revealed a large effect of treatment. Furthermore, neither patient characteristics, nor number of treatment sessions, nor length of the follow-up period predicted exposure treatment response (*Means, Edinger, 2007*).

The individual case studies provide a glimmer of optimism that treatment gains endure long term, both at 6 months and 6 years after treatment of CPAP-related claustrophobia (*McCrae and Ingmundson, 2006*).

CPAP exposure therapy is a promising intervention that is both clinically appealing and easy to implement. However, this intervention is lacking rigorous scientific evaluation; the overall state of the research support is weak, suffering from uncontrolled trials and small sample sizes. Future studies with randomized controlled trials, larger sample sizes, objective measures of CPAP adherence, and long-term outcomes are needed. Studies would also benefit from formalizing the diagnosis of CPAP-related claustrophobia, standardizing measures of claustrophobia, and further investigating treatment drop-outs and predictors of outcome. Additionally, the extant published reports have used an in vivo exposure protocol without the use of relaxation (*Casas et al., 2000*).

Thus, it remains to be determined whether the addition of relaxation training improves outcomes, at least for some individuals. Measures of treatment enactment are needed to assess adherence to assigned home practice, and its influence on outcome. Finally, there is virtually no information on whether gender or other demographic

variables influence treatment response. Despite these limitations, CPAP-related exposure therapy has become a routine part of our clinical sleep services due to its high demand and rewarding clinical outcomes (*Means, Edinger, 2007*).

Applications of adherence to CPAP in OSAS

Adherence to CPAP has many figures which have been applied in the real world. Follow up cards used in sessions for treatment of patients with OSAS using CPAP machine is a practical implementation of adherence. Also, American Sleep Apnea Association has developed guidelines for drivers who suffer from OSA which were applied as OSA and Trucker recommendations. Insurance companies have their own recommendations for paying for CPAP in treatment of OSA (*Wichniak et al., 2011*).

I. Follow up of patient with OSAS using CPAP

There are two face-to-face sessions, 1 week apart, and a follow-up phone call at 1 month (*Bardwell et al., 2001*).

I.a.Session1

It includes reviewing subject's sleep data, reviewing symptoms noticeable to the subject like anergia and excessive day time sleepiness. Then reviewing symptoms not apparent to patient such as hypertension and cardiac problems and results of performance on cognitive tests. The next step is rating the importance of treatment to the patient followed by reviewing PSG with CPAP and specifying how this might address the above problems. The last step is discussing the advantages and disadvantages of treatment and developing goals for therapy (*Means and Edinger, 2007*).

I.b.Session2

It includes examining compliance data for the first week, discussing noticeable changes with treatment, then discussing changes

not apparent to patient such as hypertension and cardiac problems followed by troubleshooting discomfort of patient. Realistic expectations of treatment are also discussed, and treatment goals are reviewed (*Morgenthaler et al., 2006*).

Ongoing Management of CPAP Usage

It is suggested that approximately 7, 30, 60 days and approximately 12 months after treatment initiation are appropriate times (*Richards et al., 2007*).

At this time the provider shall determine the patient's usage from the meter of the CPAP device and calculate the average daily hours of CPAP usage, checking the device and humidifier for satisfactory operation and Checking filters, mask and head-gear for satisfactory condition and advise the patient of any faults and suggested remedial actions (*Aloia et al., 2007*).

I.C. Exposure therapy for claustrophobic reactions to CPAP

Exposure therapy is indicated for individuals with sleep apnea who are unable to tolerate CPAP devices due to anxiety reactions. It is contraindicated in unstable psychiatric symptoms (substance use, post-traumatic stress disorder, suicidal/homicidal ideation, psychosis), inability to maintain a therapeutic relationship, or economic/domiciliary instability. Exposure therapy for CPAP-related claustrophobia can be delivered effectively in one to six sessions over 1–3 months (*McCrae and Ingmundson, 2006*).

A-Initial Session (Session 1)

It includes assessment and history taking from the patient as regard claustrophobia and CPAP therapy, patient education on sleep

apnea and CPAP therapy, building therapeutic trust and implementation of exposure therapy by presenting the rationale for treatment, establishing exposure hierarchy and setting goals and homework (*Zayfert and Becker, 2007*).

B-Follow up Sessions (Sessions 2–6)

Assessment of adherence to homework and monitoring progress as regard patient self-report and objective CPAP data such as CPAP card which is read during the session is the first step. The second step is solving obstacles. If the patient continues to report claustrophobic reactions while using CPAP at home in-session exposure trial should be conducted by asking patients to apply their CPAP as they do at home. This reveals that, for some patients, “claustrophobia” is caused by an incorrectly applied or fitted mask. claustrophobia can sometimes be ameliorated by trying an alternative mask style. Feedback and support should be provided regarding CPAP use. Once patients complete the exposure protocol and are using CPAP at home successfully, follow-up visits may be spaced at increasing intervals like 3 months, 6 months, 12 months, or as needed (*McCrae and Ingmundson, 2006*).

The CPAP exposure protocol also can be modified and implemented prophylactically to prevent anticipated claustrophobia. Exposure treatment can be employed successfully with other types of positive airway pressure delivery systems such as auto-CPAP, BiPAP. The implementation of relaxation training may be indicated for patients who are unable to reduce their level of anxiety sufficiently during the exposure protocol (*Means and Edinger, 2007*).

II. American Sleep Apnea Association OSA and Trucker recommendations

The Motor Carrier Safety Advisory Committee (MCSAC) and the Medical Review Board (MRB) of the FMCSA (Federal Motor Safety Administration) held a joint public meeting on Obstructive Sleep Apnea and made some very significant recommendations to the FMCSA. This guidance only focuses on those drivers with the highest pre-test probability of having OSA (*American Sleep Apnea Association, 2011*).

II.a.Recommendation 1

FMCSA provided new guidance for medical examiners that drivers with a Body Mass Index (BMI) of greater than 35 need to be evaluated for obstructive sleep apnea (OSA) using an objective test. The driver may be given a 60 day conditional certification during the evaluation and treatment process. A driver diagnosed with OSA may maintain certification with evidence of appropriate treatment and effective compliance and if the examiner determines that the condition does not affect the drivers ability to safely operate a commercial motor vehicle (CMV). Subsequent certification should be no longer than one year term. Future certification should depend on proof of continued compliance with treatment (*American Sleep Apnea Association, 2011*).

II.b. Recommendation 2

Immediate disqualification (from Medical Expert Panel/Medical Review Board recommendations) for individuals who report that they have experienced excessive sleepiness while driving, individuals who have experienced a crash associated with falling asleep, individuals with an AHI > 20, until such an individual has been adherent to CPAP. They can be conditionally certified based on the criteria for CPAP compliance.

Individuals who have undergone surgery and who are pending the findings of a postoperative evaluation should be immediately disqualified. Also, Individuals who have been found to be effectively non-compliant with their CPAP treatment should be disqualified (*American Sleep Apnea Association, 2011*).

III. Medicare compliance for post-PAP set up

Medicare Compliance Requirements

Medicare has specific guidelines that must be followed to qualify for a CPAP or BiPAP and specific guidelines for that equipment to be reimbursed after the initial 3 month period. Medicare does in-force these requirements and will not reimburse for the machine or supplies if these criteria are not met (*Valerie, 2011*).

The physician, the Sleep Lab and the equipment provider will work to make sure all requirements are met to ensure the initial 3 months coverage prior to getting the equipment (*Valerie, 2011*).

After patient set-up with his equipment he will need to make sure the compliance data and follow-up visit with his physician is met per the Medicare guidelines (*Valerie,2011*).

Continued coverage of a CPAP or BiPAP beyond the first three months of therapy requires that, no sooner than the 31st day but no later than the 91st day after initiating therapy, the treating physician must conduct a face-to-face clinical re-evaluation and document that the beneficiary is benefiting from PAP therapy and the symptoms of obstructive sleep apnea are improved. Objective evidence of adherence to use of the PAP device is reviewed by the treating physician (*Valerie, 2011*).

LCD Information Adherence to therapy is defined as use of PAP \geq 4 hours per night on 70% of nights during a consecutive thirty day period anytime during the first three months of initial usage (*Valerie, 2011*).

If the above criteria are not met, continued coverage of a PAP device and related accessories will be denied as not medically necessary. If the physician re-evaluation does not occur until after the 91st day but the evaluation demonstrates that the patient is benefiting from PAP therapy as defined in criteria above, continued coverage of the PAP device will commence with the date of that reevaluation(*Valerie, 2011*).

Beneficiaries who fail the initial 12 week trial are eligible to requalify for a PAP device but must have both face-to-face clinical re-evaluation by the treating physician to determine the etiology of the failure to respond to PAP therapy; and, repeat sleep test in a facility-based setting (*Valerie, 2011*).

In most cases the equipment provider will be able to download compliance data off of CPAP/BiPAP and print a report to show adherence to equipment per medicare requirements. Many physicians also have the ability to download the equipment for proof of compliance (*Valerie, 2011*).

Medicare requires documentation of the following information within 90 days of PAP initiation in order for them to continue paying for patient's CPAP or Bi-level PAP treatment for obstructive sleep apnea Table (14) (*Valerie, 2011*).

Table 14. Medicare documentation of the information to continue paying for patient’s CPAP or Bi-level PAP treatment for obstructive sleep apnea (*Valerie, 2011*).

Medicare requires documentation of the following information within 90 days of PAP initiation in order for them to continue paying for your patient’s CPAP or Bi-level PAP treatment for obstructive sleep apnea. To comply with this new Medicare regulation, please select and complete either Option 1 OR Option 2 below.

- Option 1 – Sleep Health *Centers* Manages Medicare Compliance

I would like my patient to be evaluated by a Sleep Health *Centers*’ sleep specialist to monitor compliance and response to PAP therapy. Sleep Health *Centers* will complete Medicare required documentation.

Option 2 – Physician Manages Medicare Compliance

I will comply with Medicare regulations by ensuring both of the following two criteria for Medicare have been met.

A. Documentation of Use of PAP Therapy. Over a 30 day period, your patient has used CPAP or Bilevel PAP for ≥ 4 hours/night _____ % of the time. Medicare requires 4+ hours/night of use $\geq 70\%$ of the nights in 30 consecutive days for continued coverage for PAP therapy.

Note: If this number is below 70%, please work closely with your patient in concert with our respiratory therapists to increase this number to $\geq 70\%$ before the end of the 90 day period.

B. Documentation of Symptomatic Improvement. Medicare also requires the documentation of benefit from PAP treatment of obstructive sleep apnea. There needs to be a face-to-face meeting between you and your patient documenting one of the following symptomatic improvements:

- | | |
|--|---|
| Improved Not improved Daytime sleepiness/fatigue | <input type="checkbox"/> <input type="checkbox"/> |
| Observed apneas/choking/gasping during sleep | <input type="checkbox"/> <input type="checkbox"/> |
| Morning headache | <input type="checkbox"/> <input type="checkbox"/> |
| Other: | <input type="checkbox"/> <input type="checkbox"/> |

SIGNATURE AND DATE

Suggested protocol for follow up of Positive Airway Pressure adherence of patients attending Mansoura sleep disordered breathing clinics

After reviewing different protocols used for augmentation of adherence of OSAS patients to CPAP use, we recommend the following protocol for follow up of adherence of whether using CPAP or other modes of positive airway pressure according to facilities available with us.

Sleep Disordered Breathing Unit
Chest Department
Mansoura University Hospital

Hotline: 0190974090

Patient name **Age:**

Sex: **Marital status**..... **Residence:**

special habit **BMI:** **Neck Circumference(NC) :**

BP: **ESS score:**

Berlin questionnaire :

group 1..... **group 2**..... **group 3**.....

Total score.....

Polysomnographic data:

AHI: **Supine AHI:** **Basal Spo2** **Oxygen desaturation index**.....

Minimum Spo2 **Arousal index**..... **Blood pressure**

Basal heart rate..... **Maximum heart rate** **Minimum heart rate**

Diagnosis:

.....

Comorbidity:.....

.....

Equipment:

.....

Prescribed **pressure:**

.....

Predictive equation for therapeutic pressure of CPAP:

$$\text{Therapeutic pressure} = (0.16 \times \text{BMI} + 0.13 \times \text{NC} + 0.04 \times \text{AHI} - 5.12).$$

Bed partner assessment in OSAS

- Do you notice an improvement in your own sleep quality since your partner started CPAP?

- Do you notice an improvement in your own daytime alertness since your partner started CPAP?

- Do you notice an improvement in your own mood since your partner started CPAP?

- Do you think your own quality of life is better since your partner started CPAP?

- Do you notice an improvement in your partner's sleep quality and daytime alertness since starting CPAP?

- Do you notice an improvement in your partner's mood since starting CPAP?

- Do you think your partner's quality of life is better since starting CPAP?

- Do you think CPAP therapy to your partner has improved your personal relationship?

Scores assigned: worse = 1; none=0; little=1; moderate=2; marked=3

General advice

- Patients who smoke should be advised to stop.
.....
- Alcohol should be avoided.
.....
Nocturnal sedatives or sleeping tablets should be avoided.
.....
- Advice regarding body weight and its interaction with OSA should be provided if appropriate.
.....
- Patients should be informed to avoid sleeping in supine position (eg. Using Z-Zoma).
.....
- Rapid treatment of nasal obstruction should be viewed as an adjunct to CPAP therapy, potentially improving adherence.

Exposure therapy for claustrophobic reactions to CPAP

A- Initial Session (Session 1)

- Build therapeutic trust
- Patient education on sleep apnea and CPAP therapy
- Assessment and history of previous Claustrophobic reactions and the presence of other anxiety disorders
- Identification of the cause of claustrophobia (tolerating air pressure, having the mask on the face, having the mask strapped over the head) .

- **Implementation of exposure therapy**
 - **Presentation of treatment rationale**
 - **Establish exposure hierarchy**
 - **Goal setting/homework**

Patient handout for home practice.

- **Do not try wearing CPAP during sleep until you are comfortable with it during the daytime.**
 - **Use RAMP button to keep the pressure at a low level during practices.**
- 1- **Turn the CPAP airflow ON. Hold mask over your nose, and practice breathing with machine on while awake.**

While you are doing this, keep your mouth closed and breathe regularly through your nose. Start with short periods of time (1–5 min) and gradually build up to longer periods of time.
 - 2- **Turn the CPAP airflow ON and wear the mask over your nose with the straps on your head. Practice breathing with CPAP on while awake. Wear CPAP for longer periods of time until you can have it on for 15–20 min comfortably.**
 - 3- **Take a nap during the day with CPAP machine and mask on. It is not important whether you fall asleep or not the goal is to rest comfortably in your bed with the CPAP on.**
 - 4- **Wear CPAP at night when you go to sleep.**

If you experience claustrophobia or uncomfortable feelings, go to previous step until comfortable. Then proceed to next step.

B-Follow up Sessions for Claustrophobia (Sessions 2–6)

- **Assess adherence to homework (Monitor progress)**

.....
- **Patient self-report**

.....
- **Objective CPAP data (CPAP card)**

.....
- **Problem-solve obstacles**

.....
- **Conduct in-session exposure trial (If the patient continues to report claustrophobic reactions while using CPAP at home) asking patients to apply their CPAP as they do at home.**

.....
- **Provide feedback and support regarding CPAP use (once patients complete the exposure protocol and are using CPAP at home successfully, follow-up visits may be spaced at increasing intervals (e.g., 3 months, 6 months, 12 months), or as needed)**

.....

Implementation of CPAP use

A-Session 1

- **Review subject’s polysomnographic data (AHI.....,Basal Spo2 ,Oxygen de-saturation index.....,Minimum Spo2.....,Arousal index Basal heart rate.....Maximum heart rate Minimum heart rate Blood pressure) .**
- **Review symptoms noticeable to the subject (e.g., fatigue , ESS and morning headache) .**
- **Review symptoms not apparent to the patient which have relation to sleep disordered breathing (e.g., hypertension, cardiac problems, stroke and diabetes mellitus) .**
- **Review results of performance on cognitive tests (Reaction time).**
- **Video clip (Arabic version) about OSAS and its relation to co-morbidity and impaired life style and possible accident.**
- **Rate the importance of treatment**
- **Review PSG with CPAP if available and specify how this might address the above problems(AHI ,Basal Spo2 , ODI , Minimum Spo2 ,Arousal index Basal heart rate.....Maximum heart rate , Minimum heart rate BP.....) .**
If not available(eg. Using Auto-CPAP or predictive equation) inform the patient that sure improvement of the abnormalities in PSG will occur with regular use of CPAP.
- **Discuss the advantages and disadvantages of treatment**
- **Develop goals for therapy for subsequent reassessment in the next sessions**

Ongoing Management of CPAP Usage: (7, 30, 60 days and approximately 12 months after treatment initiation)

B-At this time (SESSION 2 ,3,4,)

- **Examine adherence data for the first week (Total hours of use by machine..... Average hours of use /night..... Patients reported side effect.....) .**
- **Discuss noticeable changes with treatment**

- **Discuss changes not apparent (hypertension ,cardiac problems , stroke and diabetes mellitus).**
.....
- **Troubleshoot discomfort (Check the device and Humidifier, filters, mask , tube and head-gear)**
.....
- **Discuss realistic expectations of treatment**
.....
- **Review treatment goals for subsequent reassessment in the next sessions**
.....

نشرة تعليمية لمرضى متلازمة ضعف و انقطاع التنفس أثناء النوم

الخط الساخن: 0190974090

متلازمة ضعف و انقطاع التنفس أثناء النوم

يعرف بأنه انقطاع جزئي أو كلي في الطرق التنفسية العلوية، مما يؤدي إلى تأثيرات جسدية أو عقلية. وقد يكون:

إما: توقف تنفس انسدادى: حيث يتوقف جريان الهواء ويتحرك جدار الصدر بحركات عجانبية كتعويض على ذلك.

أو: توقف تنفس مركزي : حيث يتوقف جريان الهواء و يرافق ذلك توقف في حركة جدار الصدر.

أو: توقف التنفس المشترك: حيث يبدأ توقف التنفس بتوقف مركزي يليه توقف انسدادى.

على عكس ما يعتقد البعض بأن الشخير عبارة عن خصلة عادية تأتي نتيجة للإرهاق اليومي ، يجب الانتباه من أن هذا الأمر قد يكون علامة على إصابة الشخص بمرض توقف التنفس الإسدادي أثناء النوم، والذي له عواقب وخيمة.

ويجب التفريق بين الشخير الناجم عن الإرهاق، وحالة توقف التنفس الإسدادي أثناء النوم، حيث ينقطع التنفس عند النوم لمدة وجيزة بشكل متكرر، وهو الأمر الذي يؤدي إلى أن ينام الشخص بشكل متقطع وانخفاض نسبة الأكسجين في الدم، المسببان لأمراض القلب وضعف الذاكرة.

وتبين أن هذه الحالة تؤدي إلى زيادة الاكتئاب عند المصابين بها، مشيرة إلى أن الاضطرابات بالتنفس عادة ما تزيد من إصابة الناس بالكوابيس أو أمراض مثل اضطراب قلق الكوارث.

إضافة إلى ذلك إصابة الناس بحالات من النعاس أثناء النهار مما يؤدي إلى وقوعهم بحوادث سير وغيرها نظرا لفقدانهم التركيز وغلبة النعاس عليهم.

وعلاجا لهذه المسألة أنه "يجب على المصابين بهذه الأعراض ويعانون من الشخير الثقيل أن يستشيروا طبيبا بأسرع وقت ممكن".

آلية توقف التنفس الانسدادي أثناء النوم:

يحدث أثناء النوم كما هو معروف ارتخاء شبه كامل لكافة عضلات الجسم وتبقى المراكز الحساسة فعالة لإبقاء التنفس والقلب يعملان. لوحظ في المرضى الذين لديهم توقف التنفس الانسدادي تضيق في البلعوم يترافق مع تراجع خلفي للفك السفلى الذي يترافق بتراجع قاعدة اللسان للخلف، وبسبب الارتخاء أثناء النوم تضغط قاعدة اللسان على البلعوم وتتحرك للهاة للأسفل، وينتج عن ذلك انقطاع التنفس مع ما يرافقه من زيادة في ضغط الهواء السلبي أثناء الشهيق. إذا استمرت الآلية السابقة بدون يقظة، فإن ذلك يؤدي إلى وفاة المريض بالاختناق بسبب نقص الأكسجة الشديد وما يرافقه من توقف القلب. ولكن نقص الأكسجة يؤثر على الدماغ فيوقف المريض انعكاسياً حتى دون دراية المريض لما يجري أثناء النوم. ولكن تكرار اليقظة الليلية تجعل الإنسان مصاباً بالنعاس النهاري.

يحدث تضيق الطرق التنفسية زيادة إجهاد العضلات التنفسية، ويحدث أيضاً الشخير و يليه اليقظة كما ذكرنا. يترافق ذلك مع نقص الأكسجة وزيادة غاز ثان أكسيد الكربون في الجسم ، وهذا يسبب عند المريض احمرار الدم و تضيق الأوعية الرئوية ويحدث ذلك ارتفاع ضغط الدم واضطراب نظم القلب يحدث نقص الأكسجة أيضاً اضطراباً وتناقصاً للإدراك العقلي.

أعراض وعلامات توقف التنفس الانسدادي

- اضطراب النوم، الشخير، تقطع النوم، عدم الإشباع النومي و النعاس النهاري.
- ارتفاع ضغط الدم0
- اضطراب نظم القلب0
- السكتات الدماغية0
- حوادث الطرق 0
- التعرق الليلي.
- كثرة التبول الليلي.
- الاضطراب العقلي (اضطراب الذاكرة، اضطراب الانتباه، الصداع).
- زيادة في العصبية0
- اضطراب الجنس (نقص في الشهية الجنسية).
- ارتفاع ضغط الشريان الرئوي0
- الوفاة المفاجئة أثناء النوم0

التشخيص:

يبدأ تدبير الحالة بالشك السريري وفق ما ذكر من الأعراض والعلامات، وأهم من ذلك هو وصف شريك النوم (الزوجة أو الزوج) للحالة. يحال بعدها المريض لإجراء دراسة النوم.

نصائح عامة لعلاج توقف التنفس الانسدادي

أولاً: على المريض عمل ما يلي:

- (1) تجنب النوم على الظهر. ولضمان ذلك يمكن لصق كرة صغيرة بالظهر قبل النوم يوميا.
- (2) العمل على تخفيض الوزن.
- (3) رفع مستوى الرأس قليلا عن باقي الجسم أثناء النوم.
- (4) تجنب الأدوية المهدنة والمنومة.
- (5) تجنب التدخين والمشروبات الكحولية.
- (6) الحرص على نظافة الأنف ويمكن استعمال بعض مزيلات الاحتقان موضعيا.

ثانياً: العلاج الطبي والجراحي:

- (1) مرضى توقف التنفس الانسدادي يمكنهم استعمال جهاز لضخ الهواء بقوة خلال الأنف أو الأنف والفم من خلال قناع يثبت على الوجه.
- (2) الأجهزة التي تدعم الإزاحة الأمامية لل فك السفلي0
- (3) في الأطفال عمليه استئصال اللوزتين واللحمية.
- (4) استئصال جزء من الحنك الرخو في حاله تضخم اللهاة أو تهدل سقف الحلق جراحيا.
- (5) في بعض الحالات قد يلزم إجراء جراحه لتعديل حجم اللسان ، أو وضع أداة لإبعاد الفكين وفتح الفم عند النوم.

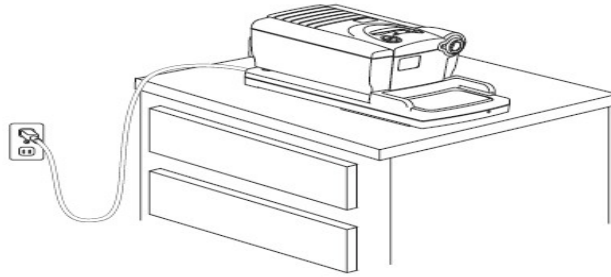
تعليمات لعلاج الخوف من استخدام كمامة جهاز ضخ الهواء المستمر لمجرى الهواء العلوى

الهدف من هذه الخطوات هو مساعدتك لتشعر بالراحة أثناء استخدامك لجهاز ضغط الهواء الإيجابي المستمر اللاتداخلي في فترات اليقظة و بالتالي يكون بإمكانك استخدامه بسهولة أثناء النوم (يجب عدم محاولة استخدامه أثناء النوم إلا بعد الراحة التامة أثناء استخدامه في فترات اليقظة) إذا كان جهازك مزود بزر (الرامب) فيمقدورك استعماله للحفاظ علي معدلات ضغط هواء منخفضة (0)

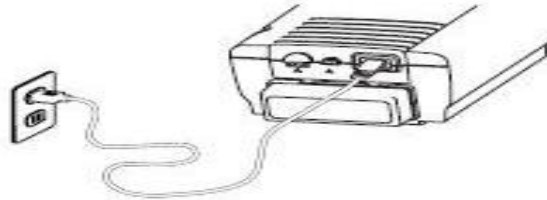
- 1- قم بتشغيل الجهاز ثم ضع القناع علي انفك (0) تمرن علي التنفس بانتظام من الأنف مع غلق الفم أثناء استعمال الجهاز في فترات اليقظة (0) أبدأ بفترات قليلة تتراوح من واحد إلي خمس دقائق ثم قم بزيادة الفترة بالتدريج (0)
 - 2- قم بتشغيل الجهاز ثم ضع القناع علي انفك مع لصق شرائط القناع علي الرأس (0) تمرن علي التنفس أثناء استعمال الجهاز في فترات اليقظة مع ارتدائه لفترات أطول لحين الوصول إلي فترة تتراوح بين خمسة عشرة دقيقة إلي عشرون دقيقة مع شعور تام بالراحة (0)
 - 3- يوضع القناع علي انفك بعد تشغيل الجهاز أثناء فترة القيلولة (0) ليس من المهم أن تنام ولكن الهدف الأساسي هو الشعور بالراحة أثناء رقادك علي الفراش (0)
 - 4- يوضع القناع علي انفك بعد تشغيل الجهاز أثناء النوم مساء (0)
- إذا شعرت بعدم الراحة أو الرهبة من استخدام الجهاز في أي خطوة من الخطوات السابقة عد للخطوة السابقة لها مع استخدام القناع لحين الشعور التام بالراحة ثم تقدم للخطوة التالية (0)

تعليمات لمستخدمي جهاز ضخ الهواء المستمر لمجرى الهواء العلوى

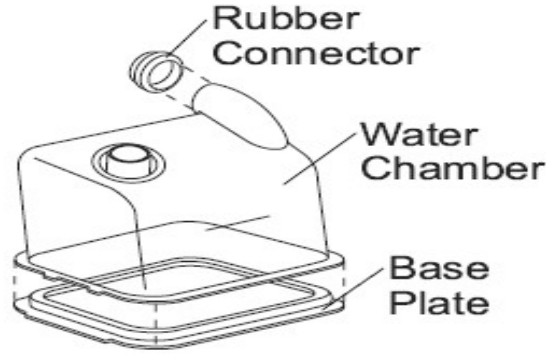
الوضع المثالي للجهاز:



- 1- يوضع الجهاز على سطح ثابت بعيدا عن جهاز التكييف أو المدفأة.
- 2- تجنب انسداد مدخل الهواء للجهاز وذلك بوجود أي شيء ملاصق لمؤخرة الجهاز و كذلك عدم وضع الجهاز فوق الوسادة أو أعلى مستوى رأس المريض.



- 3- تجنب وصل فيشة الكهرباء بمخرج كهرباء مشترك مع جهاز آخر كالثلاجة
- 4- استخدام ماء مقطر أو ماء معقم (مغلي لمدة نصف ساعة) لوحدة الترطيب بالجهاز



- 5- عدم تجاوز الماء لخط الامتلاء بوحدة الترطيب وكذلك عدم وضع أي أدوية بوحدة الترطيب.
- 6- تجنب دخول ماء بالجهاز لأن ذلك سيؤدى لتلف الجهاز .
- 7- تجنب إغلاق الفتحة المخصصة لهواء الزفير بالكمامة.
- 8- اربط الأربطة الخاصة بالكمامة بدرجة كافية فقط لمنع تسرب الهواء بين الكمامة والوجه .

تنظيف الجهاز:

- تنظيف يومي : غسل الكمامة ووحدة الترطيب وذلك بالماء والصابون (أو بالماء و الخل) تم تشطيف بالماء الفاتر و يترك للتجفيف في الهواء
- تنظيف أسبوعي : غسل الكمامة والأربطة و الخرطوم وحدة الترطيب و الفلتر وذلك بغمرها في الماء والصابون(أو الماء و الخل) لمدة نصف ساعة ثم تشطيف بالماء الفاتر و يترك للتجفيف في الهواء .
- ملحوظة : ممنوع استخدام الكحول أو الكلور في تنظيف الجهاز وكذلك عدم كوى الأربطة بعد التجفيف .
- تنظف الوحدة الرئيسية بالجهاز باستخدام قطعة قماش رطبه ثم قطعة قماش جافه مع عدم تشغيل الجهاز الا بعد التأكد من جفاف الجهاز تماما وكذلك عدم وضع الفلتر في الجهاز إلا بعد التأكد من جفافه تماما .

معلومات لمستخدمي جهاز ضخ الهواء المستمر لمجرى الهواء العلوى

- 1- ننصح باستخدام الجهاز أثناء النوم سواء بالليل أو بالنهار.
- 2- ننصح بتثبيت مواعيد النوم و اليقظة كلما أمكن .
- 3- ننصح بمراجعة الطبيب المعالج وذلك لمرضى ارتفاع ضغط الدم حيث أن ضغط الدم يقل باستخدام الجهاز .
- 4- يحدث تحسن في عدم الإشباع النومي و النعاس النهاري و كذلك الشخير بعد استخدام الجهاز لمدة 1-2 أسبوع .
- 5- يرجى استشارة الطبيب إذا عاد الشخير للظهور بعد اختفائه على الرغم من استخدام الجهاز وكذلك إذا حدث نقص أو زيادة في الوزن
- 6- يحدث انسداد بالأنف مع العطس في بداية استخدام الجهاز و تختفي في الغالب بعد مضي شهر من الاستخدام .
- 7- لابد من اصطحاب المريض للجهاز إذا ادخل المريض المستشفى لغرض العلاج أو الجراحة .

تحذيرات الأمان

- 1-ممنوع تنظيف الجهاز بوضعه في الماء.
- 2-ممنوع توصيل الكهرباء للجهاز إذا كان ميتل أو رطب.
- 3-ممنوع توصيل الكهرباء للجهاز من مخرج للكهرباء مشترك مع جهاز آخر .
- 4-ممنوع توصيل الكهرباء للجهاز باستخدام سلك كهربائي غير المخصص للجهاز .
- 5-ممنوع محاولة إصلاح الجهاز إذا حدث به عطل .

Discussion

OSAS constitutes a significant problem as OSAS is frequent in general population, affecting more than 2% of adult females and 4% of males. Moreover, prevalence rates increase with age, with OSAS occurring in 30%-80% of elderly population. So, its treatment with CPAP; which is the gold standard; is important to abolish symptoms and avoid complications of OSAS. Yet, patients' use of CPAP is often less than optimal. Non adherence is a significant barrier to OSAS treatment. Adherence failure ranges from 5% to 89% in the first week to 6 months.

General advices are given to Patients as regard stopping smoking, avoiding alcohol and nocturnal sedatives, loss of weight and the effect of sleeping in supine position and nasal obstruction on OSAS. These advices aim for modification of patients' behavior which is considered as a corner stone in OSAS management.

Smoking has a determinant role in onset of respiratory diseases. Environmental Tobacco Smoke (ETS) is recognized as a risk factor for Obstructive Sleep Apnea Syndrome. The effects of smoking on the pathophysiology of OSA may include smoking-induced upper airway inflammation, stimulant effects of nicotine on upper airway muscles, and a "rebound effect" due to nightly short-term nicotine withdrawal, or all of the above. In addition, the coexistence of OSA and smoking may have more widespread implications for cardiovascular dysfunction in patients with OSA. OSAS might be responsible for the addiction to nicotine. Nightly nicotine withdrawal as well as other respiratory and pulmonary effects of smoking may result in sleep-disordered breathing, especially OSA. This is supported by a study carried out by *Kashyap et al., 2001* to

determine whether there is a higher prevalence of smoking in patients with OSA compared with patients who do not have OSA. The prevalence of smoking in patients with OSA was found to be 35%, whereas it was only 18% in patients without OSA. Current smokers were found to be 2.5 times more likely to have OSA than former smokers and nonsmokers combined, and 2.8 times more likely to have OSA than former smokers alone. Former smokers were not more likely than never smokers to have OSA.

Also, all patients with OSA should avoid alcohol, even during the daytime, because it can depress the central nervous system, exacerbate OSA, worsen sleepiness, and promote weight gain. This is in accordance to *Randerath et al., 2011* who reported the effect of alcohol consumption on OSA which was illustrated by a series with seven patients who had varying degrees of upper airway obstruction during sleep, ranging from snoring alone to OSA. Following alcohol ingestion, the duration and frequency of obstructive respiratory events and the degree of oxyhemoglobin desaturation increased in five patients (71 percent). Two patients who had snoring alone at baseline developed frank OSA after alcohol ingestion.

The patients are also advised to avoid nocturnal sedatives and sleeping tablets as if they are used, they worsen the OSA. Certain medications that inhibit the central nervous system, such as benzodiazepines and benzodiazepine receptor agonists, as well as barbiturates, other anti-epileptic drugs, antidepressants, antihistamines, and opiates should be avoided. When these medications are felt to be necessary despite the patient's OSA, their use should be monitored closely and the dose carefully titrated if possible. However, Short acting

non benzodiazepine benzodiazepine receptor agonist can be used like Zolpidem and zaleplon according to *Veasey et al., 2006*. Also, ezopiclone can be used to help in increasing acceptance of the patient to CPAP. This is confirmed by *Danny et al., 2012* who hypothesized that eszopiclone would increase the arousal threshold and lower the AHI in patients with a low arousal threshold. Following a baseline overnight polysomnogram with an epiglottic pressure catheter to quantify the arousal threshold, 17 OSA patients, without major hypoxaemia [nadir arterial blood oxygen saturation (SPO₂) >70%], returned on two additional nights and received 3 mg of eszopiclone or placebo immediately prior to each study. Compared with placebo, eszopiclone significantly increased the arousal threshold [placebo: -14.0 (-19.9 to -10.9) compared with eszopiclone: -18.0 (-22.2 to -15.1) cmH₂O; *P* < 0.01], and sleep duration, improved sleep quality and lowered the AHI without respiratory event prolongation or worsening hypoxaemia. Among the eight patients identified as having a low arousal threshold, reductions in the AHI occurred invariably and were most pronounced (25 ± 6 compared with 14 ± 4 events/h of sleep; *P* < 0.01). They concluded that eszopiclone increases the arousal threshold and lowers the AHI in obstructive sleep apnoea patients that do not have marked overnight hypoxaemia. The greatest reductions in the AHI occurred in those with a low arousal threshold. The results of this study suggest that certain sedatives may be of therapeutic benefit for a definable subgroup of patients to help them for better acclimatization to CPAP.

Advice regarding body weight and its interaction with OSA also is provided as it is known that weight loss improves overall health, decreases the AHI, improves quality of life, and probably decreases daytime sleepiness. *Greenburg et al., 2009* found that bariatric surgery

was associated with a significant decrease in the BMI (from 55 to 38 kg/m²) and reduction in the mean AHI (from 55 to 16 events per hour of sleep).

According to *Tuomilehto et al., 2009* who reported the effects of weight loss on OSA which was illustrated by a trial that enrolled 72 consecutive overweight patients with mild OSA. The patients were randomly assigned to receive a single session of general nutrition and exercise advice, or a more intensive program that included a low calorie diet for three months plus nutrition and exercise counseling for one year. Patients in the latter group had significantly greater weight loss, reduction in the AHI, and improvement in quality of life compared to the control group.

Patients are informed about the impact of sleeping position on sleep apnea severity as we found that some patients are observed to have OSA that develops or worsens during sleep in the supine position while reviewing the data of polysomnography. This is supported by *Oksenberg et al., 2010* who demonstrated the effect of body position on REM-related obstructive sleep apnea (OSA) patients by a retrospective study performed on 100 adult OSA patients. They concluded that during REM sleep, the supine position is associated with increased frequency but not increased duration of apneas and hypopneas. These body position effects prevail over the differences between REM-related and Not-REM-related OSA patients.

Rapid treatment of nasal obstruction is viewed as an adjunct to CPAP therapy, potentially improving adherence. According to *Nakata et*

al., 2005, nasal resistance affects CPAP use and the initial acceptance of the device.

The patient's bed partner should be involved in the CPAP treatment process as their acceptance will support CPAP therapy and they provide feedback regarding the elimination of symptoms such as snoring. Also, they have important role in encouraging continued adherence with treatment. This is supported by *Cartwright, 2008* who found that CPAP users who live alone are significantly less likely to use their CPAP than those who live with someone. Spouse or bed partner sleep disturbance and sleep quality are important to patients' CPAP adherence behaviors. Patients who were more adherent to treatment had spouses or bed partners who had better sleep quality.

We recommend that the CPAP exposure therapy protocol should be implemented prophylactically to prevent anticipated claustrophobia, as prior to the diagnosis of sleep apnea some patients express a concern about being unable to tolerate CPAP on the night of their sleep titration study. Prophylactic therapy is better than treatment after having bad experience about CPAP as if patient had bad experience of CPAP or developed claustrophobia, de-conditioning and exposure therapy will be more difficult than prophylactic therapy. However prophylactic therapy takes longer time, more cost and effort but it is found that it has better results in improving adherence to CPAP therapy. Based on interviews with adherers and non-adherers carried out by *Terri et al., 2010*, it was found that adherence to CPAP was common among users who subjectively experienced initial benefit from the treatment, had positive experiences during the titration, and perceived that they received thorough, necessary information from their provider. In contrast, the

investigators suggest that the non adherent group experienced no subjective improvement with CPAP treatment, were less satisfied with their experience during CPAP titration polysomnogram, and reported a lack of anticipatory guidance with regard to the experience during CPAP titration polysomnogram.

It has been shown that not only the initial CPAP experience is important to adherence, but also the benefit perceived on the first night of treatment. The evidence suggests that the technological aspects associated with polysomnography and treatment delivery is less important in promoting adherence than a supportive environment and first impressions of ease of use and benefit of therapy according to *Drake et al., 2003*.

We can't primarily rely on modern technology of some devices such as auto-CPAP, RAMP, humidity and flexible pressure for improving adherence to CPAP. This is supported by *Resta et al., 2004* who performed a randomized, single blinded study on twenty OSAS patients after polysomnographic study with CPAP titration received either an automatic or a fixed level CPAP machine. At the end of the home treatment period (after one month of therapy) polysomnography was repeated while CPAP was administered by the same machine used at home. The correction of sleep respiratory disturbances and of sleep structure were satisfactory in both groups. No difference in any polysomnographic variable or in subjective sleepiness was found at re-evaluation. Patients with OSAS preferred auto-CPAP over fixed CPAP in the initial phase of therapy. The effectiveness auto-CPAP in improving major outcomes is equivalent to fixed CPAP.

Since auto-CPAP does not require initial titration, it is a simple and promising modality for sleep apnea home therapy. However, There are limitations for use of Auto-CPAP as *Morgenthaler et al., 2008* recommended that patients with congestive heart failure are not currently candidates for auto-PAP titration or treatment. In CHF, central sleep apnea (CSA) is common as it occur in about 47% of OSAS patients. Auto-CPAP can't abolish central sleep apnea as its sensors can't identify central apnea only obstructive events can be identified. Recently, some Auto-CPAP tracking system can differentiate CSA from OSA but not accurate like polysomnography so practice parameters don't recommend auto-CPAP. BPAP is appropriate initial therapy for patients with coexisting central sleep apnea or significant hypoventilation.

Boudewyns et al., 1998 published a case report describing the appearance of central apneas occurring during APAP titration. The apneas occurred as pressure was increased. Some of the events seemed to occur post arousal. Of note, central apneas may also occur during manual CPAP titration. However, central apneas could result in APAP devices delivering a progressive increase in pressure. This action may not be effective in inducing a resolution of these events. If excessive pressure triggers arousals, this action could cause central apnea in some patients.

A rather new device seems helpful with subjects suffering from periodic breathing of the Cheyne-Stokes type. The device auto-set pressure and oscillates around this pressure on small time bases, depending the frequency of the periodic breathing, however, it is not accurate.

Patients with significant lung disease such as chronic obstructive pulmonary disease, patients expected to have nocturnal arterial oxyhemoglobin desaturation due to conditions other than OSA (e.g., restrictive lung disease, obesity hypoventilation syndrome) might also potentially have problems during unattended Auto-CPAP titrations. These patients can desaturate during sleep in the absence of apnea or hypopnea, especially during REM sleep. Treatment with supplemental oxygen in addition to positive pressure or switch to bi-level pressure may be needed. This would not be available during an unattended APAP titration. Also, presence of hypercapnea in very severe COPD and obesity hypoventilation syndrome can't be abolished by use of auto-CPAP whose tracking system can't identify or treat hypercapnea. Thus, it seems reasonable to expect that patients with significant heart or lung disease as well as OSA may have problems with automated titrations.

Patients who do not snore (either naturally or as a result of palate surgery) are not currently candidates for auto-PAP titration or treatment. They should not use an auto-PAP device as snoring is one of the parameters that its' tracking system algorithm rely on.

Heated humidification doesn't improve adherence to CPAP, but it is one of the methods used for improving comfort of the patient during CPAP use. This is supported by *Mador et al., 2005* who performed a randomized controlled trial on ninety-eight patients with obstructive sleep apnea who had not received nasal CPAP previously. Patients received heated humidification at CPAP initiation in the treatment group. In the control group, patients could receive heated humidification only if they had upper airway symptoms that could not be treated successfully with simpler measures. There was no difference in CPAP compliance between

groups. Quality of life and subjective sleepiness improved in both groups with nasal CPAP therapy, but there was no difference in the extent of improvement between groups. The overall CPAP side effect score was similar in both groups, but individual symptoms of dry nose and dry mouth and throat were significantly lower in the heated humidification group. The addition of heated humidification when nasal CPAP was instituted did not lead to better adherence, greater improvement in sleepiness, or improved quality of life, but was associated with fewer symptoms attributable to the upper airway.

CPAP devices with the option of flexible pressure delivery (e.g., C-Flex) are thought to provide an improved degree of comfort and result in comparable therapeutic adherence with standard CPAP efficacy. This is supported by *Diana et al., 2009* who performed an international, multisite, single-blinded study with participants randomized to either C-Flex or CPAP. C-Flex and CPAP groups were comparable on baseline measures, achieved comparable AHI on titration, and had comparable PAP pressure requirements. C-Flex users had comparable average hours of use per night and total nights of use across the study, but had a trend toward achieving greater total hours of utilization ($p < .07$). While both groups had comparable decreases in sleepiness, C-Flex users reported on visual analog scales greater comfort (64.3 vs. 57.4; $p = .01$). C-Flex has comparable resolution of respiratory indices and adherence. Furthermore, C-Flex users reported greater mask comfort.

After diagnosis of OSAS, face to face contacts are recommended at 0, 7, 30, 60 days and approximately 12 months. Patient education about the nature, complications and treatment of OSA with CPAP is an important component of all treatment strategies. Patient education is easy,

cheap, applicable, have great impact on improving adherence of patient to PAP therapy.

We recommend to use the standard support (in the form of the usual care for newly diagnosed OSA patients including verbal explanation for the treatment "the CPAP device, interface and any accessories supplied. Information about cleaning and safety should be included", a 20- minute educational video in Arabic language, a 20-minute acclimatization to CPAP during waking hours, written notes in Arabic "about OSAS symptoms, diagnosis, treatment, how to deal in case of claustrophobia and instructions about CPAP ideal usage, safety and cleaning" and follow up on days 7, 30, 60 and 6 months) as it is more applicable, easy, matches our facilities than intensive support (in the form of the standard support, with CPAP education provided in the participants' homes with partners, 2 additional nights of CPAP titration in the sleep center for CPAP troubleshooting during initial CPAP exposure, and home visits by sleep nurses after 7, 14, and 28 days as well as after 4 months) which is more expensive, time and effort consuming and not easily applicable as it needs large number of educated, well-trained sleep technicians, nurses and much more facilities in sleep laboratories. However, it has better results according to *Chervin et al., 2000* who found that patients receiving intensive support have a higher likelihood of CPAP usage than standard support patients do. In the future, after availability of a reasonable number of trained technicians and nurses in our unit, the intensive support would be applied.

There is significant data suggesting that early intervention with CPAP therapy may be best and that long-term adherence to PAP is often established early in the course of treatment according to *Sin et al., 2002*.

So, we recommend early follow up after 7 days of the first session. This is supported by a study carried out by *Weaver et al., 1997* which showed that skipping CPAP for two or more nights within the first week of treatment signals potential for non-adherence and emphasizes the need for close follow-up during this period of time. The first week to month of home therapy appears to be the most critical phase for intervention and securing long-term compliance.

All reasonable attempts should be done to minimize mask and mouth leak by the selection of appropriate interfaces and the use of appropriate accessories. We recommend that the interface fit should be assessed while the patient is lying down in supine and lateral postures. The patient is given the opportunity to try a variety of CPAP interfaces to ensure optimal fit and comfort and minimal leak. Experienced side effects of CPAP by the patient such as mask leak that may cause skin or eye irritation may lead to non-adherence to therapy.

The importance of educational program including educational Arabic video is supported by *Wiese et al., 2005* who reported that a 15 min educational video during the initial visit improved the return rate to a CPAP clinic after 1 month. Also, *Adriana et al., 2012* evaluated CPAP compliance in 95 OSA patients that participated in the educational program and in 93 patients that did not. The program provided information and training, supported by video sessions and outpatient visits with the nurse at 7, 15, 30, 60 and 90 days after CPAP initiation. The control group did not receive any information or training. Both groups were evaluated at each visit, using the general sleep and Epworth Sleepiness Scale (ESS) questionnaire, for adherence and side effects of CPAP. CPAP adherence was significantly higher in the educational

program group compared with the control group as regard % of adherence and number of CPAP usage hours/night (71 vs. 56%, $p=0.02$; and 6.3 ± 1.9 vs. 5.1 ± 1.7 hrs/night, respectively, $p=0.01$) at the 90th day. In the educational program group, regular CPAP users had lower ESS scores after 90 days of CPAP therapy compared with irregular users (7.1 ± 4.9 vs. 10.2 ± 5.3 , respectively; $p=0.007$).

Motivational enhancement therapy (MET) is recommended as a line for treatment of OSAS patients for motivating adherence to CPAP. It's aims at maintenance of positive health behaviors. It is easily applied, of no cost, has been proved to improve adherence of patient to PAP therapy.

This is supported by studies done by *Aloia, et al., 2005* which show that MET has been successfully used in increasing adherence to CPAP therapy.

A randomized clinical pilot trial in older adults with OSA, naïve to CPAP was done by *Aloia et al., 2001* to examine cognitive behavioral intervention. The intervention group received two-45 minute sessions, one-on-one, that provided participant-specific information about OSA, symptoms, performance on cognitive tests, treatment relevance, goal development, symptom change with CPAP, troubleshooting advice, treatment expectations, and treatment goal refinement. The investigators suggested that providing individualized education and information influences self-efficacy and decisional balance and thereby enhances CPAP adherence. The control group received a placebo intervention consisting of two-45 minute sessions of general information about sleep, sleep architecture, and patient opinions regarding the sleep clinic

experience. No difference in CPAP use was observed at 1 and 4 week. However, at 12 week, the experimental group used CPAP for 3.2 hours more than the control group with a large effect size. This study suggests that an intervention based on cognitive behavioral constructs potentially influences CPAP adherence behaviors over time.

Follow up of the patient is done subjectively by Self-reported sleep diaries, the Epworth sleepiness scale and phone calls and objectively by reading the built-in time counter of the CPAP machine (run hours) or the effective use recorded using a pressure monitor coupled with a microprocessor (usage hours). Trying to estimate daily use time by simply asking how many hours a night the patient uses the device generally results in a considerably overestimation of actual mean treatment time per night because the patient is likely to provide the number of hours of CPAP use solely for the nights it was worn. This is supported by *Weaver et al., 2007* who reported that the objective data from the built in memory of the devices are vital. This is also supported by *Bazzani, 2007* who reported that objective monitoring of CPAP use has become the standard of care for managing patients with sleep apnea. The tracking systems are not limited to conventional CPAP alone, but also can be utilized in patients being treated with auto-CPAP, bilevel, auto-bilevel, or adaptive servo-ventilation.

Patients can be contacted via telephone within the first few weeks of CPAP set-up in order to discuss any concerns they may be having regarding to air pressure, mask fitting, leaks and other issues as they arise. They can also be contacted at the end of months 1, 2 and 3, and then quarterly thereafter, in order to receive any updates regarding pressure, leaks and mask fittings, and answer a list of questions to ensure

they are using their equipment properly. *Chervin et al., 1997* found that regular telephone support over a 2–3-month period in new or continuing CPAP users resulted in a 1.0–1.3-hour /night higher utilization compared with a usual-care group.

Sin et al., 2002 studied compliance monitoring, including consistent follow-up, “troubleshooting” and regular feedback to both patients and physicians, achieved CPAP compliance rates >85% over 6 months. There was a decrease in ESS score of 44% by 2 weeks of therapy and the patients continued to improve over the follow-up period, with the lowest mean ESS score observed at 6 months.

Tele-monitoring using a personal computer, an internet connection and the proprietary software allows tailored management of OSA with CPAP through monitoring of “time at prescribed pressure” and transmission of those CPAP compliance- and efficacy-relevant data to care providers in 24 hour cycles. It is believed that this technology is immensely useful in improving compliance and acceptance of the device in patients with sleep apnea according to *Sparrow et al., 2010*. However, it is not available to us due to high financial cost of equipments required, but we hope that it will be available in our locality in the near-future.

Summary

Obstructive sleep apnea (OSA) is a common chronic disorder that often requires lifelong care. It is estimated that 26 percent of adults are at high risk for OSA. The prevalence of OSA in the general population is approximately 20 percent if defined as an apnea hypopnea index (AHI) > fifteen events per hour. OSAS (more than 5 apneas, hypopneas, or RERAs per hour of sleep in a patient with symptoms or signs of disturbed sleep) affect more than 2% of adult females and 4% of males. Moreover, prevalence rates increase with age, with OSAS occurring in 30%-80% of elderly population

Cardinal features of OSAS in adults include Obstructive apneas, hypopneas, or respiratory effort related arousals (RERAS), daytime symptoms and signs attributable to disrupted sleep. Snoring and daytime sleepiness are the most common presenting complaints of OSA. Additional symptoms and signs include restless sleep, periods of silence terminated by loud snoring, poor concentration, nocturnal angina, and awakening with a sensation of choking, gasping, or smothering.

OSAS patients complain of various neuropsychiatric symptoms. Cognitive impairment and affective disorders such as depression are frequently encountered in OSA. In addition, a high prevalence of other psychiatric symptoms such as anxiety, somatization, attention deficit hyperactivity disorder (ADHD-type), and obsessive-compulsive symptoms have been reported in these patients. Nocturnal panic attacks, diverse parasomnias, delirium, psychosis, personality change, and violent outbursts can be also reported in some patients.

A simple questionnaire, Berlin Questionnaire is used for screening of OSAS. Also, the Epworth Sleepiness Scale (ESS), is a rapid screen to reveal excessive daytime sleepiness.

Full-night, attended, in-laboratory polysomnography is considered the gold-standard diagnostic test for OSA. It involves monitoring the patient during a full night's sleep. Patients who are diagnosed with OSA and choose positive airway pressure therapy are subsequently brought back for another study, during which their positive airway pressure device is titrated.

Positive airway pressure therapy is generally considered the first-line therapy for OSA. Continuous Positive Airway Pressure (CPAP) therapy is a highly effective treatment for OSAS, eradicating the airway closure during sleep and thereby reversing the daytime effects of OSAS. Yet, patients' use of CPAP is often less than optimal. Non adherence to the treatment is a significant problem.

For patients with mild or moderate OSA who prefer an oral appliance, it is initiated rather than positive airway pressure. This is based on recognition that most patients prefer an oral appliance, adherence is an essential aspect of successful treatment, both modalities are effective compared to no treatment or a sham treatment, and both modalities have a similar effect on symptoms.

Uvulo-palato-pharyngo-plasty (UPPP) or an alternative surgical therapy are considered when positive airway pressure or an oral appliance is declined, ineffective (after at least a three month trial of therapy), or not an option.

Initiation of CPAP treatment should include general advice on lifestyle and medical issues which may impact on the success of CPAP treatment. Overweight or obese patients should be encouraged to lose weight. Patients with positional OSA should change their sleep position. All patients should abstain from smoking and alcohol and avoid medications that may worsen their OSA such as sedatives, hypnotics and anti histaminic drugs.

Adherence to CPAP is considered as regular use of the CPAP machine; however, the precise frequency of use to attain therapeutic effect is unknown. Recommended use is between 6-8 h per night, but researchers have defined CPAP adherence as anywhere from an average of 4 h a night for 70% of nights.

There seem to be a dose-response relationship between CPAP usage and reductions in severity indices. Adherence failure is defined as use of CPAP for less than 4 hours/night in at least 70 percent of nights and /or lack of symptomatic improvement. It is not surprising that reports of CPAP adherence range from as low as 28% to more than 83%.

Adherence to CPAP can be assessed by subjective methods (assess the compliance that the patient declares through self-report, questionnaires and phone calls) and objective methods (usage hours calculated from the built-in time counter of the device). Self-reports are an inaccurate tool to determine compliance with CPAP. Objective monitoring of CPAP use has become the standard of care for managing patients with OSAS.

Modern technology has provided a variety of tools (compliance meters and data-card tracking devices) and options that may measure,

assist, track and enhance compliance, and expedite the collection and analysis of these data. Current CPAP devices can be interfaced with a computer in the office or home setting to download data. Newer modems can interface with the CPAP unit and the integrated chip to facilitate the reporting of remote data and reduce the need for face-to-face visits.

Telemedicine involves the provision or support of direct clinical care via the application of electronic and communications technology, including the remote monitoring of health status. Reports can be generated to show usage information and then forwarded electronically to referral labs or physicians without generating additional paperwork.

There is not any single factor that has been identified as consistently predictive of CPAP adherence. They include patient characteristics (Age, sex, marital status, and socio-economic status), disease characteristics (disease severity, as measured by AHI and nocturnal hypoxaemia), technological factors (approximately two thirds of patients will experience side effects from CPAP such as skin irritation, nasal stuffiness, eye puffiness, or gastric fullness), initial CPAP exposure factors (bad experience and claustrophobia) and psychosocial factors. Yet, the findings from the studies suggest that a multiplicity of factors that are highly variable between individuals, are predictive of CPAP adherence.

The following factors have been associated with better long-term adherence with CPAP therapy. They include adherence with CPAP during the first week of therapy, increased self-reported daytime sleepiness (ie, an Epworth Sleepiness Scale score >10) plus moderate to severe OSA (ie, an AHI between 15 and 30 events per hour of sleep), low

nasal resistance, CPAP titration via attended polysomnography, a nasal pillows interface, certain psychological traits (including lack of claustrophobic tendencies, presence of problem solving skills, optimism regarding the benefit of CPAP therapy, and self-efficacy), the patient made the decision to seek medical attention.

There is increasing number of intervention studies aimed at promoting CPAP adherence. These investigations can be categorized as supportive, educational, cognitive behavioral, or mixed strategy based on their reported content, methods, and theoretical framework. Supportive interventions are described as “reinforcement,” support, and/or enhanced access to sleep-specific, healthcare resources. Educational interventions focus on enhancing patient knowledge relative to the diagnosis and treatment of OSAS. Cognitive behavioral intervention strategies are explicitly described as such, theoretically-derived, and delivered by expert interventionists. Cognitive behavioral therapy (CBT) is a psychotherapeutic approach that addresses dysfunctional emotions, maladaptive behaviors and cognitive processes and contents through a number of goal-oriented, explicit systematic procedures. The name refers to behavior therapy, cognitive therapy, and to therapy based upon a combination of basic behavioral and cognitive principles and research. Most therapists working with patients dealing with anxiety and depression use a blend of cognitive and behavioral therapy. This technique acknowledges that there may be behaviors that cannot be controlled through rational thought. CBT is "problem focused" (undertaken for specific problems) and "action oriented" (therapist tries to assist the client in selecting specific strategies to help address those problems. Finally, mixed strategy describes a combination of support, education and cognitive behavioral therapy.

Several behavioral interventions have been shown to improve CPAP adherence such as frequent contact and follow-up with the health care provider, intensive patient support, and cognitive behavior therapy (CBT) plus education.

Motivational Enhancement Therapy (MET) has been successfully used in increasing adherence to CPAP therapy. It is an intervention that directly targets the constructs of readiness, importance, and confidence. It has been used successfully to alter behaviors. The provider does not directly advocate for behavior change, but rather asks key questions to help the patient explore his conflicted feelings about change, weigh the pros and cons of change, and allow the patient to realize the discrepancy between the present risky behavior and the patient's self-identified goals and values. This lack of direct advocacy is important when it comes to CPAP adherence. With MET the provider may use different methods such as open-ended questions and reflections to clarify the patient's concerns or strategic reflective listening.

Exposure therapy is indicated for individuals with sleep apnea who are unable to tolerate CPAP devices due to anxiety reactions. It should be implemented prophylactically to prevent anticipated claustrophobia. Some patients with prescribed CPAP therapy experience claustrophobia, anxiety, or panic symptoms related to wearing the mask (feeling restricted) and/or tolerating the air pressure (feeling suffocated). Exposure therapy for CPAP-related claustrophobia is a short-term behavioral intervention that typically can be delivered effectively in one to six sessions over 1–3 months.

The sleep clinician shall contact the patient at agreed intervals throughout the first year of treatment. A minimum of four such contacts shall occur. It is suggested that approximately 7, 30, 60 days and approximately 12 months after treatment initiation are appropriate times. It is preferred that all contact be face-to-face, but where this is not possible due to constraints of distance, for example, telephone consultations should be conducted. Where problems are being experienced, face-to-face contact should be arranged.

Conclusion

After reviewing the literature concerned with adherence to CPAP in OSAS, we can conclude that:

- (1) OSAS constitutes a significant problem. OSAS is frequent in general population, affecting more than 2% of adult females and 4% of males. Moreover, prevalence rates increase with age, with OSAS occurring in 30%-80% of elderly population.
- (2) CPAP is generally considered the gold standard for treatment of OSAS. Yet, patients' use of CPAP is often less than optimal. Non adherence is a significant barrier to OSA treatment. Adherence failure ranges from 5% to 89% in the first week to 6 months.
- (3) There are different methods of intervention for promoting CPAP adherence. These interventions include supportive measures, Motivational Enhancement Therapy and Exposure Therapy. They are all essential for successful integrated protocol for adherence.
- (4) Strict medical supervision for all patients of OSAS using CPAP, especially during the first month of use, is essential to accomplish effective adherence to CPAP.

Recommendation

1. General advices are given to Patients as regard stopping smoking, avoiding alcohol and nocturnal sedatives, advice for weight loss, avoiding sleeping in supine position and rapid treatment of nasal obstruction.
2. The patient and his/her spouse should be involved in the CPAP education and treatment process.
3. The CPAP exposure therapy protocol should be implemented prophylactically to prevent anticipated claustrophobia.
4. Face to face contacts are recommended at 0 ,7, 30, 60 days and approximately 12 months and on need.
5. For improving CPAP adherence, the use of the standard intervention protocol is recommended.
6. Follow up of the patients is mandatory which is done subjectively by (Self-reported sleep diaries, the Epworth sleepiness scale and phone calls) and objectively by (assessment of the effective use (usage hours) through reading the built-in time counter of the device).

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الملخص العربي

الهدف من العمل :

الهدف من هذا العمل هو دراسة العوامل المتعددة التي تؤثر على الالتزام باستخدام جهاز ضغط الهواء الايجابي المستمر في علاج متلازمة توقف التنفس الانسدادي أثناء النوم والبروتوكولات المختلفة المستخدمة لتحسين الالتزام، و من ثم، سوف يتم استخراج البروتوكول المقترح لمتابعة الالتزام باستخدام جهاز ضغط الهواء الايجابي المستمر للمرضى المترادين لعيادات اضطرابات التنفس أثناء النوم بالمنصورة .

الملخص العربي

تعد متلازمة توقف التنفس الانسدادي أثناء النوم من الاضطرابات المزمنة الشائعة التي عادة ما تحتاج إلي عناية مدى الحياة. وتشير التقديرات إلى أن ستة و عشرين في المائة من البالغين يكونون عرضة للإصابة بها. ويعتبر معدل انتشار المتلازمة بين عموم السكان ما يقرب من عشرون في المائة في حالة تعريفه على أن مؤشر توقف التنفس أثناء النوم أكثر من خمسة أحداث في الساعة، حيث يصيب أكثر من اثنان بالمائة من الإناث البالغات، و أربعة بالمائة من الذكور. وعلاوة على ذلك، تزداد معدلات الانتشار مع تقدم العمر، و التي تتراوح بين ثلاثين إلى ثمانين بالمائة من السكان المسنين.

تشتمل السمات الأساسية لمتلازمة توقف التنفس الانسدادي أثناء النوم في البالغين على توقف التنفس الانسدادي، ضعف التنفس، أو الاستيقاظ المتكرر المصاحب للمجهود التنفسي وعلی أعراض وعلامات أثناء النهار والتي تعزى إلى اضطراب النوم.

ويعتبر الشخير والنعاس أثناء النهار من الشكاوى الأكثر شيوعا المصاحبة لهذه المتلازمة .

هناك أعراض وعلامات إضافية مثل النوم غير المريح، وفترات الصمت التي تنتهي بالشخير بصوت عال، و ضعف التركيز، والذبحة الصدرية الليلية، والاستيقاظ مع الإحساس بالاختناق، اللهاث، أو الموت المفاجئ.

وغالبا ما يشتكي مرضى متلازمة توقف التنفس الانسدادي أثناء النوم من أعراض عصبية ونفسية مختلفة تشمل ضعف الإدراك واضطرابات عاطفية مثل الاكتئاب. وبالإضافة إلى

ذلك، أشارت التقارير إلي ارتفاع معدل انتشار أعراض نفسية أخرى مثل القلق، الجسدية، اضطراب نقص الانتباه وفرط النشاط، وأعراض الوسواس القهري في هؤلاء المرضى. كما أشارت أيضا إلي إمكانية حدوث نوبات الذعر الليلية، خطل نموي ، هذيان، والذهان، ونوبات عنف و تغير في شخصية بعض المرضى.

ويستخدم استبيان برلين للكشف عن المتلازمة . كما يستخدم مقياس ايبورث للنعاس للكشف السريع عن النعاس المفرط أثناء النهار.

يعد النوم لليلة كاملة في مختبر دراسة النوم وإجراء الرسم البياني المتعدد المشاهد لاختبار اضطرابات النوم هو الاختبار التشخيصي الأمثل لمتلازمة توقف التنفس الانسدادي أثناء النوم حيث أنه ينطوي على مراقبة المريض خلال ليلة كاملة من النوم ثم يتم استدعاء المرضى الذين تم تشخيصهم واختيار جهاز الضغط الهوائي الإيجابي المستمر كوسيلة لعلاجهم في وقت لاحق مرة أخرى لعمل دراسة أخرى، يتم خلالها معايرة الجهاز على الضغط الهوائي الفعال (0

ويعتبر العلاج بالضغط الهوائي الإيجابي عموما هو الخط الأول لعلاج متلازمة توقف التنفس الانسدادي أثناء النوم. يعد جهاز الضغط الهوائي الإيجابي المستمر علاج فعال للغاية للمتلازمة حيث يقوم بالقضاء على انغلاق مجرى الهواء أثناء النوم، وبالتالي عكس آثار المتلازمة خلال النهار. ومع ذلك، فإن استخدام المرضى للجهاز في كثير من الأحيان يكون أقل من المطلوب حيث يعد عدم الالتزام بالعلاج مشكلة كبيرة.

وتستخدم الأجهزة عن طريق الفم للمرضى المصابين بالمتلازمة بدرجة طفيفة إلي معتدلة والذين يفضلون الأجهزة عن طريق الفم بدلا من جهاز الضغط الهوائي الإيجابي المستمر. ويستند هذا على التسليم بأن معظم المرضى يفضلون الأجهزة عن طريق الفم، و يعد الالتزام جانب أساسي من العلاج الناجح، ويعتبر كلا من الوسيلتين فعالا بالمقارنة مع عدم المعالجة أو العلاج الصوري، وكلتا الوسيلتين لهما تأثيرا مماثلا على الأعراض.

و تؤخذ في الاعتبار جراحة الرأب بشكل اللهاة و الحنك والبلعوم أو العلاج الجراحي البديل عند رفض جهاز ضغط الهوائي الإيجابي أو الأجهزة عن طريق الفم أو عندما تكون غير فعالة (على الأقل بعد تجربة ثلاثة أشهر من العلاج) أو أن تكون هذه الأجهزة ليست خيارا.

وينبغي أن يشتمل العلاج بجهاز الضغط الهوائي الإيجابي المستمر علي نصائح عامة حول نمط الحياة والقضايا الطبية التي قد تؤثر على نجاح العلاج حيث يجب تشجيع المرضى

الذين يعانون من زيادة في الوزن أو السمنة علي إنقاص وزنهم. كما ينبغي علي المرضى الذين يعانون من المتلازمة الموضعية تغيير وضع نومهم . يجب علي كل المرضى الامتناع عن التدخين والكحوليات وتجنب الأدوية التي قد تؤدي إلي تفاقم المتلازمة الخاصة بهم مثل المهدئات، المنومات والأدوية المضادة للهستامين.

ويعتبر الالتزام بجهاز الضغط الهوائي الايجابي المستمر هو الاستخدام المنتظم للألة، إلا أن التواتر الدقيق للاستخدام لتحقيق التأثير العلاجي غير معروف .حيث يوصى باستخدام الجهاز من ست إلي ثمان ساعات في الليلة، ولكن يعرف الباحثين الالتزام بجهاز الضغط الهوائي الايجابي المستمر علي أنه أربع ساعات من الليلة في المتوسط ولمدة سبعون بالمائة من الليالي.

يبدو أن هناك علاقة بين جرعة استخدام جهاز ضغط الهواء الايجابي المستمر والاستجابة من حيث الانخفاض في شدة المؤشرات. ويعرف الفشل في الالتزام علي أنه استخدام جهاز الضغط الهوائي الايجابي المستمر لمدة تقل عن أربع ساعات في الليلة في سبعون في المائة من الليالي على الأقل و /أو عدم تحسن الأعراض .وليس من المدهش أن تتضمن التقارير عن مدى الالتزام بجهاز ضغط الهواء الايجابي المستمر علي نسب تتراوح بين ثمان و عشرين بالمائة إلي أكثر من ثلاثة و ثمانين بالمائة.

ويمكن تقييم الالتزام بجهاز الضغط الهوائي الايجابي المستمر عن طريق الوسائل الشخصية (تقييم مدى الامتثال أن يعلن المريض من خلال التقارير الذاتية، والاستبيانات والمكالمات الهاتفية) والطرق الموضوعية (ساعات الاستخدام وتحسب من خلال عداد الوقت المدمج في الجهاز) . تعد التقارير الذاتية أداة غير دقيقة لتحديد مدى الامتثال لجهاز ضغط الهواء الايجابي المستمر بينما تعد الملاحظة الموضوعية لاستخدامه هي معيار العناية في تدبير المرضى الذين يعانون من متلازمة توقف التنفس الانسدادي أثناء النوم .

وقد وفرت التقنية الحديثة مجموعة متنوعة من الأدوات (عدادات الامتثال وأجهزة متابعة على بطاقة البيانات) والخيارات التي قد تقيس، تساعد وتتابع وتعزز الامتثال والإسراع في جمع وتحليل هذه البيانات. وأجهزة ضغط الهواء الايجابي المستمر الحالية يمكن ربطها مع حاسوب في المكتب أو المنزل للإعداد لتحميل البيانات. ويمكن ربط أجهزة المودم الحديثة مع وحدة جهاز ضغط الهواء الايجابي المستمر وشريحة متكاملة لتسهيل الإبلاغ عن بيانات الاستشعار عن بعد وتقليل الحاجة للزيارات وجها لوجه.

يشتمل التطبيب عن بعد على توفير الدعم أو الرعاية السريرية المباشرة للمرضى عن طريق التطبيق لتكنولوجيا الالكترونية والاتصالات، بما في ذلك الرصد عن بعد للحالة الصحية . يمكن إنشاء تقارير لعرض معلومات الاستخدام ومن ثم إرسالها إلكترونياً إلى مختبرات الإحالة أو الأطباء دون إنشاء أوراق إضافية.

ليس هناك أي عامل منفرد تم تحديده على أنه التنبؤي على الدوام بالالتزام باستخدام جهاز الضغط الهوائي الايجابي المستمر. ولكن هناك عوامل متعددة وتشمل خصائص المريض (العمر، الجنس، الحالة الاجتماعية، والوضع الاجتماعي والاقتصادي)، خصائص المرض (شدة المرض، مقاسا بمؤشر توقف التنفس أثناء النوم ونقص تأكسج الدم الليلي)، والعوامل التكنولوجية (ما يقرب من ثلثي المرضى يعانون من الآثار الجانبية للجهاز مثل تهيج الجلد، احتقان الأنف، انتفاخ العين، أو الامتلاء المعدي)، والعوامل الأولية للتعرض للجهاز (التجربة السيئة والخوف من الأماكن المغلقة) والعوامل النفسية والاجتماعية. ومع ذلك، فإن النتائج المستخلصة من الدراسات تشير إلى تعدد العوامل التي تختلف اختلافا كبيرا بين الأفراد، هي التي تنبئ بالالتزام باستخدام جهاز ضغط الهواء الايجابي المستمر.

وقد ارتبطت العوامل التالية بتحسين الالتزام بالعلاج باستخدام جهاز الضغط الهوائي الايجابي المستمر على المدى الطويل. وهي تشمل الالتزام باستخدام الجهاز خلال الأسبوع الأول من العلاج، وزيادة النعاس خلال النهار المبلغ عنها ذاتيا (أي أن يكون معدل مقياس ايبورث للنعاس أكثر من عشرة) ، بالإضافة إلى أن تكون المتلازمة بدرجة معتدلة إلى شديدة (أي أن يكون مؤشر توقف التنفس أثناء النوم أكثر من ثلاثين حدث لكل ساعة من النوم)، وانخفاض مقاومة الأنف ، ومعايرة الجهاز عبر إجراء الرسم البياني المتعدد المشاهد لاختبار اضطرابات النوم في مختبر دراسة النوم ، واستخدام الوسائد الأنفية، وبعض الصفات النفسية (بما في ذلك عدم وجود نزعات الخوف من الأماكن المغلقة، ووجود مهارات حل المشاكل، التفاؤل بشأن الاستفادة من العلاج بالجهاز، والكفاءة الذاتية)، وقيام المريض باتخاذ قرار التماس الرعاية الطبية وتعاطم شدة متلازمة توقف التنفس الانسدادي أثناء النوم.

هناك عدد متزايد من الدراسات العلمية التداخلية تهدف إلى تشجيع الالتزام باستخدام جهاز ضغط الهواء الايجابي المستمر. ويمكن تصنيف هذه الدراسات كدراسات داعمة وتعليمية وسلوكية معرفية، أو استراتيجية مختلطة على أساس مضمونها ، والأساليب، والإطار النظري. وصفت التدخلات الداعمة باسم "التعزيز"، الدعم، و / أو تعزيز فرص الحصول على موارد

النوم الخاصة، والرعاية الصحية. تركز التدخلات التعليمية على تعزيز معرفة المريض المتعلقة بتشخيص وعلاج متلازمة توقف التنفس الانسدادي أثناء النوم. وتوصف استراتيجيات التدخل السلوكي المعرفي بوضوح على هذا النحو، من الناحية النظرية المشتقة، وما أدلى به الخبراء المتدخلون. وأخيراً، تصف الاستراتيجية المختلطة علي مزيج من الدعم والتعليم والعلاج السلوكي المعرفي.

وقد تبين أن العديد من التدخلات السلوكية تحسن الالتزام باستخدام الجهاز مثل الاتصالات متكررة والمتابعة مع مقدم الرعاية الصحية، والدعم المكثف المريض، والعلاج السلوكي المعرفي، بالإضافة إلى التعليم.

وقد استخدم العلاج بالتعزيز المحفز بنجاح في زيادة الالتزام بالعلاج عن طريق جهاز الضغط الهوائي المستمر. وهو عبارة عن التدخل الذي يستهدف بشكل مباشر ثوابت الاستعداد، الأهمية، والثقة. وقد تم استخدامه بنجاح لتغيير السلوكيات. لا يدعو المقدم لهذا العلاج مباشرة لتغيير السلوك، وإنما يسأل أسئلة أساسية مساعدة المريض على استكشاف مشاعره المتضاربة حول التغيير، ووزن إيجابيات وسلبيات التغيير، ويسمح للمريض بادراك الفرق بين السلوك المحفوف بالمخاطر الحالية و الأهداف والقيم التي تم تحديدها ذاتياً. وغياب هذا التناقض في الدعوة شيء مهم عندما يتعلق الأمر بالالتزام باستخدام الجهاز. ومع العلاج بالتعزيز المحفز يجوز للمقدم استخدام أساليب مختلفة مثل أسئلة مفتوحة وانعكاسات لتوضيح مخاوف المريض أو الاستماع الذي يعكس الإستراتيجية.

يستخدم علاج التعرض للأفراد المصابين بتوقف التنفس أثناء النوم غير القادرين على تحمل أجهزة الضغط الهوائي الايجابي المستمر بسبب تفاعلات القلق. وينبغي تنفيذ ذلك بشكل وقائي لمنع الخوف المتوقع من الأماكن المغلقة. بعض المرضى الموصوف لهم العلاج بجهاز ضغط الهواء الايجابي المستمر يعانون من الخوف من الأماكن المغلقة، والقلق، أو أعراض الهلع المرتبطة بارتداء القناع (الشعور بالقيء) و / أو التضايق عن ضغط الهواء (الشعور بالاختناق). علاج التعرض لحالات الخوف من الأماكن المغلقة المرتبطة باستخدام جهاز الضغط الهوائي الايجابي المستمر هو التدخل السلوكي على المدى القصير الذي عادة يمكن تنفيذه على نحو فعال في جلسة حتى ستة جلسات على مدى شهر إلى ثلاثة أشهر.

يتعين على الطبيب المختص بأمراض النوم الاتصال بالمريض على فترات متفق عليها خلال العام الأول من العلاج. ويجرى ما لا يقل عن أربعة من تلك الاتصالات. ويقترح أن تكون

أيام سبعة , ثلاثين, و ستين يوما واثني عشر شهرا تقريبا بعد بدء العلاج هي الأوقات المناسبة . ويفضل أن يكون كل اتصال وجها لوجه ولكن إذا كان هذا غير ممكن نظرا لضيق المسافة، على سبيل المثال، ينبغي إجراء مشاورات هاتفية . و حيثما كانت المشاكل متعودا عليها، ينبغي ترتيب الاتصال وجها لوجه.

الاستنتاج

بعد مراجعة المرجع المعني بالالتزام باستخدام جهاز الضغط الهوائي الايجابي المستمر لعلاج متلازمة توقف التنفس الانسدادي أثناء النوم ، يمكننا أن نستنتج ما يلي:

(1) تمثل متلازمة توقف التنفس الانسدادي أثناء النوم مشكلة كبيرة . فهي متكررة في عامة السكان، حيث تؤثر على أكثر من 2٪ من الإناث البالغات، و 4٪ من الذكور. وعلاوة على ذلك، تزيد معدلات انتشارها مع تقدم العمر، فتصيب ثلاثين إلى ثمانين بالمائة من السكان المسنين.

(2) يعتبر جهاز الضغط الهوائي الايجابي المستمر عامة هو المعيار الذهبي لعلاج متلازمة توقف التنفس الانسدادي أثناء النوم ومع هذا، فإن استخدام المرضى لهذا الجهاز في كثير من الأحيان يكون أقل من المستوى الأمثل. ويشكل الفشل في الالتزام عائقا كبيرا في علاج هذه المتلازمة. ويتراوح إخفاق الالتزام من خمسة بالمائة إلى تسعة و ثمانين بالمائة في الأسبوع الأول إلى ستة أشهر.

(3) هناك طرق مختلفة للتدخل من أجل تشجيع الالتزام باستخدام جهاز الضغط الهوائي الايجابي المستمر وتشمل هذه التدخلات التدابير الداعمة، العلاج بالتعزيز المحفز وعلاج التعرض. فكلها ضرورية لبروتوكول متكامل ناجح للالتزام.

(4) يعد الإشراف الطبي الدقيق لجميع مرضى متلازمة توقف التنفس الانسدادي أثناء النوم المستخدمين لجهاز الضغط الهوائي الايجابي المستمر، خاصة خلال الشهر الأول من الاستخدام، أمر ضروري لتحقيق الالتزام للجهاز بفعالية.

التوصيات

1. يتم إعطاء النصائح العامة للمرضى فيما يتعلق بالتوقف عن التدخين ، وتجنب الكحوليات والمهدئات الليلية، وفقدان الوزن وتجنب النوم في وضعية الاستلقاء والعلاج السريع لانسداد الأنف.
2. ينبغي إشراك شريك السرير للمريض في عملية العلاج بجهاز الضغط الهوائي الايجابي المستمر.
3. ينبغي تنفيذ بروتوكول علاج التعرض لاستخدام جهاز الضغط الهوائي الايجابي المستمر بشكل وقائي لمنع الخوف المتوقع من الأماكن المغلقة.
4. ينصح أن يكون الاتصال وجها لوجه في أيام 0، 7، 30، 60 يوما وحوالي 12 شهرا من بدء استخدام جهاز الضغط الهوائي المستمر.
5. لتحسين الالتزام باستخدام جهاز ضغط الهواء الايجابي المستمر نوصي باستخدام الدعم القياسي .
6. المتابعة للمريض " ويتم ذلك ذاتيا عن طريق يوميات النوم المبلغ عنها ذاتيا، مقياس إيپورث للنعاس والمكالمات الهاتفية وموضوعيا من خلال قراءة ساعات تشغيل أو الاستخدام الفعال (ساعات الاستخدام).