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Psychiatric Disorders in Patients With Obstructive Sleep Apnea Syndrome

Essay

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In Thoracic Medicine

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الاضطرابات النفسية فى مرضى متلازمة توقف التنفس الانسدادى أثناء النوم

توطئة للحصول على درجة الماجستير فى الأمراض الصدرية

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صفحة المشرفين ومساعديهم

نموذج رقم (١)

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List of Abbreviations

AASM	American Academy of Sleep Medicine
ADHD	Attention Deficit Hyperactivity Disorder
AEDs	Antiepileptic drugs
AHEAD	Action for health in Diabetes
AHI	Apnea hypopnea index
ARDS	Acute respiratory distress syndrome
BDI-I	Beck Depression Inventory I
BDI-II	Beck Depression Inventory II
BMI	Body mass index
CBT	Cognitive behavior therapy
CMS	Centers for Medicare and Medicaid Services
CNS	Central nervous system
CO2	Carbon dioxide
COPD	Chronic Obstructive Pulmonary Disease
CPAP	Continuous Positive Airway Pressure
CS	Conditioned stimulus
CS	Cycloserin
ECG	Electrocardiogram
ECT	Electroconvulsive therapy
ED	Erectile dysfunction
EDS	Excessive daytime sleepiness
EEG	Electroencephalography
EMG	Electromyogram
EPDS	Edinburgh Postnatal Depression Scale
ERV	Expiratory reserve volume
FOSQ	Functional Outcomes of Sleep Questionnaire

FOSQ-10	Modified Functional Outcomes of Sleep Questionnaire
ESS	Epworth sleepiness scale
fMRI	Functional magnetic resonance imaging
FSFI	Female Sexual Functioning Index
GAD-7	Generalized Anxiety Disorder 7
GDS	Geriatric Depression Scale
HADS	Hospital Anxiety and Depression Scale
HCO3	Bicarbonate
HRQOL	Health-Related Quality Of Life
HRT	Hormonal replacement therapy
HST	Home sleep testing
ICU	Intensive Care Unit
IIEF	International Index Of Erectile Dysfunction
IPF	Idiopathic pulmonary fibrosis
IR	Insulin resistance
KADS	Kutcher Adolescent Depression Scale
LAUP	Laser-assisted uvulopalatoplasty
MADRS	Montgomery-Asberg Depression Rating Scale
MDI	Major Depression Inventory
MDR-TB	Multidrug resistant tuberculosis
MET	Motivational enhancement therapy
MI	Motivational interviewing
MMA	Maxillary mandibular advancement
MMP	Modified Mallampati
MMSE	Mini-Mental Status Examination
MP	Mallampati
MWT	Maintenance of wakefulness test
nPAP	Nocturnal positive airway pressure

NREM	Non-rapid eye movement
OAs	Oral appliances
OCST	Out of centre sleep testing
ODI	Oxygen Desaturation Index
PAP	Positive airway pressure
PAS	Panic and Agoraphobia Scale
PDSS	Panic Disorder Severity Scale
PLMS	Periodic limb movements of sleep
PM	Portable monitoring
PR	Pulmonary Rehabilitation
PSG	Polysomnography
PAH	Pulmonary-arterial hypertension
PNB	Preliminary Neuropsychological Battery
PVT	Psychomotor Vigilance Task
QOL	Quality of life
RDI	Respiratory disturbance index
REM	Rapid eye movement
RERAs	Respiratory effort-related arousals
RLS	Restless leg syndrome
SaO2	Arterial oxygen saturation
SDB	Sleep disordered breathing
SIB	Severe Impairment Battery
SPIN	Social Phobia Inventory
SpO2	Pulse oximetry
SRI	Serotonin reuptake inhibitors
STOP-BANG	Snoring, Tired, Observed apnea, Blood Pressure, Body mass index, Age, Neck circumference, Gender
TB	Tuberculosis

UARS	Upper airway resistance syndrome
UCRs	Unconditioned responses
UCS	Unconditioned stimulus
UPPP	Uvulo-palato-pharyngo-plasty
WASO	Wake after sleep onset
WMH	White matter hyperintensities
WMS	Wechsler Memory Scale
Y-BOCS	Yale-Brown Obsessive Compulsive Syndrome

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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 ***(Shamsuzzamanet 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).***

Psychiatric manifestations are common on OSAS patients than in general population as it occur in 7%-63% for depression, 11%-70% for anxiety, 60% for impaired cognition, 24% for epilepsy in men and 9% for epilepsy in women,30%-50% for erectile dysfunction.

Treatment of psychiatric manifestations associated with OSAS will lead to better control of OSAS and better quality of life.

Aim of the work

The aims of this Essay are to study the symptoms and signs of psychiatric manifestations in OSAS for early diagnosis of these common confounding associations for better outcome.

Obstructive sleep apnea syndrome

History and definitions

The obstructive sleep apnea syndrome (OSAS) was first recognized as a significant health problem only over the last half of the 20th century. ***Burwell et al., 1956*** used the term pickwickian syndrome to describe individuals with obesity, hypersomnolence, hypercapnia, corpulmonale, and erythrocytosis. The term pickwickian was based on the character Fat Boy Joe from Charles Dickens' ***The past humorous Papers of the Pickwick Club (1837)***, who was markedly obese and tended to fall asleep uncontrollably during the day. The current terminology describing such individuals is the obesity hypoventilation syndrome (OHS).Such patients represent only 10% to 15% of the total number of patients with OSAS. ***Guilleminaultet al., 1976*** described the OSAS in patients with daytime sleepiness and obstructive apneas on polysomnography (PSG).

An apnea index of 5/hr or greater was considered abnormal ***(Guilleminault, 1985).***An apnea was defined as absent airflow at the nose and mouth for 10 seconds or more. Obstructive apneas are secondary to airway closure at a supraglottic location that reverses at apnea termination often associated with a brief awakening (arousal) ***(Remmers et al., 1978)***. Obstructive apneas are followed by a fall in arterial oxygen saturation (SaO2) of varying severity. It was soon realized the episodes of reduced airflow and tidal volume (hypopneas) that are the result of upper airway narrowing are also clinically significant ***(Gould et al., 1988).***

The definition of hypopnea has varied consider ably ***(Redline et al., 2000).***The American Academy of Sleep Medicine (AASM) scoring

manual ***(Iber et al., 2007)***recommends scoring apnea on the basis of an oronasal thermal sensor and hypopnea on the basis of nasal pressure monitoring. The recommended hypopnea definition requires a 30% reduction in nasal pressure signal for 10seconds or longer in association with a 4% or greater arterial oxygen desaturation. The alternative definition of hypopnea requires a 50% or greater reduction in the nasal pressure signal associated with either an arousal or a 3% or greater arterial oxygen desaturation. Patients with OSAS have variable proportions of obstructive, mixed, and central apneas as well as hypopneas.

Pathogenesis of the upper airway obstruction

The individual with OSAS often has a restless night's sleep because of repetitive arousals triggered by the central nervous system (CNS)**.**These arousals are a protective response to allow the airway patency to be restored. During sleep, air flow is compromised primarily because of the posterior displacement of the soft palate at the pharynx. The relaxation and displacement of soft tissues posteriorly cause an increase in upper airway resistance and negative intrathoracic pressure. The pharyngeal dilator muscles of the patient with OSAS fail to contract and maintain airway patency ***(Chung& Imarengiaye, 2002).***

The decrease or absence of air flow causes the individual's SaO2 to decrease, where as carbon dioxide (CO2) levels rise. Elevations in CO2 stimulate the CNS so the individual can arouse enough to overcome the airway obstruction, often with a forceful and inspiratory effort. Such efforts may be audible to the individual's family members as a loud snort. This results in improved airflow temporally. However, inspiratory efforts can be unsuccessful when excessive negative inspiratory pressures exceed

the dilator abductor muscle forces. The result is pharyngeal collapse and apnea. After having a night of poor quality sleep, day time hypersomnolence often results ***(Moos &Cuddeford,2006).***

During rapid eye movement (REM) sleep, there is a decrease in upper airway muscle activity, which leads to a narrowed or collapsed pharyngeal area for air flow**.** Therefore, natural REM sleep in the OSAS patient (even without anesthesia or opioids) can predispose the patient to an airway obstruction. In addition, patients with OSAS often have a more narrow and collapsible upper airway ***(McNicholas& Ryan, 2006).***

The sequence of obstruction, arousal, and sleep are repeated throughout the night, providing a fragmented, unrefreshing sleep, despite adequate hours spent asleep. In the perianesthesia phase, this pattern of airway obstruction is exacerbated because of the effects of sedation, opioids, and anesthetic agents**.** Even in the patient without OSAS, these medications can lead to respiratory depression, reduced ability to maintain airway patency, and inhibition of protective reflexes. However, in the patient with OSAS, opioids and sedatives can lead to decreased anterior–posterior diameter of the airway, as well as loss of muscle tone and pharyngeal collapse. Attempts by the patient to breathe against the obstructed airway can lead to secondary areas of pharyngeal collapse, potentially aggravating the airway obstruction ***(Tomlinson, 2007)***.

The recurrent loss of airway patency places increased demands on the cardiac, respiratory, and CNS. During the airway obstruction, there may be occurrences of bradycardia, hypoxia, hypercarbia, and decreased alveolar ventilation. The CNS is stimulated by the increase in CO2, which can trigger a period of tachycardia, or increased systemic arterial and pulmonary pressures. In addition to causing surges in blood pressure,

apneas can also trigger sympathetic over activity, or catecholamine release. These responses have been shown to cause both acute and long-term complications ***(Coccagna et al., 2006)***.

Risk factors of OSAS

1. Obesity

An association between AHI and obesity has been documented in many studies***(Young et al., 2002).***

Peppard et al., 2000 followed the effects of weight change on AHI. A 10% weight gain predicted an approximate 32% increase in the AHI. A10% weight loss predicted a 26% reduction in the AHI. A10% increase in weight was associated with a six fold increase in the risk of developing moderate to severe OSAS.

Davies et al., 1992 found that neck circumference correlated with AHI better than general obesity. However, a recent study found neck circumference to correlate best with AHI in women whereas abdominal girth correlated better in men***(Simpson et al., 2010).***

2. Male Sex

In the Wisconsin cohort study by ***(Young et al., 2002),***men had about twice the incidence of the OSAS compared with women (4% vs. 2%) (AHI ≥ 5/hr+ symptoms).

Young et al., 2002 determined the prevalence of OSAS and defined it as an AHI greater than 10/hr+ symptoms to be 3.9% in men and 1.2% in women.

3. Age

The prevalence of OSAS appears to be higher in the elderly than in middle-aged populations. The prevalence of a chronic nonfatal disease would increase as cases accumulate even if the incidence (new cases/yr) was constant or declining***(Ancoli-Israel et al., 1991).***A prevalence of OSAS, defined as an AHI greater than 10/hr, was found to be 62%. Here, hypopneas were based on changes in flow (based on two channels of respiratory inductance plethysmography) independent of changes in the SaO2 (no oximetry). This is a prevalence of about three times that in middle-aged populations. There is evidence from the Sleep Heart Health Study that the SDB prevalence increases from age 40 to around 60 ***(Young et al., 2002)***.

4. Postmenopausal Status

The Sleep Heart Health Study analysis of women older than 50 years found that postmenopausal women are at increased risk of developing OSAS if they are not on hormonal replacement therapy (HRT) treatment ***(Shahar et al., 2003).***

5. Ethnicity

An investigation by ***(Redline et al., 1997)***found an increase in the risk of having OSAS greater in African Americans than in whites only for those younger than age 25 years. ***Ip et al., 2001*** found a similar prevalence of OSAS in a Chinese population as in whites. The fact that OSAS is common in Asian areas where obesity is much less common has led to the hypothesis that craniofacial characteristics of the Asian population might predispose to OSAS.

6. Smoking

Current cigarette smokers are at a greater risk for OSAS than never smokers. Heavy smokers are at the greatest risk ***(Wetter et al., 1994).***

7. Alcohol Intake

Most studies of acute ingestion of alcohol in patients with snoring or OSAS have found an increase in the AHI***(Scanlan et al., 2000).***Alcohol does suppress REM sleep and, in this sense, could reduce the longer events associated with REM sleep during the early part of the night. However, more severe desaturations and presumably longer events do occur in some patients in the early portion of the night if bedtime alcohol is consumed***. Stradlinget al., 1991*** found alcohol consumption to be associated with the presence of OSAS in a group of middle-aged men.

In a study of the effect of alcohol on the arousal response to mask occlusion in normal subjects, ethanol ingestion delayed the time to arousal during non–rapid eye movement (NREM) sleep ***(Berry et al., 1982).***

8. Hypothyroidism

Hypothyroidism has been thought to be associated with OSAS***(Pelttari et al., 1994)***.

9. Acromegaly

Weiss et al., 2000 found that 75% of a group with acromegaly had OSAS. Independent predictors of OSAS included increased activity of acromegaly (higher growth hormone), older age, and an increased neck circumference. Potential pathophysiologic mechanisms of the association between acromegaly and OSAS include macroglossia and increased

muscle mass of the upper airway. Patients with acromegaly may have central as well as obstructive apnea.

TABLE (1) Relative degree of evidence for different risk factors for OSAS.

Risk Factors for Obstructive Sleep Apnea	
RISK FACTOR	**EVIDENCE**
Obesity—present in roughly 70% of OSA	+++
Male sex	+++
Aging	++
Postmenopausal state	++
Black race	+ (some studies)
Alcohol	++
Smoking	+

OSA = obstructive sleep apnea.
Adapted from Malhotra A, White DP: Obstructive sleep apnea. Lancet 2002;360:237–245.

Diagnosis of OSAS

Clinical guidelines for the evaluation, management, and long-term care of OSAS in adults recommended that high-risk populations for OSAS be questioned in detail concerning symptoms of OSAS ***(Epstein et al., 2009).***

TABLE (2) Common symptoms and signs of OSAS.

Common Symptoms and Manifestations of Obstructive Sleep Apnea	
Symptoms	Nocturnal Behavior
Excessive daytime sleepiness Nonrestorative sleep Frequent awakenings Morning headaches Dry mouth in the morning Personality change Intellectual changes Erectile dysfunction Nocturia	Loud habitual snoring Choking, gasping during sleep Breathing pauses/witnessed apnea Body movements, restlessness in bed

TABLE (3)

Recommendations for Evaluation of General Medical Patients and Populations at High Risk for Obstructive Sleep Apnea

HIGH-RISK PATIENTS FOR OSA	ROUTINE QUESTIONS AND OBSERVATIONS	COMPREHENSIVE SLEEP EXAMINATION QUESTIONS
Obesity (BMI > 30)	Obesity?	Witnessed apneas?
Congestive heart failure	Retrognathia?	Snoring?
Atrial fibrillation	Daytime sleepiness?	Gasping/choking at night?
Refractory hypertension	Snoring?	Nonrefreshing sleep?
Type 2 diabetes	Breathing pauses?	Total sleep amount?
Nocturnal arrhythmias	Hypertension?	Sleep fragmentation?
CVA		Nocturia?
Pulmonary hypertension		Morning headaches?
High-risk driving populations		Decreased concentration?
Preoperative for bariatric surgery		Memory loss?
		Decreased libido?
		Irritability?

BMI = body mass index; CVA = cerebrovascular accident; OSA = obstructive sleep apnea.
From Epstein LJ, Kristo D, Strollo PJ, et al: Clinical guideline for the evaluation, management and long-term care of obstructive sleep apnea in adults. J Clin Sleep Med 2009;5:263–276.

The patient or bed partner frequently reports excessive daytime sleepiness, loud habitual snoring, gasping/choking or witnessed apnea, personality change, morning headache, or non restorative sleep. Patients may also report a progression of symptoms with recent weight gain or nasal congestion***(Berry, 2012)***.

The Epworth Sleepiness Scale (ESS), a subjective estimate of the propensity to doze off in eight situations, is often (but not invariably) increased. The range of the scale is 0 to 24 with greater than 10 indicating excessive daytime sleepiness ***(Johns, 1993).***

TABLE (4) Epworth Sleepiness Scale.

Epworth Sleepiness Scale Scores in Mild, Moderate, and Severe Obstructive Sleep Apnea

	MEAN AHI (MEAN ± SD)	TOTAL NUMBER OF SUBJECTS	ESS (MEAN ± SD)	RANGE OF ESS
Mild OSA (AHI > 5–15)	8.8 ± 2.3	22	9.5 ± 3.3	4–16
Moderate OSA (AHI > 15–30)	21.1 ± 4.0	20	11.5 ± 4.2	5–20
Severe OSA (AHI > 30)	49.5 ± 9.6	13	16.0 ± 4.4	8–23

AHI = apnea-hypopnea index; ESS = Epworth Sleepiness Scale; OSA = obstructive sleep apnea; SD = standard deviation.
From Johns MW: Daytime sleepiness, snoring, and obstructive sleep apnea. The Epworth Sleepiness Scale. Chest 1993;103:30–36.

Physical examination

The physical examination of patients with suspected OSAS should target abnormalities known to be associated with the syndrome. These include measurement of BMI and systemic blood pressure as well as careful examination of the nose, ears, and oropharynx ***(Nuckton et al., 2006).***Observation of the oropharynx usually reveals a crowded upper airway and examination of the patient's face in profile may reveal retrognathia. Measurement of neck circumference and observation of signs of right heart failure may also be revealing. A neck size greater than 17 inches in men and 16 inches in women suggests the possibility of OSAS. However, a smaller neck circumference does not rule out OSAS.

The Mallampati (MP) score of the upper airway was developed to predict the risk of difficult endotracheal intubation. The patient's oropharynx is examined with tongue protruded. The modified Mallampati score (MMP), also called the Friedman score***(Friedman et al., 1999),***is similar but the patient simply opens the mouth *without* saying "ah" or tongue protrusion. ***Friedman et al., 1999*** found that the MMP, tonsil size, and BMI were reliable predictors of OSAS.

Zonatoet al., 2003 found a significant correlation between the AHI and the MMP and BMI. Although retrognathia was not correlated with AHI, this abnormality was more frequent in patients with severe OSAS as compared with snorers. ***Nucktonet al., 2006*** analyzed over 30 variables reflecting airway anatomy, body habitus, symptoms, and medical history and found the MMP and MP to be independent predictors of the presence and severity of OSAS. The variables associated with an increased risk of OSAS in their study included increased neck circumference, witnessed apnea, and hypertension.

TABLE (5) Physical examination in patients suspected of having OSAS.

Obstructive Sleep Apnea Physical Examination—Key Elements in Patients Suspected of Having Obstructive Sleep Apnea
Increased BMI*
Presence of nasal obstruction
Increased Mallampati or modified Mallampati score*
High-arched palate (narrow airway)
Retrognathia
Increased neck circumference (>17 inches in men, >16 inches in women)
Evidence of right heart failure (JVD, pedal edema)

*Independent predictor of the presence of OSA.
BMI = body mass index; JVD = jugular venous distention; OSA = obstructive sleep apnea.
From Nuckton TJ, Glidden DV, Browner WS, Claman DM: Physical examination: Mallampati score as an independent predictor of obstructive sleep apnea. Sleep 2006;29:903–908.

FIGURE (1) Mallampati and modified Mallampati airway classification. In the Mallampati maneuver, patients are instructed not to emit sounds but to open the mouth as wide as possible and protrude the tongue as far as possible ***(Friedman et al., 1999)***. In the modified Mallampati, the patient is instructed to open the mouth as wide as possible without emitting sounds***(Nuckton et al., 2006).***

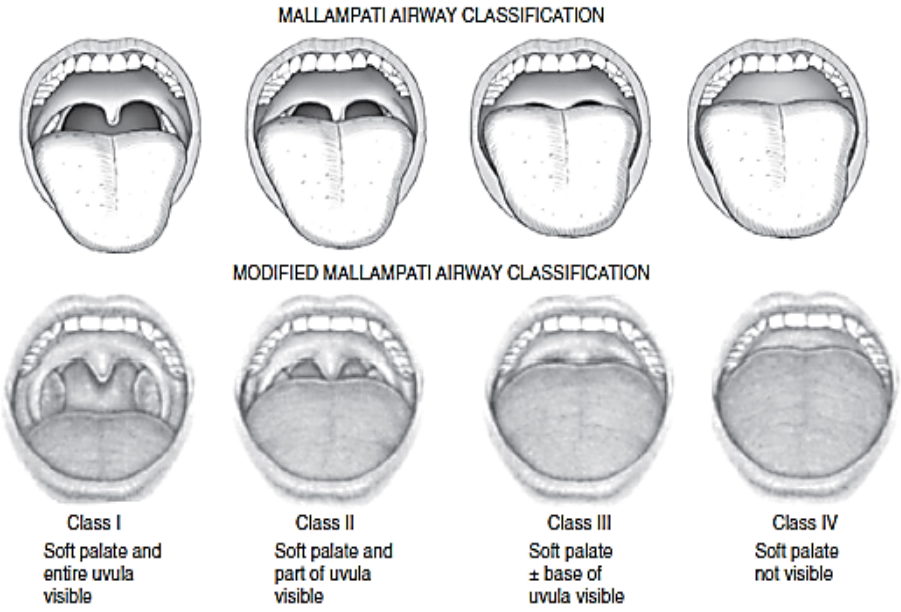


FIGURE (2) A patient with severe retrognathia on the **left** and orthognathia (normal) on the **right** ***(Friedman et al., 1999)***.

Laboratory testing in OSAS

Laboratory testing in patients with OSAS is usually not indicated apart from routine health maintenance unless a particular problem such as hypothyroidism is suspected. In patients with severe nocturnal

hypoxemia, polycythemia(increased hematocrit) may be present. An unexplained elevation in the serum CO2 (composed primarily of HCO3)on electrolyte testing is suggestive of chronic compensation for hypercapnia (in the absence of evidence of causes of metabolic alkalosis)***(Berry&Sriram, 2009).***Pulmonary function testing, chest radiography, and arterial blood gas testing are indicated in patients with a low awake SaO2 or suspected hypoventilation to eliminate pulmonary causes of impaired gas exchange***(Berry &Sriram, 2009).***

Prediction of the presence of OSAS

Flemons, 2002 used an adjusted neck circumference to classify patients as low, moderate, or high probability. The population in which the prediction value was developed was predominantly male, hypopneas were defined as a reduction in airflow associated with a 3% or greater desaturation, and the presence of OSAS was defined by an AHI greater than 10/hr.

TABLE (6) Adjusted neck circumference.

Prediction of Obstructive Sleep Apnea
NC = measure neck circumference (cm) A. If hypertension present, +4 B. If habitual snoring present, +3 C. If gasping or choking present, +3
Adjusted neck circumference = NC + A + B + C
<43 cm (17 inches) low probability 43–48 cm (17–19 inches) moderate probability >48 cm (19 inches) high probability
From Flemons WW: Obstructive sleep apnea. N Engl J Med 2002;347:498–504.

The Berlin Questionnaire consists of three categories: category 1 concerns snoring and witnessed apnea, category 2 concerns being sleepy/tired/fatigued more than three times a week or nodding off while driving a vehicle, and category 3 concerns the presence of hypertension. After questionnaire completion, patients were studied by portable monitoring (PM). The Berlin Questionnaire identified patients with an AHI ≥ 5/hr (based on assignment to the high-risk group) with a sensitivity of 0.86 and a specificity of 0.77. The STOP-BANG (snoring, tired, observed apnea, [blood] pressure, body mass index, age, neck circumference, gender) Questionnaire is a screening tool that has been used for preoperative evaluation to detect sleep apnea. A study of 2467 patients found sensitivities of 84%,92%, and 100% for AHI cutoffs of greater than 5/hr, greater than 15/hr, and greater than 30/hr***(Ong et al., 2010).***

STOP-BANG Scoring Model

(BMI = body mass index; OSAS= obstructive sleep apnea syndrome).

1. ***S***noring

   Do you snore loudly (louder than talking or loud enough to be heard through closed doors)? Yes No

2. ***T***ired

   Do you often feel *t*ired, fatigued, or sleepy during daytime?

   Yes No

3. ***O***bserved

   Has anyone *o*bserved you stop breathing during your sleep?

   Yes No

4. Blood ***p***ressure

   Do you have or are you being treated for high blood *p*ressure?

   Yes No

5. ***B***MI

   *B*MI >35 kg/m2? Yes No

6. ***A***ge

   *A*ge older than 50 yr old? Yes No

7. ***N***eck circumference

   *N*eck circumference >40 cm? Yes No

8. ***G***ender

   *G*ender male? Yes No

- *High risk of OSAS:* Answering yes to three or more items.
- *Low risk of OSAS:* Answering yes to less than three items.

Diagnostic testing for suspected OSAS

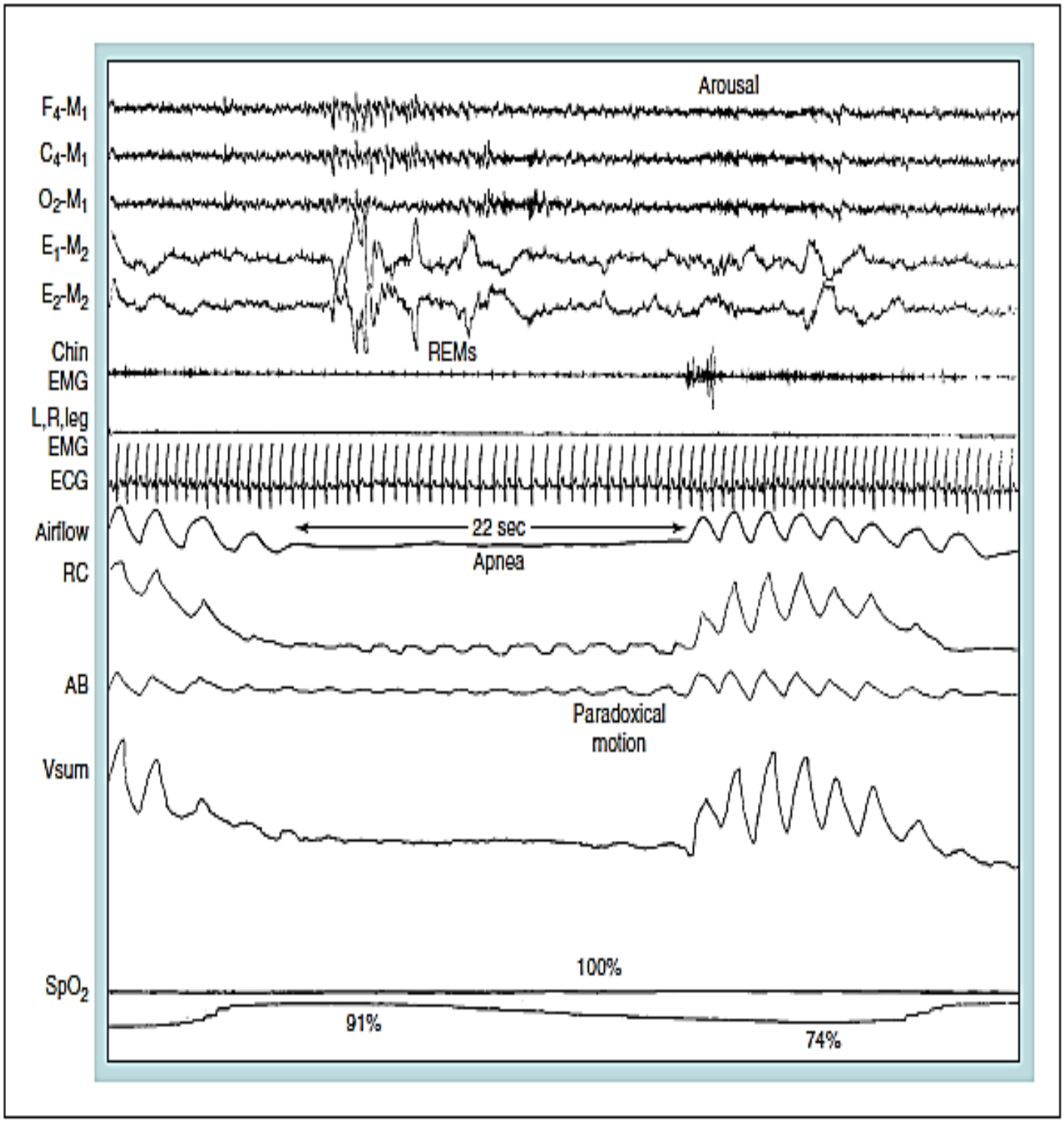
I) polysomnography (PSG)

Attended polysomnography (PSG) is the gold standard to determine whether OSAS is present and to classify the severity ***(Kushida et al., 2005).*** The abnormality in the sleep architecture varies with severity. Patients with severe OSAS usually have a high arousal index, increased wake after sleep onset (WASO) and stage N1 sleep, and decreases in stage N3 or stage R sleep. A typical classification of OSAS severity based on the AHI (or RDI in some sleep centers) is 5 to fewer than 15/hr mild, 15 to 30 moderate, and greater than 30/hr severe OSAS. Whereas the AHI(RDI) is the most widely used index for classification of severity, it is also important to characterize the severity of arterial oxygen

desaturation. A widely accepted standard for the characterization of the severity of desaturation does not exist. It is common to present the number of desaturations(usually defined as a drop in the SaO2 >4%), the lowest SaO2,the average SaO2 at desaturation, and the time below various saturations. For example, a commonly used metric is the time at or below an SaO2 of 88%.PSG can diagnose OSAS either with an entire night of monitoring or by the initial diagnostic portion of a split (partial-night) study. The second part of the study is used as a PAP titration ***(Oksenberg et al., 2010).***

The diagnosis of postural OSAS is usually made when the AHI-supine is greater than twice the AHI-non supine. Some clinicians define REM-associated OSAS as a normal AHI during NREM sleep associated with an elevated AHI during REM sleep. Other clinicians use the term to denote patients with an AHI-REM/AHI-NREM greater than 2 ***(Berry, 2012)***.

FIGURE (3) An obstructive apnea during rapid eye movement (REM) sleep. The longest respiratory event duration and most severe arterial oxygen desaturation are usually during REM sleep (especially supine REM sleep). Note the slowing of the heart rate at the start of the apnea and speeding at apnea termination. Here Vsum is the sum of rib cage (RC) and abdominal (AB) respiratory inductance plethysmograph bands. ECG = electrocardiogram; EMG = electromyogram; SpO2 = pulse oximetry



There is also an interaction between body position and sleep stage. The AHI tends to be higher during supine than during non supine REM sleep ***(Oksenberg et al., 2010).***Patients with a mild to moderate overall AHI are more likely to have REM-related OSAS. An occasional patient with REM-related OSAS may exhibit very severe arterial oxygen desaturation and long apneas during REM sleep but a relatively normal AHI-NREM.

Kass et al., 1996 identified a group of patients with overall AHI less than 10/hr but AHI-REM greater than 15/hr who had objective evidence of daytime sleepiness. A large population based analysis of the Sleep Heart Health Study cohort found a relationship between the AHI-NREM but not the AHI-REM and daytime sleepiness ***(Chami et al., 2010).***

Cyclic variation in heart rate is typically noted during their beated episodes of obstructive events. The heart rate slows at the start of the events and increases at event termination. Often, the heart rate remains between 60and 100 bpm. A standard part of most PSG reports is to present the maximum and minimum heart rate along with notations of abnormalities (premature ventricular contractions, atrial fibrillation, sinus pauses) ***(Bonsignore et al., 1997).***

TABLE (7) Polysomnographic finding of OSAS.

Polysomnographic Findings in Obstructive Sleep Apnea
EEG FINDINGS
Increased WASO and stage N1 Reduced stage N3 Reduced stage R (REM sleep) Increased respiratory arousals
RESPIRATORY FINDINGS
Snoring Obstructive, mixed apneas, and central apneas Obstructive hypopneas AHI: mild 5 to <15/hr, moderate 15–30/hr, severe > 30/hr AHI supine > 2 × AHI nonsupine – postural OSA AHI REM > AHI NREM common Apnea duration REM > NREM
ARTERIAL OXYGEN DESATURATION
Lowest SaO_2 during REM sleep Longest REM periods in the early morning hours typically have the worst desaturation
CYCLIC VARIATION IN HEART RATE
Slowing of heart rate at apnea onset and speeding at event termination
AHI = apnea-hypopnea index; EEG = electroencephalogram; NREM = non–rapid eye movement; OSA = obstructive sleep apnea; REM = rapid eye movement; SaO_2 = arterial oxygen saturation; WASO = wake after sleep onset.

FIGURE (4) The heart rate during an obstructive apnea. The heart rate signal is the output of the oximeter (a moving time average) that tends to lag behind the actual change in heart rate. Changes in the heart rate can be noted by changes in the RR interval, which widens at apnea onset then narrows at apnea termination. ECG = electrocardiogram; SpO2 = pulse oximetry.

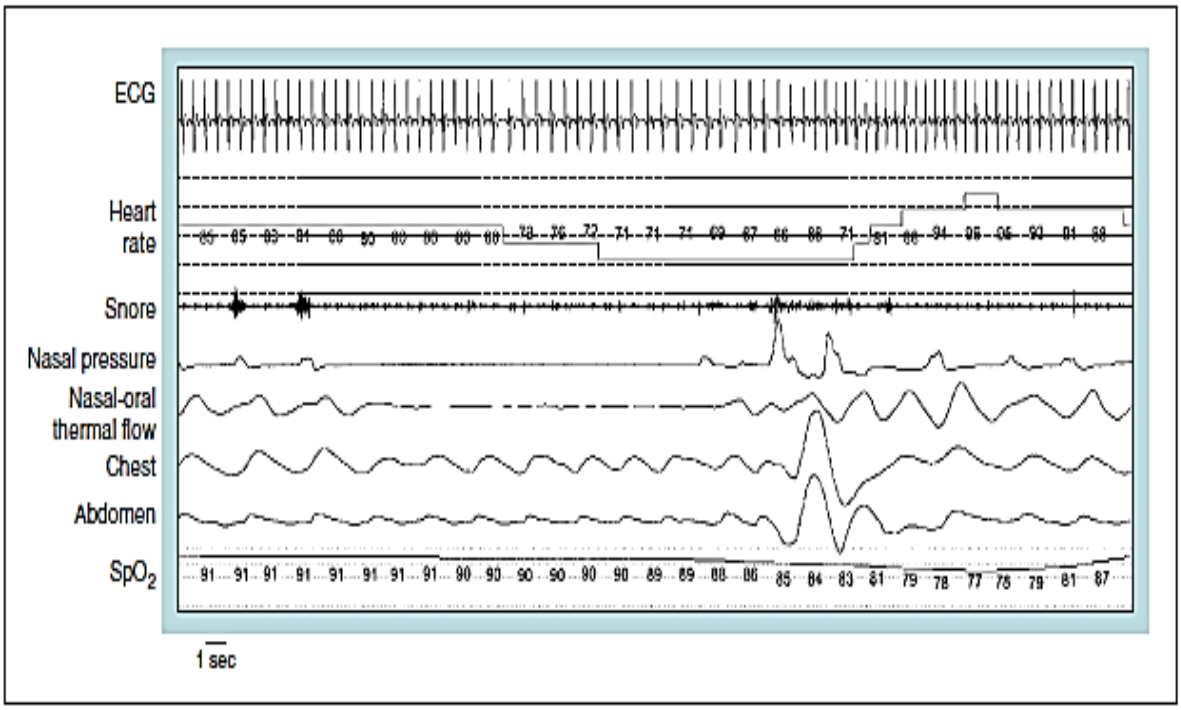


TABLE (8) Typical presentation of respiratory events.

Typical Presentation of Respiratory Events					
	TOTAL				
TST (min)	360				
NREM (min)	290				
REM sleep (min)	70				
% supine	16				
	TOTAL	SUPINE	NONSUPINE	NREM	REM
TST in condition	360	60	300	290	70
OA (#)	24	14	10	4	20
MA (#)	5	5	0	0	5
CA (#)	2	1	1	1	1
Hypopnea (#)	24	19	5	20	4
Total (#)	55	39	16	25	30
AHI (#/hr)	9.2	39.0	3.2	5.2	25.7

AHI = apnea-hypopnea index; CA = central apnea; MA = mixed apnea; NREM = non–rapid eye movement; OA = obstructive apnea; REM = rapid eye movement; TST = total sleep time.

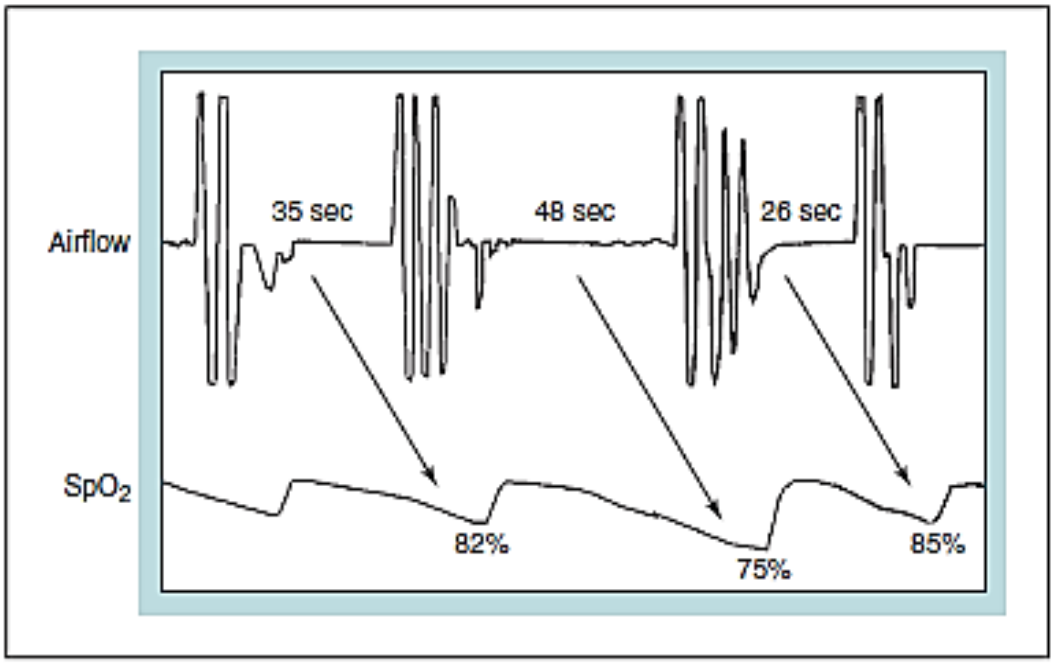
Arterial oxygen desaturation typically occur during REM sleep in the second part of the night. Split- or partial-night sleep studies (initial part diagnostic, second part PAP titration) are frequently used in severe patients (by AHI). However, because there is often minimal REM sleep during the diagnostic portion, the severity of arterial oxygen desaturation based on a split study may be dramatically underestimated. A full night of diagnostic monitoring provides the best estimate of the typical severity of arterial oxygen desaturation. A low baseline awake SaO2, long apnea time (long apnea duration with short intra-apnea ventilation), and a low expiratory reserve volume (ERV) are associated with more severe arterial oxygen desaturation ***(Bradley et al., 1985).***

A large study of the Wisconsin cohort found that a higher BMI was associated with more severe arterial oxygen desaturation independent of age, gender, sleeping position, baseline SaO2, and event duration ***(Peppard et al., 2009).***A higher BMI had a greater effect on desaturation during REM than during NREM sleep. In addition, a fall in tidal volume had a greater effect on arterial oxygen desaturation when the BMI was higher. The predicted change in theSaO2 was also higher in the supine position than in the lateral position, in men than in women, and in smokers than in non smokers. Another study found that obstructive events in the supine position tended to be longer, were associated with more severe desaturation, and were more likely to be associated with an arousal at event termination***(Oksenberg et al., 2000).***

TABLE (9) Severity of associated arterial oxygen desaturation.

Severity of Associated Arterial Oxygen Desaturation
FACTORS ASSOCIATED WITH SEVERE DESATURATION
Low awake SaO_2 Long apnea time (long apnea duration, short ventilatory period between apneas) Low ERV (FRC – RV) Low FRC—obesity High RV—obstructive lung disease
GROUPS WITH SEVERE DESATURATION
Severe obesity Obesity-hypoventilation syndrome OSA + COPD (overlap syndrome)
COPD = chronic obstructive pulmonary disease; ERV = expiratory reserve volume; FRC = functional residual capacity; OSA = obstructive sleep apnea; RV = residual volume; SaO_2 = arterial oxygen saturation.

FIGURE (5) Variable arterial oxygen desaturation in a patient with severe OSAS. Note as expected longer apnea resulted in more severe arterial oxygen desaturation. SpO2 = pulse oximetry***(Berry, 2003).***



Diagnostic criteria for OSAS

The apnea-hypopnea index (AHI) is defined as the number of apneas and hypopneas per hour of sleep. The respiratory disturbance index (RDI) is the number of apneas, hypopneas, and respiratory effort–related arousals (RERAs) per hour of sleep (RDI = AHI + RERA index). RERAs are events associated with increased respiratory effort (as evidenced by esophageal pressure manometry or flattening of the nasal pressure signal) for 10 seconds or longer that are associated with arousal but do not meet criteria for hypopnea ***(Iber et al., 2007)***. Whereas the recent AASM scoring manual defines RERAs, the manual does not define the term RDI. The Centers for Medicare and Medicaid Services (CMS) guidelines ***(Revision of NCD240.4)*** regarding reimbursement criteria for continuous positive airway pressure (CPAP) define the RDI as the number of respiratory events per hour of monitoring time. A commonly used diagnostic criteria for OSAS has been an AHI ≥5/hr associated with symptoms or an AHI ≥15/hr with or without associated symptoms ***(Loube et al., 1999).***Some diagnostic criteria have used the RDI ([apneas + hypopneas+ RERAs]/hour of sleep) instead of the AHI. The International Classification of Sleep Disorders, 2nd edition (ICSD-2), criteria for a diagnosis of the OSAS in adults ***(American Academy of Sleep Medicine: ICSD-2 International Classification of Sleep Disorders, 2nd ed, 2005)*** requires either score able respiratory events (apneas, hypopneas, or RERAs) of 5/hr or greater with associated symptoms or score able respiratory events of 15/hr or greater with or without symptoms.

TABLE (10) International classification of sleep disorders, 2nd edition, Criteria of OSAS.

International Classification of Sleep Disorders, 2nd Edition, Criteria for Obstructive Sleep Apnea

Diagnosis = A + B + D or C + D

A. At least one of the following applies:
   i. Complaints of unintentional sleep episodes during wakefulness, daytime sleepiness, unrefreshing sleep, fatigue, or insomnia.
   ii. Awakenings with breath-holding, gasping, or choking.
   iii. Bed partner reports loud snoring and/or breathing interruptions during the patient's sleep.

B. Polysomnography shows the following:
   i. Scoreable respiratory events (apneas + hypopneas + RERAs)/hr of sleep ≥ 5/hr.
   ii. Evidence of respiratory effort during all or a portion of each respiratory event (in the case of RERAs, respiratory effort is best detected by esophageal manometry).

OR

C. Polysomnography shows the following:
   i. Scoreable respiratory events (apneas + hypopneas + RERAs)/hr of sleep ≥ 15/hr.
   ii. Evidence of respiratory effort during all or a portion of each respiratory event.

D. The disorder is not better explained by another current sleep disorder, medical or neurologic disorder, medication use, or substance use disorder.

RERAs = respiratory effort–related arousal.
From American Academy of Sleep Medicine: ICSD-2 International Classification of Sleep Disorders, 2nd ed. Diagnostic and Coding Manual. Westchester, IL: American Academy of Sleep Medicine, 2005.

II) Portable Monitoring

The use of portable monitoring (PM), also known as home sleep testing (HST) or out of center sleep testing (OCST), is most appropriate when PSG is difficult due to immobility or safety issues, when there is a delay in obtaining a PSG due to access or availability and the clinical situation is urgent, when there is a high probability of OSAS, when

complicated comorbidities are not present, and when coexisting sleep disorders that may benefit from PSG are not present ***(Collop et al., 2007).***

AHI derived from testing without electroencephalogram (EEG) provides a number of events per hour of monitoring time, not total sleep time. For example, if PSG and PM both identify 100 events, the total sleep time is 6 hours, and the monitoring time is 7 hours, then the AHI PSG = 100/6 and the AHI PM = 100/7. Therefore, the AHI by PM will likely be less than by PSG, even if similar numbers of respiratory events are detected. There is also the concern for a false-negative PM study. This phenomenon is more likely if the patient does not sleep well during PM with the result that monitoring time greatly exceeds total sleep time. If there is a high index of suspicion for OSAS but the PM is negative for OSAS, a PSG should be performed ***(Collop et al., 2007).***

Complications of OSAS

Hypertension is found in50% of patients diagnosed with OSAS ***(Moos & Cuddeford, 2006)***; as apneas and hypopneas cause transient, acute blood pressure elevations throughout the night. These night time elevations may be partially responsible for the development of hypertension and cardiovascular disease ***(Shamsuzzaman et al., 2003).***

The nightly occurrences of sympathetic over activity put patients at increased risk of pulmonary hypertension, nocturnal angina, myocardial infarction, and other long-term systemic complications. Cardiac dysrhythmias such as bradycardia, premature ventricular contractions, and atrial fibrillation are often seen in patients with OSAS***(Coccagna et al., 2006)***.Intermittent periods of hypoxia and stimulation of the CNS may have important roles in atherosclerosis and resulting cardiovascular complications for the patient with untreated OSAS***(Coccagna et al.,***

2006).Over time, the severity of symptoms can worsen because of recurrent vibratory trauma and further airway narrowing***(American Society of Anesthesiologists, 2006).*** OSAS is also comorbid with obesity, diabetes, acid reflux, Parkinson's disease, hypothyroidism ***(Bassiri & Guilleminault, 2000).***

Complications Associated With OSAS*(Chung et al., 2008).*

- Systemic hypertension
- Ischemic heart disease
- Congestive heart failure
- Corpulmonale
- Pulmonary hypertension
- Cerebrovascular disease
- Polycythemia
- Impaired cognition, fatigue
- Increased risk of accidents (eg, motor vehicle, occupational)
- Anxiety, depression
- Shorter life expectancy

Treatment of OSAS

Patient education before treatment

Following polysomnography (PSG) or portable monitoring (home sleep testing, limited-channel sleep testing), the physician ordering the study should discuss the findings and the consequences of untreated sleep apnea with the patient ***(Epstein et al., 2009)***.

TABLE (11) Patient education and co-morbid disorders.

Patient Education and Co-morbid Disorders
PATIENT EDUCATION
• Testing results—discuss with the patient without excessive medical jargon, answer questions
• Pathophysiology of OSA
• Consequences of untreated OSA
• Treatment options—pros and cons
• Drowsy driving counseling
EVALUATION AND TREATMENT OF CO-MORBID DISORDERS
• RLS
• Depression
• Insomnia
• Insufficient sleep
• Chronic pain
• Narcolepsy
OSA = obstructive sleep apnea; RLS = restless legs syndrome.

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 trouble shooting 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 OSAS 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 on going 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 OSAS were randomly assigned to receive CBT plus education or the same amount of therapist contact without information about OSAS or CPAP**.** CBT plus education consisted of two 45 minute sessions during which subjects were informed about the consequences of OSAS 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).***

Figure (6) 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 OSAS patients that aims at motivating adherence to Positive Airway Pressure (PAP) in obstructive sleep apnea ***(Golay et al., 2006).***

I.1-Rationale for METand 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 road blocks to using CPAP. Commonly cited road blocks 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).***

I.2-Indications

This treatment modality has been tested with patients who have been diagnosed recently with Obstructive Sleep Apnea Syndrome

(OSAS) 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).***

I.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 ***(Naegele´ et al., 1995).***

I.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 and Rollnick, 2002)***.

I.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).***

I.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 (12) Guiding principles of MET and their application to OSAS patients ***(Aloia et al., 2004).***

Guiding principle	Example
I. Feedback	Conduct a thorough assessment and offer personalized feedback about changes in clinical measures of OSAS 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
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.

I.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).***

I.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 and 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).***

- ***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).***

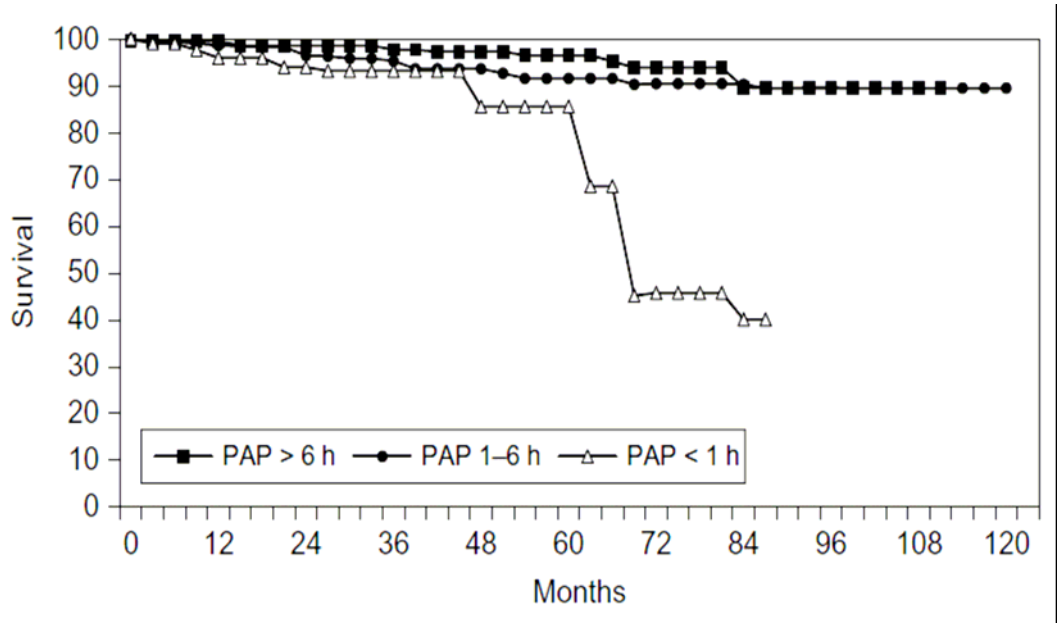
- ***Review of symptoms***

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 OSAS 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 7). 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 (7) 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 OSAS 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 ***(Saunamaki 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 OSAS; patient reaction to feedback on the PSG; medical conditions the patient may be at risk for with untreated OSAS; 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).***

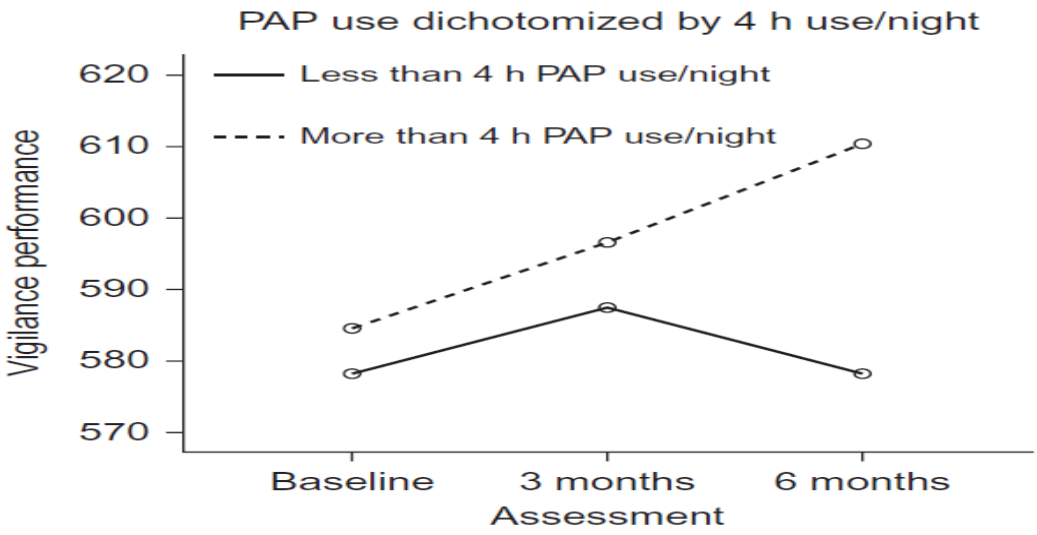
- ***Information exchange: PAP benefits for health and functioning***

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 ≥ 4hours a night compared with those who use PAP <4 hours a night, over a 6-month period of time (Figure 8). 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 (8) 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 OSAS, 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 there by 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 it's 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 13. Exposure therapy session components. ***(Means and Edinger, 2007).***

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 (14) 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 shows 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 maintains 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 peace 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 et al., 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 and 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 and Edinger, 2007).***

Should mild OSAS patients be treated?

Whereas most clinicians recommend treatment of patients with an AHI of 15/hr or greater even if not symptomatic, treatment of mild OSAS remains controversial***(Littner, 2007).***The Wisconsin cohort study found a 2.03 greater risk of incident hypertension in patients with an AHI in the range of 5 to 14.9 compared with individuals without sleep apnea (AHI = 0/hr)***(Peppard et al., 2000).***The fact that even mild OSAS could have adverse consequences is supported by a recent finding that snoring alone can cause carotid atherosclerosis***(Lee et al., 2008).***Effective treatment of patients with mild OSAS can improve symptoms. A meta-analysis of treatment studies of mild to moderate OSAS found that CPAP significantly reduced subjective sleepiness (the ESS decreased by 1.2 points) and improved objective wakefulness (measure of maintenance of wakefulness test [MWT] sleep latency increased by 2.1 min) ***(Marshall et al., 2006).***Conversely, whereas suboptimal CPAP adherence has often been reported in studies of patients with mild OSAS, several studies

found 40% to 60% of patients had greater than 4 hours use for 70% of nights***(Brown, 2007)***.

Whom to treat?

At least four treatment considerations affect the decision to treat a patient with OSAS. The first category is the severity of OSAS as based on the AHI or extent of arterial oxygen desaturation. The second consideration is presence or absence of symptoms. Symptomatic OSAS should always be treated but the choice of treatment may vary, as noted later. The third consideration is the impact of OSAS on the sleep of the Patient's bed partner. Loud snoring and apnea may cause marital discord and impair the sleep of the patient's bed partner ***(Beninati et al., 1999).***In some cases, sleeping in separate bedrooms is the most acceptable solution. However, if this is not acceptable, treatment of the OSAS patient can significantly improve the sleep of the bed partner ***(Beninati et al., 1999).***The fourth category is the increased risk of adverse cardiovascular morbidity and mortality associated with untreated sleep apnea. The evidence that untreated sleep apnea is associated with an increased risk of death or adverse cardiovascular event is strongest for severe OSAS (AHI >30/hr) and in men who are 40 to 70 years of age ***(Punjabi et al., 2009).***

TABLE (15) Consideration-Whom to treat?

Consideration—Whom to Treat?			
	SYMPTOMATIC	ASYMPTOMATIC	ASYMPTOMATIC
AHI		No significant medical morbidities	Significant medical morbidities
Mild	Treat	Observation or conservative treatment	? Treat
Moderate	Treat	Treat	Treat
Severe	Treat	Treat	Treat
Conservative treatment: weight loss, side sleep position, treat nasal congestion, avoid alcohol.			
AHI = apnea-hypopnea index.			

TREATMENT SELECTION

TABLE (16) Treatment alternatives for OSAS (Adults).

Treatment Alternatives for Obstructive Sleep Apnea (Adults)[4-6]				
	SNORING	MILD	MODERATE	SEVERE
Primary	Treat nasal congestion Lateral positioning	Oral appliance or Upper airway surgery	PAP	PAP
Secondary	Oral appliance or Upper airway surgery	PAP (if symptomatic)	Oral appliance or Upper airway surgery	Upper airway surgery or Oral appliance
Adjunctive	Weight loss	Weight loss Lateral positioning	Weight loss Lateral positioning	Weight loss Lateral positioning
PAP = positive airway pressure.				

Snoring

For snoring patients for whom treatment is felt necessary, a number of therapies are available including weight loss, medical treatment of nasal congestion, the side (lateral)sleeping position, and avoidance of alcohol. If medical treatment does not improve nasal congestion, nasal surgery may address this problem, although improvement in snoring is variable ***(Koutsourelakis et al., 2008)***. Oral appliances (OAs) or upper airway surgery involving the palate may improve snoring in many patients ***(Caples et al., 2010).***The surgical procedures commonly used for snoring include laser-assisted uvulopalatoplasty(LAUP), radio frequency palatoplasty, and uvulopalatopharyngoplasty (UPPP) ***(Caples et al., 2010).***

The Pillar procedure involves insertion of Teflon strips into the palate. The success of this procedure is variable, and sometimes additional strip insertion is needed ***(Friedman et al., 2008)***.A controlled study comparing the Pillar procedure with a sham surgery found modest improvement in snoring without a significant improvement in the AHI***(Steward et al., 2008)***.The recently published American Academy of Sleep Medicine(AASM) practice parameters for surgical treatment of OSAS state "palatal implants may be effective in some patients with mild OSAS who cannot tolerate or are unwilling to adhere to positive airway pressure (PAP) therapy, or in whom oral appliances have been considered and found ineffective or undesirable." ***(Aurora et al., 2010)***.

Mild OSAS

For mild OSAS, one may again begin with conservative measures in (especially in asymptomatic patients) including treatment of nasal congestion, weight loss, or the lateral sleeping position. Weight loss takes

time, and if the patient is symptomatic, weight loss should be considered a secondary (adjunctive) treatment. Both OAs and upper airway surgery are reasonably effective for mild OSAS ***(Caples et al., 2010).***PAP is very efficacious at reducing the AHI ***(Epstein et al., 2009).***

PAP treatment is not indicated in mild OSAS if patients area symptomatic and have no co-morbid cardiovascular disorders ***(Loube et al., 1999),*** but in symptomatic patients with mild OSAS, some patients may prefer a trial of PAP rather than an OA or upper airway surgery. PAP treatment is safe and it is often difficult to predict who will benefit. If the patient is motivated to try PAP, the dictum "when in doubt, pressurize the snout should be considered ***(Personal communication from Philip Westbrook, 1983).***

Moderate OSAS

For moderate OSAS, the treatment of choice is some form of PAP, as OA treatment and upper airway surgery are less reliable than PAP at reducing the AHI to less than 10/hr. However, they may be more acceptable to some patients and more "effective" if patients do not have reasonable PAP adherence ***(Caples et al., 2010)***.

AASM task force review concluded that OA was about 50% effective for moderate OSAS (defined as a treatment AHI <10/hr) ***(Ferguson et al., 2006).***A meta-analysis of surgery in OSAS patients concluded that palatal surgery with or without genioglossus advancement was approximately 30% effective, using a strict outcome measure of reduction in the AHI to less than 10/hr ***(Elshaug et al., 2007).***

The AASM practice parameters for the surgical treatment of OSAS state "Uvulopalatopharyngoplasty (UPPP) as a sole procedure, with or

without tonsillectomy, does not reliably normalize the AHI when treating moderate to severe OSAS. Therefore, patients with severe OSAS should initially be offered PAP therapy, while those with moderate OSAS should initially be offered either PAP therapy or oral appliances ***(Aurora et al., 2010)***.

Severe OSAS

For severe OSAS, PAP is the treatment of choice because of its effectiveness and safety ***(Epstein et al., 2009).***

Unfortunately, acceptance and adherence can be as low as 50% depending on the definition of adherence. The maxillary mandibular advancement (MMA) operation can result in acceptable residual AHIs in many patients with severe OSAS ***(Caples et al., 2010).***

Some surgeons perform MMA only after other surgical procedures have failed. However, in very obese patients with retrognathia, performing MMA as the first surgical procedure is recommended by some clinicians. Tracheostomy is effective when life-threatening OSAS and respiratory failure are present and the patient is not compliant with PAP treatment ***(Aurora et al., 2010)***.

Follow-up and outcomes assessment

Following treatment initiation, careful follow-up is essential because OSAS is a chronic disease, a follow-up sleep study is recommended after upper airway surgery for moderate to severe OSAS***(Aurora et al., 2010)***, and after final adjustment of an OA as treatment for all severities of OSAS ***(Kushida et al., 2006)***.

TABLE (17) Assessment for treatment follow-up.

Assessments for Treatment Follow-up
ASSESSMENTS
• Objective adherence
• Epworth Sleepiness Scale, resolution of sleepiness
• Quality of life
• Patient and spousal satisfaction
COUNSELING
• Increased adherence
• Proper maintenance of equipment
• Avoidance of factors worsening disease
• Weight loss
• Adequate amount of sleep

Medical treatment for OSAS

Practice parameters of "medical" treatments for OSAS including weight loss, positioning, medications, oxygen, and alerting agents ***(Morgenthaler et al., 2006)***.

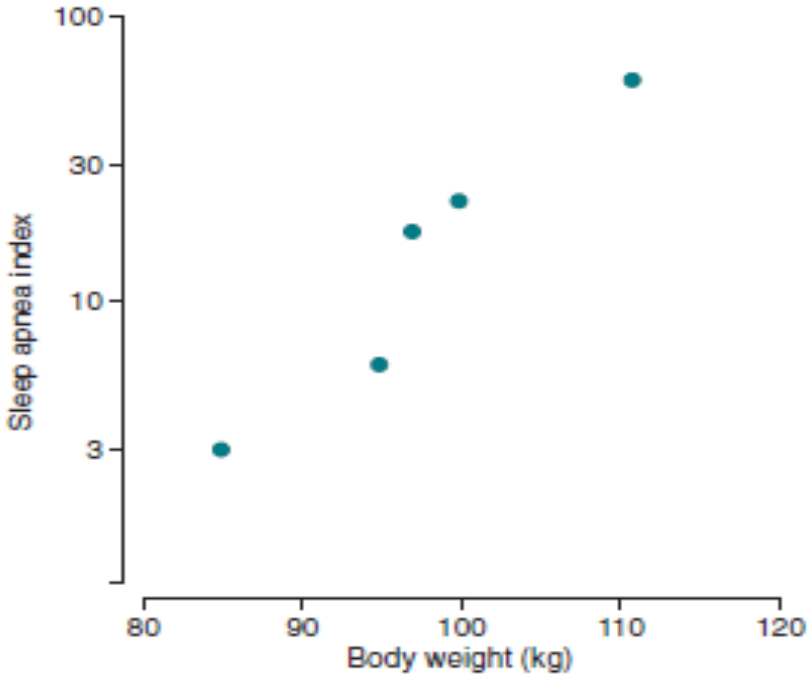
TABLE (18) Medical treatment for OSAS.

American Academy of Sleep Medicine Practice Parameter Recommendations for Medical Treatment of Obstructive Sleep Apnea
WEIGHT REDUCTION
• Successful dietary weight loss may improve the AHI in obese OSA patients. (Guideline)
• Dietary weight loss should be combined with primary treatment of OSA. (Option)
• Bariatric surgery may be **adjunctive** in treatment of OSA in obese patients. (Option)
POSITIONAL THERAPIES
• Positional therapy, consisting of a method that keeps the patient in a nonsupine position, is an effective secondary therapy or can be a supplement to primary therapies for OSA in patients who have a low AHI in the nonsupine versus the supine position. (Guideline)
OXYGEN SUPPLEMENTATION
• Oxygen supplementation is not recommended as a primary treatment for OSA. (Option)
NASAL CORTICOSTEROIDS
• Topical nasal corticosteroids may improve the AHI in patients with OSA and concurrent rhinitis and, thus, may be a useful adjunct to primary therapies for OSA. (Guideline)
MODAFINIL, ARMODAFINIL
• Modafinil is recommended for treatment of residual excessive sleepiness in OSA patients who have sleepiness despite effective PAP treatment and who are lacking any other identifiable cause for their sleepiness. (Standard)
OTHER TREATMENTS (NOT RECOMMENDED)
• Protriptyline, SSRIs, aminophylline, estrogen preparations with or without progesterone, and short-acting decongestants.
AHI = apnea-hypopnea index; OSA = obstructive sleep apnea; PAP = positive airway pressure; SSRIs = selective serotonin reuptake inhibitors.

Weight Loss

In the Wisconsin cohort study, for every standard deviation increase in the body mass index (BMI), there was a fourfold increase in the prevalence of OSAS***(Young et al., 1993)***.Many studies have documented that weight loss of modest proportions (5–10%) can produce significant improvement in sleep apnea***(Foster et al., 2009)***.

FIGURE (9) Decrease in apnea-hypopnea index (AHI) with weight loss. The y axis is a logarithmic scale. The weight loss was due to medication ***(Browman et al., 1984)***.



Peppard et al., 2000 followed the effects of weight change on AHI. A 10% weight gain predicted an approximate 32% increase in the AHI. A 10% weight loss predicted a 26% reduction in the AHI. A 10% increase in weight was associated with a six fold increase in the risk of developing moderate to severe OSAS.

Simpson et al., 2009 found that neck fat was the best predictor of a high AHI in women whereas central obesity was a better predictor in men.

Lettieri et al., 2008 reported a reduction in required CPAP from11.5 to 8.4 cm H2O after weight loss (BMI dropped from 51to 32 kg/m2) in a group of patients undergoing bariatric surgery.

Johansson et al., 2009 studied the effects of a very low energy diet on OSAS inpatients on CPAP (intervention vs. control group) with

moderate to severe OSAS. At 9 weeks, the intervention group had lost weight (–18.8 kg) with a drop in AHI of 23/hr.

Although both the weight and the AHI decreases were small, a significant number of the intervention group dropped their AHI below 5/hr. In the AHEAD (Action for Health in Diabetes) study, weight loss also resulted in a drop in the AHI***(Foster et al., 2009).***

A study of the effect of the weight reduction found a reduction of 7.9 kg was associated with a 30% reduction in the AHI (change in AHI 16/hr)***(Phillips et al., 2009).***

Nowadays, bariatric surgery is performed as a treatment for morbid obesity. However, it could be considered as an adjunctive treatment for OSAS. The most common bariatric operation is a Roux-en-Y procedure, although other surgery such as laparoscopic gastric banding may be tried for less obese patients. The mortality of the Roux-en-Y procedure is less than 2% ***(Flancbaum & Belsley, 2007)***.

Greenburg et al., 2009 performed a meta-analysis of bariatric surgery and the effects on OSAS in morbidly obese patients. Twelve studies including 342patients were analyzed. The mean BMI was reduced by17.9 kg/m2 (baseline 55.3 kg/m2) and the AHI was reduced from 54.7 to 15.8/hr. The authors concluded that bariatric surgery does result in both dramatic weight loss and improvement of the AHI, but not always to normal levels. Many patients will still likely require treatment of OSAS (CPAP and others). The AASM practice parameters for use of medical treatments for OSAS recommended that weight loss be combined with a primary treatment for OSAS ***(Morgenthaler et al., 2006).***

TABLE (19) Weight loss treatment for OSAS.

Weight Loss Treatment for Obstructive Sleep Apnea
• Weight loss is considered a secondary or adjunctive treatment.
• Modest weight loss can be helpful (10% weight loss is associated with a 26% reduction in the AHI).
• Recurrence of OSA can occur after previous weight loss despite maintenance of lower weight.
• Bariatric surgery rarely cures patients of OSA but may reduce the required level of CPAP.
AHI = apnea-hypopnea index; CPAP = continuous positive airway pressure; OSA = obstructive sleep apnea.

Psychiatric disorders in chest diseases

Psychiatric disorders in Bronchial Asthma

Asthma is a chronic lung condition characterized by small airway constriction and episodic inflammation. It is associated with significant medical morbidity and personal, social, and economic impact ***(Lavoie et al., 2006)***.

Scott et al., 2007 reported that the prevalence of major depression among individuals with asthma to be 2% to 26%. Generally, rates of depressive and anxiety disorders in asthma patients are at least double those of the general population ***(Lavoie et al., 2006).*** Among mental disorders, anxiety disorders are themost strongly and consistently associated with asthma ***(Roy-Byrne et al., 2008).***

A case-control study of youth with asthma found a two fold increase in prevalence of one or more anxiety or depressive disorders, with greater rates of anxiety disorders compared with mood disorders ***(Katon et al., 2007)***.

However, in older adults, asthma was significantly more associated with depression than anxiety disorders***(Shanmugam et al., 2007).***

Psychiatric conditions, particularly anxiety and depression, in asthma patients have a substantial negative impact on health outcomes, including asthma control, functional impairment, increased health care use, and cost***(Roy-Byrne et al., 2008).***

Both depressive and anxiety disorders are associated with worse asthma-related quality of life, and depressive disorders are associated with worse asthma control ***(Lavoie et al., 2006).***

The relationship between asthma and mental health may be bidirectional. A large-scale survey of a nationally representative sample reported a dose–response relationship between asthma and poor mental health in which degree of poor mental health appeared to increase the risk of having asthma ***(Chun et al., 2008)***.

Negative mood states such as depression can also result in poor self-care and negative health behaviors, which can ultimately lead to poor asthma control ***(Lavoie et al., 2006)***.

Psychiatric disorders in Chronic Obstructive Pulmonary Disease

Overview:-

COPD is an essentially irreversible and progressive disease of airflow limitation in the lung caused by small airway disease and parenchymal destruction; it most often develops in long-time smokers ***(Rabe et al., 2007)***. It is expected to be the third-leading cause of death globally by 2020 and is currently the fourth-leading cause of death in the United States ***(Rabe et al., 2007)***, afflicting up to 14% of adults ***(Maurer et al., 2008)***. Psychiatric comorbidities, including depression, anxiety, and psychosis, have been well documented as significant factors in the morbidity and mortality of COPD patients; COPD is also a risk factor for the development of psychiatric illness ***(Solano et al., 2006).***

Prevalence:-

The incidence of depression among COPD patients is about 37% to 71% from a systematic review of 64 articles and text books focusing on patients with severe disease ***(Solano et al., 2006).***

Di Marco et al., 2006 used the Zung Self-Rating Depression Scale and found that the prevalence of depression to be 18.8% for patients with COPD, compared with 3.5% for healthy controls. Other depression estimates range from 10% to 42% among patients with stable COPD and up to 62% among patients with severe COPD. Among patients who recently recovered from an acute exacerbation of COPD, the prevalence ranges from 19% to 50% ***(Maurer et al., 2008)***.

Vogele and Von Leupoldt, 2008 compared 20 patients with mild to moderate COPD with a control group of 20 patients hospitalized for degenerative orthopedic conditions showed a 55% prevalence rate of anxiety disorders among COPD patients, compared with a 30% prevalence rate among the orthopedic group.

Along with depression and anxiety, schizophrenia also has been found to be associated with COPD. Patients with schizophrenia had a10.8% prevalence of COPD, compared with 3.6% among controls ***(Carney et al., 2006).***

Chaves and Shirakawa, 2008 found that the smoking rate which is a major risk factor for developing COPD is 57.8% in a sample of 83 patients with schizophrenia.

Ng et al., 2009 found that COPD patients who reported depressive symptoms were significantly more likely to report disability with activities of daily living,poor to fair self-reported health, and poor scores onthe 12-item Health Survey physical and mental component summary. Most studies indicate worse mortality among COPD patients with depression; the most recent study reported depressive symptoms associated with a two fold increase in mortality at 1-year follow-up ***(Ng et al., 2007).***

Stage et al., 2005 demonstrated that COPD patients with anxiety, when compared with COPD patients without anxiety, have greater disability, poorer functional status, and increased length of hospitalization. The bidirectional impact is also evident, as worsening COPD status is associated with increasing prevalence of anxious symptoms ***(Cully et al., 2007).***

Etiology:-

Chronic hypoxemia may lead to disruptions of noradrenergic and dopaminergic synthesis release, and replenishment—as evidenced in rats—that ultimately lead to anxiety and depression; also chronic hypoxemia may lead to poor oxygenation in the periventricular and subcortical regions of the brain, which are vulnerable regions to hypoperfusion, and lead to similar brain MRI changes as seen in patients with depression ***(Norwood, 2006).***

Mikkelsen et al., 2004 further outlined possible explanatory models for a common pathophysiology between COPD and anxiety, including the hyperventilation model, carbon dioxide hypersensitivity model, and cognitive-behavioral model.

In addition, panic disorder was found to have the highest hazard ratio in predicting progression to nicotine dependence in COPD patients ***(Sartor et al., 2008).***

Treatment/intervention:-

Putman-Casdorph and McCrone, 2009 have summarized the pharmacologic studies between1992 and 2007 identified the selective serotonin reuptake inhibitors sertraline and paroxetine as being well tolerated overall and possibly providing brief symptom relief for anxiety

and depression. Buspirone, often preferred for anxiolytic treatment over benzodiazepines (as there is less concern for respiratory depression), was also found to be well tolerated. However, as with selective serotonin reuptake inhibitors, study results for buspirone have been mixed with regard to statistically significant improvement in anxiety. The tricyclic antidepressant nortriptyline previously was found to reduce symptoms of depression, anxiety, certain respiratory symptoms, and overall physical discomfort.

Pulmonary rehabilitation (PR) provides an opportunity for a collaborative care model between mental health professionals and a multidisciplinary pulmonary team. Although several studies have shown improvement of anxiety using PR alone, some patients may benefit from specific or individualized psychiatric interventions beyond PR ***(Kayahan et al.,2006)***.

Furthermore, lung volume reductionsurgery was highlighted as improving psychomotor speed, verbal memory, and naming skills and reducing depressive symptoms when compared with PR alone ***(Kozora et al., 2008)***.

COPD must be optimally managed before starting electroconvulsive therapy (ECT) as it may cause prolonged seizures in COPD patients receiving theophylline***(Schak et al., 2008).***

Psychiatric disorders in Tuberculosis (TB)

Psychiatric disorders and illness perceptions in tuberculosis:-

It is truly amazing how little scientific information is available regarding psychiatric disorders and illness perceptions of tuberculosis patients ***(Lalit Kant and Nagpaul, 2001)***. Traditional wisdom ordains

that tuberculosis occurring in an individual is always an interruption in life physically, psychologically, economically as well as socially. Indeed, when the diagnosis is first divulged to a patient seemingly having ordinary cough and or fever, a sudden psychological trauma occurs***(Betty Chang et al., 2004).***

How do people cope with the situation, immediately and much later when the effects of the initial shock have worn off and myriad problems of treatment, disruption in family life and social adjustments stare them in the face? ***(Hudelson, 1996)***.

There are plentiful conceptions in public mind regarding these aspects based on numerous observations and long experiences from the early days of the tuberculosis epidemic but before the days of scientific studies. According to these, there is often a brief or prolonged period of denial. The patient may either ignore the diagnosis or resort to self-treatment or seek different opinion from other sources ***(Lalit Kant and Nagpaul, 2001).***

It was observed in tuberculous patients in sanatoria or under early domiciliary care, glaring examples of strong emotions like fear, jealousy, vindictive behavior, anger, non-cooperation, guilt or a sense of shame. Rarely, suicides occurred, especially when rest of the family tried to segregate the afflicted in a distant institution or even stooped to a complete abandonment ***(Thomas et al., 1999).***

Anxiety and depression in tuberculosis:-

Prevalence of depression and anxiety in TB patients is further higher than already reported mean prevalence of anxiety and depression. Several

studies of TB patients have shown that more than half of the patients had depression ***(Aghanwa and Erhabor, 1998).***

The data of several studies suggest that raised depression and anxiety scores were associated with an increase in the number of TB symptoms reported, more serious perceived consequences and less control over the illness ***(Mirza and Jenkins, 2004)***.

Risk factors for psychiatric disorders with tuberculosis:-

Moran, 1985 related several social factors (such as loss of job, loss of role in family, long hospitalization, or a perception of being infected or dirty) with the depressive symptoms in tuberculosis patients.

Depression and lack of perceived control were independently associated with poor adherence. Thus treating psychological problems in patients with tuberculosis may substantially improve treatment adherence, although further research is needed ***(Dearman et al., 2004)***. Taking into consideration low literacy levels and other cultural barriers, new treatment strategies should incorporate psycho education and cognitive behavioral therapy to achieve these treatment outcomes ***(Husain et al., 2008)***.

Impact of tuberculosis on quality of life:-

Marra et al., 2004 had revealed that TB has a large impact on affected individuals' quality of life (QOL) through issues related to its diagnosis, treatment, social support and functioning, and health behavior. Specifically, that the domains of QOL that were affected by TB included those that are typically affected by most illnesses such as physical functioning, emotional and mental well-being.

There are numerous aspects of active TB that may lead to a reduction in QOL. Treatment of active TB requires prolonged therapy (at least 6 months) with multiple, potentially toxic drugs that can lead to adverse reactions in a significant number of patients ***(Yee et al., 2003).*** Also, there is considerable social stigma associated with active TB leaving the individual feeling shammed and isolated from their friends and families ***(Kelly, 1999).***

Fear of infection is one of the factors contributing to social stigma, which may produce social isolation, diminished marriage prospects, limited social support, and result therefore in the denial of diagnosis and consequent rejection of treatment. Despite the fact that TB infection is not necessarily associated with specific 'risk behaviors', TB patients are still generally held responsible for their illness and blamed for not taking better care of themselves ***(Sweetland et al., 2002).***

TB patients' social functioning was also affected through isolation, variable social support by family and friends, and the ability to continue with social and leisure activities. Also, the process of getting treatment for TB from the initial hospitalization to the daily medication schedules adversely affected the lives of the patients, although, almost all recognized the need for appropriate Treatment ***(Marra et al., 2004).***

Further, poverty alone has a clear association with increased risk for mental illness ***(Patel and Kleinman, 2003)***. Tuberculosis is strongly associated with material deprivation, including poor housing conditions ***(Smith, 1989)***. This association can be explained in terms of two broad causal mechanisms: increased exposure to infection and increased susceptibility to disease. Virtually all transmission of infection occurs in enclosed environments as exposure to ultraviolet radiation is known to

kill the bacterium ***(Snider, 1994).*** Thus, tuberculosis is associated with crowded living conditions and high incidence rates have been reported in prisons, shelters and nursing homes ***(Snider and Hutton, 1989)***.

However, whilst crowding increases the risk of exposure to infection this does not invariably lead to disease. It is known that immunological status is instrumental in whether or not an individual develops tuberculosis. Impoverished housing conditions, inadequate diet, physical hardship and psychological stress are some frequent consequences of poverty and all are associated with suppressed immunity ***(Halpern, 1995).***

It is unsurprising, therefore, that a number of studies find high rates of active tuberculosis amongst the homeless, with levels between 150 and 300 times that of the general population ***(Imperato, 1992).*** There is also evidence that the increase in tuberculosis notifications is predominantly associated with the poorest areas of the country and that individuals living in such areas are at greater risk of developing the disease ***(Bhatti et al., 1995).***

Several authors have described how these psychosocial factors complicate adherence to drug regimens, and emphasize the importance of attention to mental health needs to ensure positive treatment outcomes ***(Fullilove et al., 1993).*** One of the main causes of treatment failure and rise in the prevalence of TB is due to poor treatment adherence ***(Vanderwerf et al., 1990)***.

Psychiatric disorders with multidrug resistant tuberculosis:-

Psychiatric disorders were frequently observed in patients with multidrug resistant tuberculosis (MDR-TB). Many patients under

treatment suffered from depression and this was partly attributable to a loss of confidence in the health services due to previous treatment failures. Patients' trust in the treatment they are receiving is crucial if they are to continue with such a long treatment regimen. Treatment managers need to be aware of this and ensure that staff administering treatment regimens do all they can to win the trust and build the confidence of the patients in their care ***(Törün et al., 2005).***

Some drugs used in MDR-TB treatment also cause psychiatric disorders for example psychosis and depression has been reported as a side effect of Cycloserin (CS) and fluoroquinolones***(Vega et al., 2004).***

Psychiatric disorders in Respiratory Intensive Care Unit (ICU)

The most common reason for ICU admission is respiratory failure and the need for a mechanical ventilation ***(Angus et al., 2001)***. The terms intensive care unit (ICU) syndrome and ICU psychosis have been used interchangeably to describe a cluster of psychiatric symptoms that are unique to the ICU environment. It is often postulated that aspects of the ICU, such as sleep deprivation and sensory overload or monotony, are causes of the syndrome. ICU syndrome does not differ from delirium and caused exclusively by organic stressorson the central nervous system ***(McGuire et al., 2000).*** Delirium has received little attention in ICU settings because it is (1) rarely aprimary reason for admission, (2) often believed to be iatrogenic due to medications, (3) frequently explained away as "ICU psychosis,"and (4) believed to have no adverse consequences in terms of patients' ultimate outcome ***(Ely et al., 2004).*** The development of delirium in these mechanically ventilated patients was associated with a 3-fold increase in risk of death after controlling for

preexisting comorbidities, severity ofillness, coma, and the use of sedative and analgesic medications ***(Ferreir et al., 2001).***

Delirium is a disturbance of consciousness and attention; a change in cognition or perceptual disturbances,such as hallucinations; a rapid onset; and the assumption of an underlying medical cause ***(American Psychiatric Association, 1994)***. Management of patients with sepsis and multiorgan failure has traditionally been centered on dysfunction in the heart, lungs, or kidneys rather than the brain, though the brain is one of the organs most commonly involved ***(Papadopoulos et al., 2000).***

Monitoring for Delirium in the ICU:-

In the absence of data linking delirium to outcomes, few ICUs routinely monitor for delirium ***(Ely et al., 2004)***. Monitoring for delirium with the confusion assessment methods, which is easily incorporated by nurses into their daily work and takes only 1 to 2 minutes, could allow the medical team to consider causes and modifications in their treatment of the patient experiencing this organ dysfunction ***(American Psychiatric Association, 1999).***

Perhaps the greatest benefit of incorporating delirium monitoring would be the enhanced detection of the hypoactive delirium subtype, often called "quiet" delirium because it is characterized by a flat affect or apathy and often present in otherwise calmand seemingly alert patients. This is in contrast to the readily detected hyperactive delirium that is characterized by agitation, restlessness, attempting to remove catheters or tubes, hitting, biting, and emotional liability ***(Truman et al., 2003)***.

Potentially Risk Factors for delirium in ICU:-

Numerous risk factors for delirium have been identified, including preexisting cognitive impairment; use of psychoactive drugs; mechanical ventilation; untreated pain; and a variety of medical conditions such as heart failure, prolonged immobilization, abnormal blood pressure, anemia, sleep deprivation, and sepsis ***(Granberg et al., 2002).***

Some of the most readily implemented opportunities for improving care could be to correct brain ischemia and hypoxemia. Data from **Hopkins *et al., 1999*** study, showed that at the time of hospital discharge all acute respiratory distress syndrome (ARDS) survivors had observable cognitive impairments, including impaired memory, attention, concentration, and decreased mental processing speed. The cognitive impairments at the time of hospital discharge are likely to impair the patients' ability to remember and follow medication and discharge instructions.One year after onset of ARDS, we found that 78% of ARDS survivors demonstrated impaired cognitive function including impaired memory,attention, and concentration; 48% had decreased mental processing speed; and 30% had global cognitive decline.

Therapeutic approaches would be to modify the administration of psychoactive medications ***(Dubois et al., 2001)***, and to aggressively treat both underlying infection and the manifestations of severe sepsis, especially in elderly patients ***(Vincent et al., 2002).***

Psychiatric disorders in Pulmonary-arterial hypertension

In the context of living longer with Pulmonary-arterial hypertension (PAH) related symptoms, patients also must learn to face their unpredictable future and manage the complex treatments that can be

associated with severe adverse effects and may require significant changes in lifestyle.As a result, significant psychosocial issues (e.g., depression, anxiety, panic disorder, marital and work related problems) have emerged that are proving to be difficult for many patients ***(Kim et al., 2000).***

Löwe et al., 2004 examined psychiatric comorbidity in patients with PAH. Panic disorders and depression were significantly more prevalent in the PAH group. Moreover, the prevalence of psychiatric disorders showed a positive correlation with the functional status of the patients.

Coping and Emotional Well-Being:-

Coping has been defined as the active cognitive and behavioral adjustments made by an individual to manage both external and internal demands that are seen to be overwhelming and threatening to their sense of self. People cope in an attempt to maintain a sense of consistency, stability, and meaning while going through change ***(Lazarus and Folkman, 1984).***

This suggests that, for the PAH patient for whom reduced physical functioning is the norm, it is not the decrease in functioning that affects emotional well-being, but the impact of that change on the patient's ability to engage in activity that provides meaning and allows them to fulfill roles that they view as important.

The contributions of chronic illness-related stressors to an individual's sense of self and ability to adjust are many. For example, individuals with chronic illness often cope with some level of stigma, which can affect how they see themselves, whether they remain socially involved or become socially isolated, and, ultimately, how well they cope

with their illness ***(Sperry, 2006).*** Individuals must also cope with side effects from and adherence to medications, impaired activities of daily living, and fears related to disease progression ***(Horne and Weinman, 1999)***.

Psychosocial Adjustment:-

Successful psychosocial adaptation can be described as the ability to function within given interpersonal, social, and cultural norms, given new personal, social, and environmental demands created by one's illness. Factors affecting this adaptation include 1-psychological functioning (mood, self-concept, coping style), 2-disability-related consequences (level of functioning, ability to remain engaged in role activities or to redefine one's roles, employment), and 3- social systems (partner, family, friends, and community) ***(Livneh and Wilson, 2003).***

The reduced physical activity and exercise capacity found in this population is likely to affect more than social functioning. There is evidence that exercise benefits those suffering from depression and anxiety ***(Lawlor and Hopker, 2001).***

Psychiatric disorders in interstitial lung diseases

Idiopathic pulmonary fibrosis (IPF) is the most common of the interstitial lung diseases. Limited available data suggest that patients with IPF have significantly impaired health-related quality of life in both physical and psychological functions ***(Tomioka et al., 2007).*** Patients with IPF appear to have similar impairments in health-related quality of life as those with COPD, and the worsening quality-of-life score in IPF patients did not correlate with measures of dyspnea or pulmonary function ***(Swigris et al., 2005).***

Ryerson et al., 2012 reported that depression is common in IPF. In addition ***De Vries et al., 2001*** reported that approximately 25% of patients with IPF experience depressive symptoms. The burden of illness is commonly shared by a care partner who may also experience significant burden as the illness progresses. For this reason, care partners have been termed "the hidden patient"***(Roche, 2009)***.

Sarcoidosis primarily affects the respiratory system but often involves other organs. It is associated with a higher rate of psychiatric comorbidity, especially mood and anxiety disorders, and poorer quality of life *(Goracci et al., 2008)*. In addition, neurosarcoidosis, which affects 5% to 10% of sarcoidosis patients, can be the source of psychiatric symptoms, including depression, dementia, and psychosis ***(Shanmugam et al., 2007)***. Stress is thought to be an important factor in controlling sarcoidosis. The magnitude of stressful life events was significantly higher in patients with sarcoidosis when compared with healthy controls. In a recent study of 80 patients, 44% endorsed at least one major psychiatric diagnosis using a structured interview, with major depressive disorder being the most common ***(Goracci et al., 2008).*** In this study, individuals with multisystemic involvement, asthenia, and a more severe radiographic stage, along with those receiving steroids reported a poorer quality of life ***(Goracci et al., 2008).*** No study has evaluated specific psychological treatments in this patient population. However, one study showed that pulmonary rehabilitation(PR) is effective in improving exercise endurance and quality of life and in reducing hospital admissions in patients withrestrictive lung disease ***(Naji et al., 2005).***

Psychiatric disorders in Lung Cancer

Symptoms of psychological distress are common in lung cancer. One study showed that newly diagnosed lung cancer patients had frequent insomnia (52%), loss of libido (48%), loss of interest or ability to work (33%), concerns about their families (29%), and poor concentration (19%) ***(Ginsburg et al., 1995).*** Predictors of such psychological distress in ambulatory lung cancer patients included being female, living alone, having no children as confidants, relying on nursing staff as confidants, and having a helpless or hopeless coping style ***(Akechi et al., 1998).***

Surprisingly the type of lung cancer may influence the rate of depression. In one study, the rate of depression was nearly three times higher in those with small-cell cancer (25%) than in those with non–small cell cancer (9%). The most important risk factor for depression was functional impairment ***(Hopwood and Stephens, 2000)***. Another study showed that Pain management was the key in relieving depression ***(Akechi et al., 2001)***.

Fatigue is more common at diagnosis with lung cancer than most other cancers: 50% of those with inoperable non–small cell cancer reported severe fatigue. Key factors contributing to fatigue include disease burden, dyspnea, pain, and psychological distress ***(Stone et al., 2000)***. Adaptive behaviors can reduce fatigue even with low hemoglobin levels. Lung or colon cancer subjects who used non adaptive routines namely, disorganization, inertia, and overexertion reported more fatigue ***(Olson et al., 2002).***

Tanaka et al., 2002 found that dyspnea, very common in advanced lung cancer, was significantly correlated with psychological distress. Given the high rate of dyspnea in lung cancer patients, one might expect

that many patients would cease tobacco use. However, this is not the case, continued smoking after lung cancer diagnosis decreases treatment efficacy, increases complications, increases risk of recurrence and occurrence of another primary tumor, decreases survival time, emotional distress, greater nicotine dependence, and less self-efficacy (***Schnoll et al., 2002)***.

Do psychological factors alter the course of lung cancer? One study of lung cancer patients found that self-report of depressive coping was an independent predictor of decreased survival time at 8- and 10-year follow-up ***(Faller and Bulzebruck, 2002)***. Psychological factors may also influence response to treatment in other ways. For example anxiety or depression could predict increased nausea scores after chemotherapy ***(Takatsuki et al., 1998).***

Little has been written on coping in lung cancer. Four common coping strategies among patients with stages III and IV adenocarcinoma of the lung were suggested: seeking social support, problem solving, self-control, and positive reappraisal ***(Chernecky, 1999).***

Psychiatric disorders in Lung Transplantation

Psychiatric disorders are common in patients with end stage lung disease who are candidates for lung transplantation ***(Smoller et al., 1996).*** Half of the applicants for transplantation had a lifetime history of psychiatric illness such as lack of hope for the future, poor energy, poor sleep, and poor concentration ***(Craven and Toronto, 1990)***.

Although psychosocial contraindications to transplantation are difficult to quantify, active problems with cigarettes, alcohol, or illegal drug use have been associated with poor compliance with post

transplantation regimens and subsequently worsened likelihood of successful transplantation ***(Woodman et al., 1999).*** There are few data examining the impact of psychiatric diagnoses on lung transplantation outcomes and none specifically on survival in lung transplantation ***(Chacko et al., 1996)***.

Psychiatric Disorder and Quality of Life in *Lung Transplant*:-

Given the scarcity of donor lungs and the risk associated with the transplant surgery, careful screening including a thorough psychological assessment of potential lung transplant candidates is essential to ensure optimal outcomes ***(*Rockville*, 2001).***

As mentioned before end-stage pulmonary diseases are associated with significant psychological distress, with up to 47% of lung transplant candidates meeting criteria for at least one psychiatric disorder. There is a large body of evidence suggest that psychiatric illness and other psychosocial problems are related to poor post-transplant outcomes, such as more rejection episodes ***(Shapiro et al., 1995),*** increased length of hospitalization ***(Napolitano, et al., 2002),*** and higher levels of emotional distress ***(Beck et al., 1996).***

Although QOL is considered an important measure of post-transplant outcome, little attention has been devoted to the relation between psychiatric illness and QOL in candidates for this surgery. Because many patients awaiting lung transplantation will not survive long enough to receive a transplant, it is important to examine factors influencing quality of life before as well as after transplant. One implication of these findings is that identifying and treating psychiatric disorder in patients awaiting lung transplantation may improve their overall quality of life ***(Carney et al., 2003)***. Patients with end-stage

pulmonary disease who also are awaiting lung transplantation experience unique stressors, including separation from their support system due to relocation, a progressing course of illness, and fear of not receiving donor lungs in time**,** that make the waiting period a time of especially high risk for psychological distress ***(Goldberg, 1972)***.

Psychiatric disorder in patients awaiting transplant may be linked to adverse health outcomes as well as reduced QOL. For example, one study identified quality of well-being, a measure of physical and social functioning, as the strongest predictor of survival in lung transplant candidates ***(Scheier et al., 1994).***

Within the lung transplant literature, several studies showed that transplantation is associated with improvements in the QOL of patients ***(Tenvergert et al., 1998).*** More specifically, transplantation results in increased "overall QOL" and energy level, fewer physical and role limitations, as well as improved mental health, social functioning, and health perceptions ***(Limbos et al., 1997).***

Delirium in Lung transplantation:-

Because of the surgery, medications and hospitalization, it iscommon to experience sleep disturbance, restlessness, anxiety.

These symptoms may progress to delirium with patients experiencing agitation, hallucinations, confusion, changes in memory and concentration, and problems with speech and movement which may be caused by medications, changes in blood chemistry, and infection ***(University Health Network , 2010).***

Psychiatric disorders in OSAS

Introduction

Aloia et al., 2005 have investigated the presence of depression in patients with OSAS. Sleep apnoea is associated with clinical depression or increased levels of depressive symptoms, either as a direct consequence of the sleep deprivation or an indirect consequence of social effects of this illness.Moreover, 25% increase in hazard risk of mood and anxiety disorder has been observed in OSAS subjects ***(Bixler et al., 2005).***

Patients are aware of their sleeping problems, often reporting impaired sleeping habits. Considering the patients' everyday life style, many difficulties emerged, varying from poor parents and couple relationships to sexual disturbances. Daytime OSAS consequences are in fact, from the patient's perspective, extremely important (more than the nocturnal events often focused on by clinicians) (***Engleman and Douglas, 2004***).

1. OSAS and Depression

Farney et al., 2004 have shown that patients diagnosed with OSAS are at high risk of having had depression which may be caused by OSAS pathology or its symptoms ***(Harris et al., 2009).***

Peppard et al., 2006 has also shown an increased risk of developing depression as OSAS develops or worsens, and the relationship between severity of OSAS and depression is obvious.

Prevalence of OSAS in depression:-

Depression and OSAS are bidirectional interactive. Depression is associated with low adherence to CPAP treatment, increased sensitivity to symptoms and daytime fatigue similar to that caused by OSAS. It should be noticed that some hypnotics used as adjunct therapy in patients with depression, which may make OSAS more severe and change sleep staging, are prescribed extensively***(Feng et al., 2010).***

Traditionally, for major depression, polysomnography (PSG) findings reveal a shortened rapid eye movement (REM) latency with an increase in total percentage of REM sleep during the night, as well as in its eye movement density.The sleep of patients with OSAS is also fragmented, and contains a lot of transitional sleep stages (stage 1) at the expense of REM sleep and particularly of slow wave sleep (stages 3), in contrast to the sleep PSG of depressed patients, which characteristically shows a shorter latency of REM sleep, sleep apnea patients with depression displayed an increase in REM latency***(Feng et al., 2010)***.

Bardwell et al., 2000 compared sleep architecture of a group of 106 patients with and without OSAS and found that depressed patients who also had OSAS displayed a decrease in sleep latency compared to depressed patients without OSAS. OSAS subjects with depressive symptoms also had a higher percentage of REM sleep than OSAS subjects without depression. A study that included 40 treatment-naive OSAS subjects and 61 control subjects without diagnosed psychopathology, evaluated whole-brain maps of T2 relaxation time, the OSAS group was separated into 13 symptomatic and 27 asymptomatic subjects. The control group included 56 asymptomatic subjects. Relative to asymptomatic controls, symptomatic OSAS patients showed extensive

brain functional damage. Therefore, neural injury differed between OSAS patients with and without depressive symptoms. Depressive symptoms may exacerbate brain injury accompanying OSAS, or introduce additional extensive damage in the brain***(Cross et al., 2008).***

A substantive number of the individuals referred to a sleep center for evaluation of possible OSAS had been prescribed antidepressant medications prior to referral. When evaluated with the BDI, one can find that about 41% of these patients had a score suggesting at least mild symptoms of depression, and about 12% had a score suggesting moderate to severe symptoms of depression. These numbers seemed disproportionate to the incidence of depression in the general population***(Schwartez and Karatinos, 2007).***

Patients with OSAS will frequently report feeling tired, fatigued, sleepy, and poorly motivated***(Goncalves et al., 2004).***They may have difficulty concentrating or remembering factual data,they may become irritable or withdrawn, and they may find themselves losing interest in, or deriving little pleasure from activities which should be an integral part of their lives***(Bardwell et al., 2003).*** These consequences of OSAS may affect the individual's ability to perform their job and the daily activities of living, which is similar to depression. Whether the diagnostic problem is that the presenting symptoms in both conditions (OSAS and depression) are similar enough to result in the potential for the practitioner to confuse one with the other, leading to a misdiagnosis, or whether one disorder might increase the likelihood of the other occurring, is unknown***(Farney et al., 2004).***

Evaluation of depression in OSAS at primary care and sleep centers

Systematic assessment of depression symptoms with standardized clinical questionnaires in OSAS patients is often part of the evaluation process in all major sleep disorder centers. However, these questionnaires have not been specifically designed to assess depression in OSAS patients ***(Andrews and Oei, 2004).*** Therefore, they may not exactly accurate to assess depression in this population, given that it is still unclear if OSAS and depression display a true comorbidity or only share similar symptoms ***(Baran and Richert, 2003).*** Typically, patients with severe depression symptoms should be referred to a psychiatrist, particularly if such symptoms do not regress or if fatigue lingers after efficient treatment of OSAS***(Bardwell et al., 2003).***

Primary care physicians should also be aware of OSAS in women. Generally speaking, depression is seen in a statistically higher number of women than men. However, depression in a lot of these cases may be a complication of OSAS***(Wahner-Roedler et al., 2007).***When they complain of depression symptoms associated with OSAS, it is very important to consider referring women for sleep studies although a lower apnea-hypopnea index (AHI) may be identified, even if only non-specific symptoms are presented***(Dursunoglu et al., 2009).*** 92 men and 29 women were diagnosed with moderate to severe OSAS and were reviewed for depression symptoms using the BDI scores. Among them, a higher levels of depression symptoms in women than men. Mild depressive symptoms were found in 62% of women and 39% of men and moderate depression was present in 28% of women and 6% of men. It was suggested that depression symptoms in female patients with OSAS originate from factors other than traditional measures of OSAS severity because the BDI was positively related to the desaturation nadir and

milder oxygen desaturation nadirs were associated with worse depression scores***(McCall et al., 2006).***

The impact of depression on OSAS:-

Though there has been little research on the impact of depression in OSAS, patients who have depression as well as OSAS also appear worse off than their counterparts with OSAS only. OSAS patients with high levels of depression are those with most daytime sleepiness ***(Kjelsberg et al., 2005),***fatigue ***(Bardwell et al., 2007),*** and lowest quality of life scores ***(Akashiba et al., 2002).***

Effect of OSAS on depression

Schroder and O'Hara, 2005 found in a small sample of 17 older patients with major depression, that 17.6% also had an OSAS syndrome, compared to 4.3% of 23 healthy elderly controls. This suggests that OSAS might be an important confounding factor for studies on mood disorders in general, as its presence is not routinely determined in either research studies examining mood or clinical settings.

Clinically, this is of particular concern, as sedative antidepressants and adjunct treatments for depression may actually exacerbate OSAS. Notably hypnotics prescribed to treat depression-related insomnia might further decrease the muscle tone in the already functionally impaired upper airway muscles, blunt the arousal response to hypoxia and hypercapnia as well as increase the arousal threshold for the apneic event, therefore increasing the number and duration of apneas***(Guilleminault, 1990)***.

Possible mechanisms underlying the association between depression and OSAS

Sleep fragmentation and hypoxemia

The two main factors suspected to be responsible for depressive symptoms in OSAS are sleep fragmentation and oxygen desaturation during sleep. Sleep fragmentation is a direct consequence of the recurrent microarousals associated with the apneas and hypopneas, and the nocturnal hypoxemia is due to the intermittent drops in oxygen saturation caused by the respiratory events ***(Cohen-Zion et al., 2001).*** Sleep fragmentation is the primary cause of EDS in OSAS patients, and is suggested to result in the depressive symptomatology in OSAS. This last perspective gains support from the finding that EDS as measured by the Epworth Sleepiness Scale (ESS) and the Maintenance of Wakefulness Test (MWT) was found to be correlated with higher depression scores on the Hospital Anxiety Depression Scale in 44 patients with OSAS***(Sforza et al., 2002).*** Furthermore, a Canadian study on 30 OSAS patients showed a significant correlation between the severity of psychological symptoms and less total sleep time, as well as percentage of wake time after sleep onset and ESS scores ***(Yue et al., 2003)***. Preliminary imaging data suggests that hypoxemia related to OSAS might also play a role in impacting mood. Cerebral metabolic impairment resulting from recurrent nocturnal hypoxemia in OSAS have had previously been observed in several imaging investigations on OSAS. Independently, white matter hyperintensities (WMH) have been linked to depressive symptomatology in studies on affective disorders***(Firbank et al., 2004)***. In a small sample of older patients with OSAS, more subcortical WMH in the brain MRI of patients with a severe OSAS as compared to those with minimal OSAS, and a tendency for a positive correlation between these subcortical

hyperintensities and depression scores on the Hamilton Depression Scale ***(Aloia et al., 2004).***

Neurobiology of depression and upper airway control in OSAS: the role of serotonin

The high comorbidity of OSAS and depression also suggests that both disorders may share a common neurobiological risk factor. On the neurotransmitter level, the serotoninergic system has a central role as a neurobiological substrate underlying impairments in the regulations of mood, sleep-wakefulness cycle, and upper airway muscle tone control during sleep. Depression is associated with a functional decrease of serotoninergic neurotransmission, and is mostly responsible for the alterations in sleep***(Adrien, 2002)***. The physiopathology of OSAS involves numerous factors, among whose the abnormal pharyngeal collapsibility during sleep is one of the most compelling. Serotonin delivery to upper airway dilatator motor neurons has been shown to be reduced in dependency of the vigilance state ***(Veasey, 2003)***. This leads to reductions in dilator muscle activity specifically during sleep, which may contribute to sleep apnea. However, whereas the role of serotonin in mood disorders has been largely documented, its involvement in the pathophysiology of sleep apnea remains to be clarified. Interestingly, molecules increasing 5-HT neurotransmission such as the Serotonin reuptake inhibitors (SSRI) are widely prescribed antidepressant molecules that are suggested to similarly improve the apnea hypopnea index in OSA. Serotoninergic drugs such as fluoxetine, protryptiline and paroxetine have already been tested for OSAS, with limited success and numerous adverse effects***(Gami and Somers, 2004)***.

Shared risk factors

OSAS and depression share common risk factors, which may partly explain their high comorbidity in the general population. Very frequently in studies of the impact of OSAS on cognitive and psychological functioning, a conglomerate of disorders is shown to contribute to the overall neuropsychological outcome. Therefore, the presence of a polypathology often associated with OSAS, such as obesity, cardiovascular disease, hypertension and diabetes, should increase the suspicion of an underlying or coexisting OSAS in a depressed patient***(Schroder and O'Hara, 2005).***

Both, depression and OSAS, have independently been shown to be associated with metabolic syndrome, and also with the development of cardiovascular disease ***(Lett et al., 2004)***. The association between depression and metabolic syndrome has been suggested to be reciprocal and a priori not attributable to genetic factors ***(McCaffery et al., 2003)***. In particular, insulin resistance (IR) has been suggested to contribute to the pathophysiology of depressive disorder and has been proposed to subserve the association between depression and cardiovascular disease. Similarly, OSAS has been observed to be independently associated with the cardiovascular risk factors comprising metabolic syndrome ***(Coughlin et al., 2004)***, in particular IR. Up to 20% of all patients presenting with a diagnosed depressive syndrome may also have OSAS, and vice versa***(Schroder and O'Hara, 2005)***.

OSAS and depression share common risk factors, which may partly explain their high comorbidity in the general population. Both depression and OSAS have independently been shown to be associated with metabolic syndrome, cardiovascular disease***(Lett et al., 2004)*** and

antihypertensive or antidepressant prescribed medications ***(Farney et al., 2004).***

Ohayon's, 2003 large European cross-sectional study found that the association between OSAS and major depression persisted even when obesity and hypertension were controlled. Other factors associated with depression, such as age, gender, marital status, education, income (***Akhtar-Danesh and Landeen,2007)***and other chronic medical illnesses ***(Bentonet al., 2007)***could be included as possible confounders in observational studies of depression in OSAS.

Obesity is the most commonly suggested confounder that causes depression in some OSAS populations ***(Hashmi et al., 2006),*** Exercise is associated with both reduced depressive symptoms and reduced prevalence of OSAS ***(Daley, 2008),*** Gender shows higher rates of depression in women than in men***(Wahner-Roedler et al., 2007).*** However, these studies report high rates of depression or depressive symptoms in people with OSAS and point to higher rates among women than in men. Rates of depression have also been extracted from 24 reports of studies in OSAS to give a range of 7–63% (***Saunamaki and Jehkonen, 2007***), while in women depression scores were higher than men in those OSAS patients ***(Harris et al., 2009).***

Evidence and necessity of CPAP treatment on depression in OSAS:-

Schroder and O'Hara, 2005 have shown that depression symptoms improve with treatment of OSAS, and that untreated OSAS may contribute to treatment resistance in some cases of depression. Some depression symptoms commonly seen in the primary care setting are known to be associated with OSAS, and treatment of OSAS along with appropriate therapy for depression presents an opportunity to

simultaneously improve both conditions. Evidence showed more than 50% of patients with OSAS may experience depression; treatment of OSAS can decrease depression symptoms associated with this sleeping disorder***(Hirshkowitz, 2008).*** CPAP addresses the symptoms of OSAS and may reduce the risk of heart disease and depression associated with this sleep disorder. As a result, CPAP still should be the standard therapy for this comorbidity***(Rosenberg and Doghramji, 2009).***

Nasal CPAP is a highly effective treatment for OSAS. It significantly reduces sleepiness***(Giles et al., 2006)*** and seems to reduce hypertension***(Hla et al., 2002)***and improve sensitivity to insulin***(Babu et al., 2005)***. The most common reason for lack of patient benefit from CPAP is low compliance, and low rates of CPAP compliance are typical***(Noseda et al., 2000).*** CPAP compliance is likely to decrease if a patient tries treatment but does not perceive any treatment-related improvement***(Weaver et al., 2003)***and some patients do not experience a benefit***(Rosenthal et al., 2000).***Depression may diminish the subjective benefits of CPAP, thereby depriving the patient of one of the most important reasons to maintain this intrusive treatment. Across medical illnesses, depressed individuals report more symptoms, independent of the physiological severity of their medical condition. This seems to be true for individuals with OSAS as well. It is suggested that depressed individuals with OSAS may have more complaints about sleepiness and fatigue than nondepressed individuals with OSAS. The improvements in daytime sleepiness caused by CPAP may not be as salient if depression-related fatigue is present***(Katon and Ciechanowski, 2002)***. Therefore, low compliance presents a significant obstacle to the successful treatment of OSAS, and the perception of improvement of symptoms may influence CPAP adherence. It is possible, however, that there is a bidirectional

relationship, with adherence improving symptoms, and symptom improvement promoting sustained adherence to CPAP. The feedback loop between symptom improvement and CPAP adherence may be attenuated by the presence of depression. Individuals with depression tend to report more symptoms, regardless of the severity of their illness, but depression symptoms and daytime OSAS symptoms seem to improve concurrently during OSAS treatment***(Wells et al., 2007)***.

When treating patients with depression, OSAS should be considered as a contributing factor, and the treatment of OSAS could potentially improve not only the compliance to pharmacological antidepressant treatment, but also the treatment response rate for depression. In clinical practice, the presence of depression symptomatology is often assessed in patients with OSAS, and may be accounted for and followed up when considering treatment approaches and the response to treatment***(Schr der and O Hara, 2005)***.

Hypnotics prescribed to treat depression-related insomnia may decrease the muscle tone in the already functionally impaired upper airway dilatator muscles, blunt the arousal response to hypoxia and hypercapnia as well as increase the arousal threshold for the apneic event, therefore increasing the number and duration of apneas. These effects may differ depending on the patient population and the severity of OSAS.

Older depression subjects are of particular concern. Both the frequency of OSAS and the depression symptoms increase with age, as do consumption of prescription and sedative psychotropic medications. Pharmacologic treatment of depression and depression-related insomnia in this age group should therefore routinely consider the potential presence of a concomitant OSAS***(Feng et al., 2010)***.

In OSAS patients, it was confirmed that depression symptoms are dramatically and independently associated with increased fatigue and sleepiness, which are the major symptoms of OSAS. While the independent contributors to OSAS severity varied between studies, depression symptoms were the strongest predictor of fatigue and sleepiness. Depression symptoms explained ten times the variance in fatigue in OSAS patients than did OSAS severity itself: that is to say, OSAS severity explained 4.2% of the variance in fatigue while depression symptoms explained an additional 42.3%***(Bardwell et al., 2007)***.

Assessment and treatment of depression, not just treatment of OSAS itself, might reduce diurnal symptoms in OSAS patients, and that would be the most important in maintaining the compliance with CPAP treatment***(Bardwell et al., 2007)***.The efficacy of CPAP is contingent on patient compliance, and ≥4 hours of therapy per night may be required for patients with OSAS to experience significant clinical benefits. The association between daytime OSAS symptoms and CPAP compliance may reflect a "dose-response" relationship, with higher CPAP compliance associated with greater improvement***(Wells et al., 2007)***.

Pharmacologic therapies, like modafinil and armodafinil, may be of use in patients with OSAS to improve compliance with CPAP***(Rosenberg and Doghramji, 2009)***. Relief of the obstructive respiratory events with CPAP might ameliorate the symptoms of depression by improving sleep continuity, by ameliorating the adverse effects of various neurotransmitters (catecholamines or cortisol-related peptides)***(Buckley and Schatzberg, 2005)***, and by alleviating the adverse effects of any attendant hypoxemia***(Bardwell et al., 2007)***.

A 30% of 122 OSAS patients were non-compliant with CPAP treatment one month after beginning treatment. Decision-tree analysis indicated that it was possible to classify correctly 85.7% of the non-adherent patients using three baseline factors: emotional reactions score, age, and total score on the Apnea Beliefs Scale. Logistic regression analyses also confirmed depression symptoms as independent predictors of compliance. That means to improve compliance, supportive and educational measures for psychological well-being should be addressed to avoid patients abandoning CPAP treatment***(Poulet et al., 2009 and Baron et al., 2009).***

To assess the sustainability of improvements in depression symptoms using CPAP therapy in patients with OSAS, patients who demonstrated a significant response to CPAP were evaluated for symptoms of depression using the BDI. ***Schwartz et al., 2005*** found that CPAP therapy could result in a significant decrease in symptoms of depression assessed by the BDI at short-term (4–6 weeks) and long-term follow-up (at least 1 year) periods***(Schwartz and Karatinos, 2007)***. In 54 OSAS patients treated by CPAP, the BDI was administered before polysomnographic evaluation. A card embedded in the CPAP device electronically recorded compliance. The BDI was administered 1 to 2 months after the baseline measurements were obtained. Baseline depression symptoms were not correlated with mean duration of CPAP use per night, while reported improvements in OSAS symptoms correlated positively with CPAP adherence. There were significant positive correlations between improvements in depression symptoms and OSAS symptoms after initiation of CPAP therapy***(Wells et al., 2007)***.

Patients who had a respiratory disturbance index (RDI) ≥15 and demonstrated a significant response to CPAP (≥50% drop in RDI) were

evaluated for the symptoms of depression using the BDI. They were tested before and after 4 to 6 weeks of treatment with CPAP. CPAP therapy resulted in a significant decrease in depression symptoms. This change in BDI was noted both in those individuals who had received an antidepressant prescription prior to referral and in those who had not. Therefore, in these comorbidity patients, the symptoms of depression may be ameliorated after CPAP treatment***(Schwartz et al., 2005)***.

Depression symptoms experienced by OSAS patients are not solely the result of physical OSAS symptoms but include a mood component as well. ***Means et al., 2003*** investigated changes in both somatic and affective/cognitive symptoms of depression associated with the introduction of CPAP treatment for OSAS. The BDI was administered prior to treatment and again 3 months after CPAP. They found that after CPAP treatment, both somatic and affective/cognitive symptoms of depression improved in the same time, although adherence was predicted by baseline levels of depression***(Ayalon et al., 2006).*** Individuals with depression symptoms that continue during CPAP treatment are more likely to report continued fatigue and/or sleepiness, even with adequate CPAP treatment. Individuals who experience an improvement in depression symptoms also report an improvement in OSAS symptoms***(Wells et al., 2007)***.

Wells et al., 2007reported that depression symptoms and self-reported measures of daytime sleepiness both improved post CPAP treatment. If an individual does not report improvement in fatigue or sleepiness after using CPAP regularly, depression may be behind the disease.

Henke et al., 2001 found no difference in improvement in depression scores between the treatment and the control group over a short treatment duration (1–3 weeks). There were no significant differences with regards to the sample size, the initial severity of OSAS or the duration of CPAP therapy; which might explain the differences between studies observing an improvement after CPAP therapy and those who did not***(Henke et al., 2001)***. ***Lewis et al., 2004*** did not find any association between baseline depression scores and subsequent CPAP use for the first month of treatment. The effects of CPAP on mood may have been a placebo effect. Depression symptoms may respond differently during the course of CPAP treatment, depending on whether they precede the onset of OSAS, develop before but are exacerbated by OSAS, or are secondary to OSAS. Perhaps depression symptoms that begin during the course of OSAS respond more readily to CPAP, although long-standing symptoms of recurrent major depression disorder tend to persist even after successful CPAP treatment

Bardwell et al., 2007 parsed out the separate effects of treatment on depression symptoms in 38 OSAS patients; they were therapeutic CPAP, placebo CPAP, or O_2 supplementation. Depression decreased with O_2 supplementation but not with therapeutic CPAP or placebo CPAP. Results may suggest hypoxemia plays a stronger role than sleep disruption associated with OSAS related depression.

Monasterio et al., 2001 found that individuals with mild OSAS (AHI 10–30) had no significant improvement in depression scores after using CPAP. Perhaps the perceived benefits of treating milder OSAS do not outweigh the annoyances of wearing CPAP for some individuals. Similarly, the polysomnographic variables measured did not predict improvement in daytime OSAS symptoms or CPAP compliance. Those

individuals with baseline AHI between 40 and 80 experienced more symptom improvements than those with AHI <40 or >80, but patients who continued to experience OSAS symptoms after CPAP treatment also tended to have more depression symptoms after CPAP treatment. The individuals with AHI >80 also showed smaller improvements than those individuals with an AHI between 40 and 80. Perhaps other health problems caused individuals with AHI >80 to continue to sleep poorly despite CPAP use. Regression analyses indicate a curvilinear relationship exists between baseline severity of OSAS and perceived improvements post CPAP treatment***(Wells et al., 2007)***.

Giles et al., 2006 confirmed that continuous positive airway pressure (CPAP) therapy was effective in reducing sleepiness and improving quality of life measures in OSAS including depression.

Diagnostic scales for depression

There is a lot of scales for diagnosis of depression for example; The Beck Depression Inventory (BDI-I, BDI-II), created by ***Dr. Aaron T. Beck***, the three versions of the BDI—the original BDI, first published in 1961 and later revised in 1978 as the BDI-I, and the BDI-II, published in 1996.

Other scales for diagnosis of depression include:Beck Hopelessness Scale***(Beck***, ***1988),***Edinburgh Postnatal Depression Scale (EPDS)***(Cox et al., 1987)***, Geriatric Depression Scale (GDS)***(Yesavage et al., 1982-1983)***, Hamilton Rating Scale for Depression (HAM-D)created by ***Dr. Max Hamilton*** who originally published the scale in 1960 and revised it in 1966, 1967,1969, and 1980. Kutcher Adolescent Depression Scale (KADS)***(Kutcher, 2003)***, Major Depression Inventory (MDI) ***(Bech et al., 2001)***, Montgomery-Asberg Depression Rating Scale

(MADRS) (***Montgomery and Asberg, 1979)***, and Zung Self-Rating Depression Scale ***(Zung, 1965)***.

Lettau et al., 2010 found that almost all suicides are associated with and preceded by major depression. This medical depression results from altered brain chemistry and function due to the cumulative stress of ongoing inadequate sleep (either insufficient quantity or poor quality or both).

The challenge is to correct the root cause of the sleep problem, whether it is simple deprivation, obstructive sleep apnea syndrome (OSAS), or restless legs syndrome (RLS)/periodic limb movements in sleep (PLMS), and then observe the effect on depression, ideally without using anti-depressant drugs (***Novati et al., 2008***). Effective treatment of OSAS results in sustained improvement in depressive symptoms (***Schwartz &Karatinos, 2007***); in fact, one case report shows a patient whose intense suicidal ideation rapidly resolved solely in response to treatment of OSAS (***Krahn et al., 2008***).

Persons who get adequate sleep feel better and handle the stresses of everyday life better. It seems logical that restoration of an adequate quantity or quality of sleep would improve or even resolve depression and that a reduction in suicides would follow (Lettau ***et al., 2010***).

2. OSAS and Anxiety

Several studies have investigated the association of OSAS with anxiety; however, the relationship is still poorly understood increased awareness of the relationship between this psychiatric comorbidity and OSAS might significantly improve diagnostic accuracy as well as treatment outcome for both disorders ***(Schroder& OHara, 2005).***

Prevalence of anxiety varied widely, even between studies using the same method of assessment, showing a marked variety in the prevalence of anxiety (11–70%) (***Saunamaki & Jehkonen, 2007***).

Diagnostic scales for anxiety

Anxiety has been assessed by many scales such as Beck Anxiety Inventory***(Beck and Steer, 1993)***, Hamilton Anxiety Scale (HAM-A) ***(Maier et al., 1988),*** Hospital Anxiety and Depression Scale***(Zigmond and Snaith, 1983)***, Generalized Anxiety Disorder 7 (GAD-7)***(Spitzer etal., 2006)***, Panic and Agoraphobia Scale (PAS)***(Bandelow, 1995),*** Panic Disorder Severity Scale (PDSS)***(Shear et al., 1997),*** PTSD Symptom Scale – Self-Report Version***(Foa et al., 1997),*** Social Phobia Inventory (SPIN)***(Connor et al., 2000),*** Trauma Screening Questionnaire***(Brewin et al.,2002),*** Yale–Brown Obsessive Compulsive Scale (Y-BOCS)***(Goodman etal., 1989),*** and Zung Self-Rating Anxiety Scale ***(Zung, 1971)***.

Some patients prescribed positive airway pressure (PAP) therapy for OSAS experience claustrophobia, anxiety, or panic symptoms related to wearing the mask (feeling restricted) and/or tolerating the air pressure (feeling suffocated)***(Melanie et al., 2011)***.

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 et al., 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.

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).***

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).***

Exposure therapy for claustrophobia

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).***

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***(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).***

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).***

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 face 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).***

Koontz et al., 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).***

Additional therapeutic strategies for claustrophobia

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).***

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).***

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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 and 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 sleepservices due to its high demand and rewarding clinical outcomes ***(Means and Edinger, 2007).***

3. OSAS and Cognition impairment

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).***

OSAS patients complain of various neuropsychiatric symptoms. Cognitive impairment and affective disorders such as depression are frequently encountered in OSAS***(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.

Nocturnal panic attacks, diverse parasomnias, delirium, psychosis, personality change, and violent outbursts can be also reported in some OSAS patients ***(Sharafkhaneh et al., 2005)***.

Considerable research has examined neuropsychological deficits associated with OSAS. A meta-analysis of 25 studies (involving >2000 patients) was conducted to examine patterns of neuropsychological deficits in OSAS. 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 OSAS on general intelligence and verbal ability. The effects of OSAS 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 OSAS 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 after12 weeks 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, a measure of signal sensitivity, and the total number of target omissions ***(Chugh and Dinges, 2002)***.

OSAS 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. OSAS 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 (PVT) 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 (10) Screen shot of Perceptual Vigilance Test ***(Walker, 2009).***

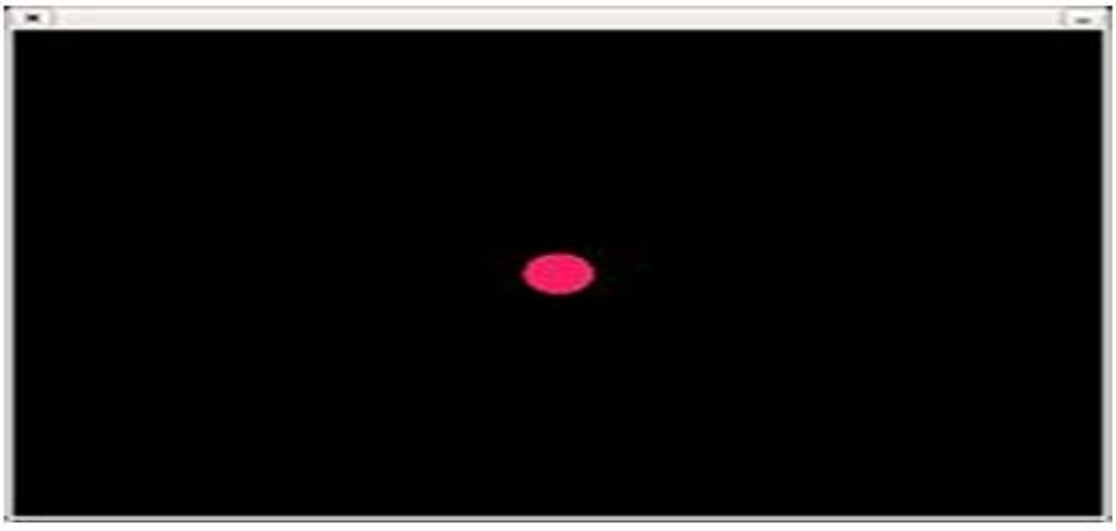
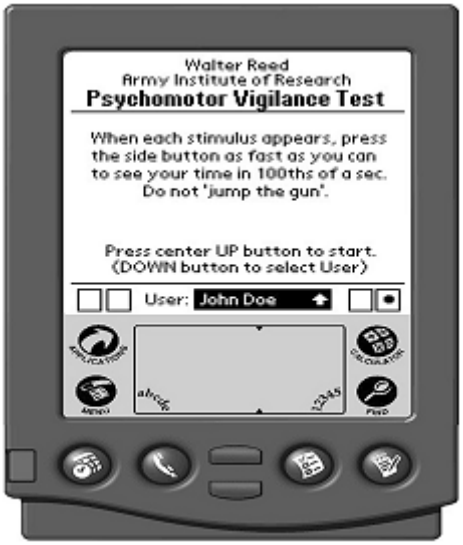


Figure (11) Psychomotor vigilance testing of professional drivers in the occupational health clinic ***(Wichniaket al., 2011).***

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 stimulantdrug 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 (12) Walter Reed Psychomotor vigilance test ***(Rollnick et al., 2008).***



- ***Memory impairment***

It should be noted that among OSAS 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 OSAS patients ***(Engleman et al., 2000).*** Diminished performance was described on short-term memory in patients with moderate and severe sleep apnea, although only the sever 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 OSAS 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 OSAS 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 OSAS, 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 OSAS 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 OSAS severity or 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 OSAS may be associated with frontal lobe dysfunction ***(Aloia et al., 2004)*** the prefrontal model has been proposed as a conceptual framework for the relationship between sleep disruption and nocturnal hypoxemia and primarily frontal deficits in OSAS. This model hypothesizes that OSAS-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 ***(Beebe and Gozal, 2002).***

In this model, OSAS 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)***.

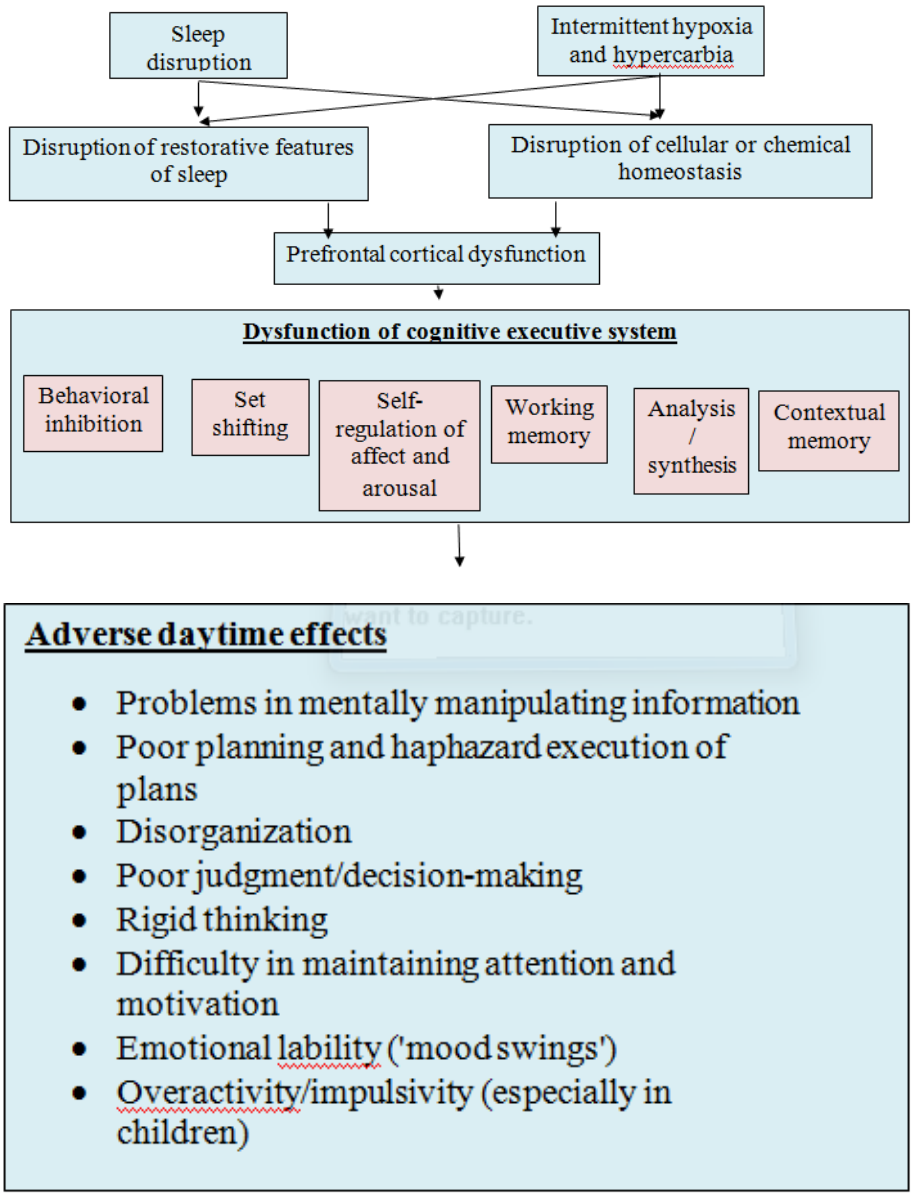
Volumetric studies in OSAS patients also show diminished gray matter in the hippocampus, nearby cerebral cortex, and cerebellar cortex and deep nuclei ***(Macey et al., 2002).*** The cerebellar damage in OSAS may contribute to loss of coordination of upper airway muscle activity(hypotonia of upper airway 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 OSAS. In an functional magnetic resonance imaging (fMRI) study, it was reported that untreated OSAS 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 OSAS patients by fMRI and found that verbal learning performance was similar in both the OSAS and control groups, but

OSASpatients 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 OSAS patients was felt to reflect an adaptive compensatory recruitment response.

Figure (13) The proposed prefrontal model. ***(Beebe and Gozal, 2002)***.



4. OSAS and Epilepsy

Excessive daytime sleepiness (EDS) is among the most common complaints of people with epilepsy, reported by 18–50% of adult patients ***(Piperidou et al., 2008).*** Excessive daytime sleepiness is often uniquely attributed to antiepileptic drugs (AEDs) and seizures. However, EDS is also one of the most common symptoms of obstructive sleep apnea syndrome (OSAS). By itself, OSAS is a highly prevalent disorder, affecting 24% of men and 9% of women (***Young et al, 1993***).

Sleep deprivation is an established seizure trigger. As such, OSAS has been identified as a contributor to excessive daytime sleepiness (EDS) and poor seizure control in epileptic patients (***Chihorek et al., 2007***). ***Malow et al., 2000*** reported a polysomnographic (PSG) study that involves patients with drug-resistant epilepsy unselected for sleep disorder symptoms, 33% of 39 adults had OSAS defined by an apnea–hypopnea index (AHI) ≥ 10. Those with OSAS were more likely to be male and have a higher BMI, snoring, witnessed apnea, and nocturnal seizures.

Small series suggest that nocturnal positive airway pressure (nPAP) therapy for OSAS reduces seizures in some cases ***(Vendrame et al., 2011).*** Given that seizures are incompletely controlled in 30–40% of epileptic patients. Those with OSAS were more likely to be male and have a higher BMI, snoring, witnessed apnea, and nocturnal seizures***(Mohanraj and Brodie, 2006***).

In another study, OSAS was confirmed by ambulatory PSG in 73% of 40 patients identified as OSAS suspects using a structured interview. This rate is similar to that achieved when assessing pre-test probability

for OSAS using validated screening tools and/or by clinicians with expertise in OSAS (***Farney et al., 2011***). Subjects with OSAS were also older, had a higher BMI, and were more likely to be male and older at the time of the first seizure.

In a study involving 21 adult patients, OSAS was confirmed in 82% of subjects with seizure onset/worsening at or after 50 years of age versus only 20% of those with earlier onset and seizure free or improving after age 50. While the groups were comparable in age, BMI, number of the anti-epileptic drugs (AEDs) and frequency of nocturnal seizures, males predominated in the late-onset/worsening group (***Chihorek et al., 2007***). Nevertheless, this study suggests that OSAS contributes to worsening seizure control or new-onset seizures in older adults.

Potential factors contributing to the increased prevalence of OSAS in epilepsy patients include the effects of AEDs (e.g., barbiturates, benzodiazepines) on upper airway tone, drug-induced weight gain, inactivity(***Ben-Menachem, 2007***).

Treatment of OSAS has been shown to reduce seizures in 40–86% of patients including adults and children (***Vendrame et al., 2011).***

Vendrame et al., 2011 found that nPAP adherence reduced seizure frequency by more than 15% (from 1.8 to 1 seizure per month) in 41 adults in the absence of AED adjustments. In addition, nPAP therapy reduced the spike rate in 8 adults with epilepsy and OSAS (***Oliveira et al., 2000),*** suggesting that sleep consolidation and perhaps correction of desaturation and hypercapnea reduce epileptogenicity.

The Psychomotor Epilepsy Scale is the only scale used for diagnosisof epilepsy***(Ali, 1994)***.

5. OSAS and Sexual dysfunction

Petersen et al., 2010 found that both general and functional aspects of sexuality were worse in patients with (untreated) OSAS when compared with normal persons. Both aspects were dependent on age, obesity, social factors and concomitant medication but not on the severity of OSAS as reflected by the apnoea-hypopnoea index or subjective sleepiness.

Several studies have suggested that reduced androgen secretion is associated with OSAS in middle-aged men. This is jointly caused by obesity and aging, with hypoxia and sleep fragmentation as contributing factors that may also decrease testosterone concentrations ***(Luboshitzky et al., 2001).***Some studies have indicated an association between OSAS and reduced testosterone release in the absence of complications. It is clear that in obese patients suffering from OSAS, the severity of hypoxia may be an additional factor in the reduction of testosterone levels, regardless of BMI and abdominal fat distribution ***(Gambineri et al., 2003).*** Furthermore, ***Cistulli et al., 1994*** demonstrated that testosterone levels were associated with an increase in upper airway collapsibility during sleep. This may be the mechanism by which testosterone induces or exacerbates OSAS. This suggests that both OSAS and coexisting comorbidities contribute to low testosterone concentrations and consequently may cause erectile dysfunction(ED).

Jankowski et al., 2008 suggested a possible pathway for the development of ED from OSAS through hypertension and diabetes, both of which are known to exhibit relatively strong associations with sleep apnea, even subclinical disturbances. The age-adjusted prevalence for AHI > 15 and OSAS were associated with a higher risk of ED

complaints. Thus, these factors are further evidence of the multiple ways that sleep disturbances can lead to alterations in erectile function through vasculogenic mechanisms (diabetes and stroke are connected with an increased risk of ED complaints with a key role for testosterone.

Fanfulla et al., 2000 observed that 48% of men with OSAS had sexual dysfunction and ED occurs in 30%-50% of men with OSAS.

Subramanian et al., 2010 have found that prevalence of sexual dysfunction is high among women with OSAS than men with OSAS.

Outcome of CPAP treatment on intimate and sexual relationships in men with obstructive sleep apnea:-

OSAS patients were impaired in intimate and sexual relationships. Following treatment, patients were significantly more alert and had reported improved intimate and sexual relationships, with the greatest change occurring in those with the most disease severity which means that OSAS has an adverse impact on intimate and sexual relationships that is related to subjective sleepiness and improved with CPAP treatment (***Reishtein et al., 2010).***

Diagnostic scales for sexual dysfunction

Sexual dysfunction has been assessed by many scales such asThe International Index of Erectile Function (LIFE-5) Questionnaire ***(Rosen, 1999)***, The Female Sexual Functioning Index (FSFI) ***(Rosen et al., 2000)***, Questions to Guide Sexuality Assessment among Older Adult ***(Wallace, 2000)***.

6. OSAS and Health- Related Quality Of Life

Another important finding in OSAS is the health-related quality of life (HRQOL), The findings that the HRQOL may play an important role in anxious or depressive state is reasonable because this reduced HRQOL reflects deficits in functioning across a number of areas (***Haynes, 2005***). Such difficulties may be so severe that job performances and family life may be affected, leading in turn to emotional disturbances and personality changes. These findings support the hypothesis that some of the psychopathological changes described in patients with OSAS are likely to reflect the reduced alertness and the reduced HRQOL related to breathing disorders (***Sforza et al., 2002***).

Moreover, the reduced HRQOL present in OSAS patients may affect their general health perception and their functional and emotional well-being inducing, in turn, personality changes such as aggressiveness, irritability, anxiety or depression, all expressing adaptation of the patients to their worsening life Condition (***Cassel,1993).***

Scales for assessment of psychiatric manifestations of OSAS

The scales used to diagnose common psychiatric manifestations of OSAS are important to be known by sleep specialists to help early diagnosis and rapid treatment so achieving better control of OSAS.

I. Depression

There is a lot of scales for diagnosis of depression for example; the Beck Depression Inventory (BDI-I, BDI-II), created by ***Dr. Aaron T. Beck***, the three versions of the BDI—the original BDI, first published in 1961 and later revised in 1978 as the BDI-I, and the BDI-II, published in 1996.

Other scales for diagnosis of depression includes Beck Hopelessness Scale***(Beck*,*1988),***Edinburgh Postnatal Depression Scale (EPDS)***(Cox et al., 1987)***, Geriatric Depression Scale (GDS)***(Yesavage et al., 1982-1983)***, Hamilton Rating Scale for Depression created by ***Dr. Max Hamilton*** who originally published the scale in 1960 and revised it in 1966, 1967,1969, and 1980.Kutcher Adolescent Depression Scale (KADS)***(Kutcher, 2003)***, Major Depression Inventory (MDI)***(Bech et al., 2001)***, Montgomery-Asberg Depression Rating Scale (MADRS) (***Montgomery and Asberg, 1979)***, and Zung Self-Rating Depression Scale ***(Zung, 1965)***.

The psychiatristsprefer to use Beck Depression Inventory (BDI-II) as it is one of the most widely used instruments for measuring the severity of depression, it is also translated into Arabic by ***Ibrahim Abd-***

Elsattar and is used in Arabian environment for about many years ago, it also has a sensitivity of (83 % -100 %) and specificity of (72% - 80%) ***(Low, 2007)***; therefore it is used in many studies ***(Hedayati 2006, Hermanns 2006 and Golden 2007).***

Beck Depression Inventory (BDI-II) composed of 21-question multiple-choice self-report inventory, in its current version the questionnaire is designed for individuals aged 13 and over, and is composed of items relating to symptoms of depression such as sadness, pessimism, sense of failure, lack of satisfaction, guilty feelings, sense of punishment, self-hate, self-accusations, self-punitive wishes, cry spells, irritability, social withdrawal, indecsiveness, worrying about body image, work inhibition, sleep disturbances, fatigability, loss of appetite, weight loss, somatic preoccupations, loss of lipido***(Beck, 2006).***

Beck's Depression Inventory Questionnaire (BDI-II):*(Beck et al., 1996)*

This depression inventory can be self-scored.

1. ***(Mood)***

0- I do not feel sad.
1- I feel sad
2 - I am sad all the time and I can't snap out of it.
3 - I am so sad and unhappy that I can't stand it.

2. ***(Pessimism)***

0- I am not particularly discouraged about the future.
1- I feel discouraged about the future.
2 - I feel I have nothing to look forward to.
3 - I feel the future is hopeless and that things cannot improve.

3. ***(Sense of failure)***

0 - I do not feel like a failure.
1 - I feel I have failed more than the average person.
2- As I look back on my life, all I can see is a lot of failures.
3 - I feel I am a complete failure as a person.

4. ***(Lack of satisfaction)***

0- I get as much satisfaction out of things as I used to.
1 - I don't enjoy things the way I used to.
2 - I don't get real satisfaction out of anything anymore.
3 - I am dissatisfied or bored with everything.

5. ***(Guilty feelings)***

0 - I don't feel particularly guilty
1- I feel guilty a good part of the time.
2- I feel quite guilty most of the time.
3- I feel guilty all of the time.

6. ***(Sense of Punishment)***

0- I don't feel I am being punished.
1 - I feel I may be punished.
2 - I expect to be punished.
3 - I feel I am being punished.

7. ***(Self-hate)***

0 - I don't feel disappointed in myself.
1 - I am disappointed in myself.
2 - I am disgusted with myself.
3- I hate myself.

8. ***(Self-Accusations)***

0- I don't feel I am any worse than anybody else.
1- I am critical of myself for my weaknesses or mistakes.
2- I blame myself all the time for my faults.
3- I blame myself for everything bad that happens.

9. ***(Self-punitive wishes)***

0- I don't have any thoughts of killing myself.
1- I have thoughts of killing myself, but I would not carry them out.
2-I would like to kill myself.
3- I would kill myself if I had the chance.

10.*(Cry Spells)*

0 - I don't cry any more than usual.
1- I cry more now than I used to.
2 - I cry all the time now.
3 - I used to be able to cry, but now I can't cry even though I want to.

11. ***(Irritability)***

0 - I am no more irritated by things than I ever was.
1 - I am slightly more irritated now than usual.
2 - I am quite annoyed or irritated a good deal of the time.
3 - I feel irritated all the time.

12.*(Social withdrawal)*

0 - I have not lost interest in other people.
1- I am less interested in other people than I used to be.
2 - I have lost most of my interest in other people.
3 - I have lost all of my interest in other people.

13.*(Indecsiveness)*

0 - I make decisions about as well as I ever could.
1 - I put off making decisions more than I used to.
2- I have greater difficulty in making decisions more than I used to.
3-I can't make decisions at all anymore.

14.*(Body image)*

0 - I don't feel that I look any worse than I used to.
1 - I am worried that I am looking old or unattractive.
2- I feel there are permanent changes in my appearance that make me look unattractive.
3- I believe that I look ugly.

15. ***(Work inhibition)***

0 - I can work about as well as before.
1- It takes an extra effort to get started at doing something.
2- I have to push myself very hard to do anything.
3- I can't do any work at all.

16.*(Sleep disturbances)*

0- I can sleep as well as usual.
1- I don't sleep as well as I used to.
2- I wake up 1-2 hours earlier than usual and find it hard to get back to sleep.
3 - I wake up several hours earlier than I used to and cannot get back to sleep.

17.*(Fatigability)*

0 - I don't get more tired than usual.
1- I get tired more easily than I used to.
2 - I get tired from doing almost anything.
3- I am too tired to do anything.

18.*(Loss of appetite)*

0 - My appetite is no worse than usual.
1 - My appetite is not as good as it used to be.
2- My appetite is much worse now.
3- I have no appetite at all anymore.

19.*(Weight loss)*

0- I haven't lost much weight, if any, lately.1- I have lost more than five pounds.
2- I have lost more than ten pounds.
3- I have lost more than fifteen pounds.

20.*(Somatic Preoccupations)*

0 - I am no more worried about my health than usual.
1 - I am worried about physical problems like aches, pains, upset stomach, or constipation.
2- I am very worried about physical problems and it's hard to think of much else.

3 - I am so worried about my physical problems that I cannot think of anything else.

21.*(Loss of Lipido)*

0 - I have not noticed any recent change in my interest in sex.
1- I am less interested in sex than I used to be.
2- I have almost no interest in sex.
3 - I have lost interest in sex completely.

The standard cut-offs are as follows:*(Beck et al., 1996)*

- 0–13 : Minimal depression
- 14–19: Mild depression
- 20–28: Moderate depression
- 29–63: Severe depression

II. Anxiety

Anxiety has been assessed by many scales such as Beck Anxiety Inventory***(Beck and Steer, 1993)***, Hamilton Anxiety Scale***(Maier et al., 1988)***, Hospital Anxiety and Depression Scale***(Zigmond and Snaith, 1983)***, Generalized Anxiety Disorder 7 (GAD-7)***(Spitzer et al., 2006)***, Panic and Agoraphobia Scale (PAS)***(Bandelow, 1995),***Panic Disorder Severity Scale (PDSS)***(Shear et al., 1997),***Post trauma stress disorder (PTSD) Symptom Scale – Self-Report Version***(Foa et al., 1997),*** Social Phobia Inventory (SPIN)***(Connor et al., 2000),*** Trauma Screening Questionnaire***(Brewin et al., 2002),*** Yale–Brown Obsessive Compulsive Scale (Y-BOCS)***(Goodman et al., 1989),***and Zung Self-Rating Anxiety Scale***(Zung, 1971)***.

Beck Anxiety Inventory: *(Beck and Steer, 1993)*

During the test, the respondent can select an answer from four choices:

- Not at all
- Mildly ("but it doesn't affect me much")
- Moderately ("it was unpleasant at times"), and
- Severely ("it bothered me greatly").

These answers are ascribed by the respondent according to the specific symptoms described in the inventory questions, such as muscle tension and tingling sensations, feelings of dread, hot or cold sweats, and so on. Every answer comes with a corresponding rating. The total sum of the ratings is then used to measure the respondent's anxiety level. The highest possible score is 63.

The following includes the list of symptoms of anxiety asked in the Beck inventory. Review them individually and choose one of the answers, 1 being "Not at all", 2 "Mildly", 3 "Moderately" and 4 "Severely".

1. Feeling hot
2. Muscle numbness or tingling
3. Feeling unable to relax
4. Dizzy or light headed
5. Feeling wobbly in the legs
6. Feeling unsteady
7. Heart racing or pounding
8. Nervousness
9. Choking feeling
10. Trembling hands

11. Unsteadiness
12. Terror or fear
13. Afraid of losing control
14. Indigestion
15. Flushed face
16. Hot or cold sweats
17. Feeling scared
18. Having laborious breathing
19. Feeling the fear of dying
20. Feeling like the worst is happening
21. Feeling faint

Interpretation of Scores

- 0-21: Very low anxiety
- 22-35: Moderate anxiety
- ≥ 36: Severe anxiety

Taking the Beck anxiety inventory test is easy and provides relatively accurate results about stress and anxiety levels. The inventory can be retaken at different periods of time to view one's progress with dealing with their anxiety issues.

Beck Anxiety Inventory:

Nº	Symptoms	How much you were bothered			
		Nothing *0*	*Weak* *1*	*Moderate* *2*	*Strong* *3*
		It did not bother at all	*It bothered a little*	*It bothered me a lot but I could stand it*	*I almost could not stand it*
1	Numbness or tingling				
2	Hot sensation				
3	Wobbly				
4	Incapable of relaxing				
5	Fear of the worst happening				
6	Dizziness or lightheadedness				
7	Heart pounding or racing				
8	Restless				
9	Terrified				
10	Nervous				
11	Feeling of suffocation				
12	Hands trembling				
13	Trembling				
14	Fear of losing control				
15	Difficulty breathing				
16	Fear of dying				
17	Frightened				
18	Indigestion or discomfort in the abdomen				
19	Fainting				
20	Red Face				
21	Sweating (not due to heat)				
SCORE:					

The psychiatrists prefer to use Hospital Anxiety and Depression Scale as it iscommonly used by doctors to identify cases (possible and probable) of anxiety disorders and depression among patients in nonpsychiatric hospital clinics especially in Arabicenvironment as it is translated by ***Sameh EL-Gamal*** and has a specificity for anxiety (HADS-A) about 78% and a sensitivity 90%, for depression, HADS-Dgave a specificity of 79% and a sensitivity of 83%***(Bjelland et al., 2002);*** therefore it is used in many studies ***(Stafford et al., 2007, Haworth et al., 2007, Golden et al., 2007).***

The Hospital Anxiety and Depression Scale (HADS) is a fourteen item scale that generates ordinal data. Seven of the items relate to anxiety and seven relate to depression. ***Zigmond and Snaith, 1983*** created this outcome measure specifically to avoid reliance on aspects of these conditions that are also common somatic symptoms of illness, for example fatigue and insomnia or hypersomnia. This, it was hoped, would create a tool for the detection of anxiety and depression in people with physical health problems.

The HADS is a self-administrated scale, which consists of seven questions related to anxiety and seven related to depression with answering categories ranging from 0 to 3.For this scale, it was found that a score of 7 or less for non – case (normal), scores of 8-10 for doubtful cases (borderline) and scores for 11 or more for definite cases **(Zigmond et al., 1983)**.

The HADS measures depression and generalized anxiety in inpatients and outpatients and in community settings. It contains 14 statements describing symptoms of depression and anxiety (for example " I feel tense and irritable "). Response options for each questions range from o to 3 and ask patients about their agreement with the statements or how often they apply (for example "most of the time, often, from time to time or not at all ").

<u>Hospital Anxiety and Depression Scale (HADS):</u> (Zigmond et al., 1983).

(*) A ——>> Anxiety

(**) D ——>>Depression

1- *A-I feel tense or 'wound up':

3- Most of the time

2- A lot of the time

1- From time to time, occasionally

0- Not at all

2- **D- I still enjoy the things I used to enjoy:

0- Definitely as much.

1- Not quite so much.

2- Only a little.

3- Hardly at all.

3- *A- I get a sort of frightened feeling as if something awful is about to happen:

3- Very definitely and quite badly.

2- Yes, but not too badly.

1- A little, but it doesn't worry me.

0- Not at all.

4- **D- I can Iaugh and see the funny side of things:

0- As much as I always could.

1- Not quite so much now.

2- Definitely not so much now.

3- Not at all.

5- *A-Worrying thoughts go through my mind:

3- A great deal of the time.

2- A lot of the time.

1- From time to time but not too often.

0- Only occasionally.

6- **D- I feel cheerful:

3- Not at all.

2- Not often.

1- Sometimes.

0- Most of the time.

7- *A-I can sit at ease and feel relaxed:

0- Definitely.

1- Usually.

2- Not often.

3- Not at all.

8- **D- I feel as if I am slowed down:

3-Nearly all the time.

2-Very often.

1-Sometimes.

0-Not at all.

9- *A-I get a sort of frightened feeling like 'butterflies in the stomach:

0- Not at all.

1- Occasionally.

2- Quite often.

3- Very often.

10-D- I have lost interest in my appearance:**

3- Definitely.

2- I don't take so much care as I should.

1- I may not take quite as much care.

0- I take just as much care as ever.

11-*A-I feel restless as if I have to be on the move:

3-Very much indeed.

2-Quite a lot.

1-Not very much.

0-Not at all.

12-D-I look forward with enjoyment to things:**

0-As much as ever I did.

1-Rather less than I used to.

2-Definitely less than I used to

3-Hardly at all.

13-*A-I get sudden feelings of panic:

3-Very often indeed.

2-Quite often.

1-Not very often.

0-Not at all.

14-D-I can enjoy a good book or radio or TV programme:**

0-Often.

1-Sometimes.

2-Not often.

3-Very seldom.

Anxiety cutoff ≥10.

Depression cutoff ≥7.

GRADING:

- 0 - 7 = Non-case
- 8 – 10 = Borderline case
- 11+ = Case

III. Cognitive impairment

- ***Sustained attention (vigilance)***

Vigilance 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.

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 and 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).***

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).***

- ***Memory impairment***

The most common scales used for assessment of memory impairment are the Wechsler Memory Scale (WMS)***(Kaufman and Alan, 2006)***,the Mini-Mental Status Examination Scale(MMSE)***(Folstein et al., 1975),***the Severe Impairment Battery (SIB) ***(Panisset et al., 1994)*** and the Preliminary Neuropsychological Battery (PNB)***(Cossa et al., 1996),***but psychiatrists chose the MMSE as it is probably the most widely used screening instrument for the detection of an individual's cognitive impairment and quantification of its severity. The scale is available in many languages and has been validated in various countries***(Tombaugh and McIntyre, 1992).*** Althoughthe MMSE scale was developed with the intent to provide a short and global assessment of cognitive function in order to differentiate between organic and functional psychiatric patients, it has attained widespread use among clinicians and researchers concerned in both primary care***(White et al., 2002)***and community-based settings***(Aevarsson and Skoog, 2000 and Maki et al., 2000).*** Rapid mental status assessments have gained increasing popularity as a mean of evaluating the presence and severity of neurobehavioral deficits and exploring the integrity of cognitive abilities among patients.

Folstein et al.,1975 originally described the MMSE as consisting of 11 items grouped into five cognitive domains-orientation, registration, attention-calculation, recall, and language including the item of visual construction, in order to capture similar information as that of a typical neuropsychological or neurological mental status examination.

Mini Mental State Examination (MMSE) Scale:*(Folstein et al., 1975*)

I. **ORIENTATION** (Ask the following questions; correct = ☑)	**Record Each Answer:**	(Maximum Score = 10) Score: ____
What is today's date?	Date (eg, May 21)	1 ☐
What is today's year?	Year	1 ☐
What is the month?	Month	1 ☐
What day is today?	Day (eg, Monday)	1 ☐
Can you also tell me what season it is?	Season	1 ☐
Can you also tell me the name of this hospital/clinic?	Hospital/Clinic	1 ☐
What floor are we on?	Floor	1 ☐
What city are we in?	City	1 ☐
What county are we in?	County	1 ☐
What state are we in?	State	1 ☐
II. IMMEDIATE RECALL	(correct = ☑)	(Maximum Score = 3) Score: ____
Ask the subject if you may test his/her memory. Say "ball, "flag," "tree" clearly and slowly, about on second for each. Then ask the subject to repeat them. Check the box at right for each correct response. The first repetition determines the score. If he/she does not repeat all three correctly, keep saying them up to six tries until he/she can repeat them	Ball	1 ☐
	Flag	1 ☐
	Tree	1 ☐
		NUMBER OF TRIALS: _____
III. **ATTENTION AND CALCULATION**		
A. Counting Backwards Test	(Record each response, correct = ☑)	(Maximum Score = 5)
Ask the subject to begin with 100 and count backwards by 7. Record each response. Check one box at right for each correct response. Any response 7 or less than the previous response is a correct response. The socre is the number of correct subtractions. For example, 93, 87, 80, 72, 66 is a score of 4; 93, 86, 78 70, 62, is 2; 92, 87, 78, 70, 65 is 0.	93	1 ☐
	86	1 ☐
	79	1 ☐
	72	1 ☐
	65	1 ☐
B. Spelling Backwards Test		
Ask the subject to spell the word "WORLD" backwards. Record each response. Use the instructions to determine which are correct responses, and check one box at right fore each correct response.	D	1 ☐
	L	1 ☐
	R	1 ☐
C. Final Score	O	1 ☐
Compare the scores of the Counting Backwards and Spelling Backwards tests. Write the greater of the two socres in the box labeled FINAL SCORE at right, and use it in deriving the **TOTAL SCORE**.	W	1 ☐
		FINAL SCORE_____ (Max of 5 or Greater of the two Scores)
IV. **RECALL**	(correct = ☑)	(Maximum Score = 3) Score: ____
Ask the subject to recall the three words you previously asked him/her to remember. Check the Box at right for each correct response.	Ball	1 ☐
	Flag	1 ☐
	Tree	1 ☐
V. Language	(correct = ☑)	(Maximum Score = 9) Score: ____
Naming	Watch	1 ☐
Show the subject a wrist watch and ask him/her what it is. Repeat for a pencil.	Pencil	1 ☐
Repetition		

Ask the subject to repeat "No, ifs, ands, or buts."	Repetition	1 ☐
Three -Stage Command		
Establish the subject's dominant hand. Give the subject a sheet of blank paper and say, "Take the paper in your right/left hand, fold it in half and put it on the floor."	Takes paper in hand Folds paper in half Puts paper on floor	1 ☐ 1 ☐ 1 ☐
Reading		
Hold up the card that reads, "Close your eyes." So the subject can see it clearly. Ask him/her to read it and do what it says. Check the box at right only if he/she actually closes his/her eyes.	Closes eyes	1 ☐
Writing		
Give the subject a sheet of blank paper and ask him/her to write a sentence. It is to be written sponataneously. If the sentence contains a subject and a verb, and is sensible, check the box at right. Correct grammar and punctuation are not necessary.	Writes sentence	1 ☐
Copying		
Show the subject the drawing of the intersecting pentagons. Ask him/her to draw the pentagons (about one inch each side) on the paper provided. If ten angles are present and two intersect, check the box at right. Ignore tremor and rotation.	Copies pentagons	1 ☐

The maximum total score is 30.

- 23-30 = Normal
- 19-23 = Borderline
- <19 = Impaired

MMSE also has both good test–retest reliability (80%–95%) ***(Tombaugh and McIntyre, 1992)*** and acceptable sensitivity (86%) and specificity (92%) in what the authors define 'organic mental disorders ***(O'Connor et al., 1989).***

During the last two decades, several studies have used the MMSE scale ***(Noale et al., 2006, Schultz-Larsen et al., 2007a and Schultz-Larsen et al., 2007b).***

- ***Executive functions***

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 OSAS, 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).***

IV- Epilepsy

The Psychomotor Epilepsy Scale is the only scale used in the international neuropsychological environment to help psychiatrists, neurologists and psychologists to confirm the diagnosis of epilepsy in association with symptoms and signs even without electroencephalography (EEG) inspite of its importance; In addition, it has a validity of (85%-100%) and reliability of (75%-100%) ***(Ali, 1994)***.

The Psychomotor Epilepsy Scale consists of 30 items presenting 30 symptoms that are most famous in psychomotor epileptical fits which are divided into:

1) Memory and Cognitive impairment: 1, 7, 13, 19, 25.

2) Affective disorder: 2, 8, 14, 20, 26.

3) Sensory disorder: 3, 9, 15, 21, 27.

4) Autonomic Nervous System disorder: 4, 10, 16, 22, 28.

5) Perceptual disorder: 5, 11, 17, 23, 29.

6) Thought disorder: 6, 12, 18, 24, 30.

So, every set of the scale has 5 items and are arranged in a circular manner with 4 possibilities (almost, sometimes, rarely, never), and patient has to choose the item that fits him and every item has a score (0, 1, 2, 3).The net result ranges from (0- 90) and the cutoff point is ≥45.

مقياس الأعراض النفسية الحركية

مقياس (ص . ن . ح)

إعداد:

د . سامى عبد القوى على

مدرس علم النفس

جامعة عين شمس

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-الإسم :

- الجنس : ذكر ( ) - أنثى ( )

-السن:

- المستوى التعليمى:

- تعليمات تطبيق المقياس:

أمام كل عرض عدة اختيارات (غالبا ، أحيانا ، نادرا ، أبدا) ، ومطلوب اختيارإجابة واحدة فقط، تعبر عن مدى شدة وجود العرض .

الرقم	السؤال	غالبا	أحيانا	نادرا	أبدا
١	هل لاحظت إن تركيزك في الأيام اللى فاتت قل عن الطبيعى ؟				
٢	هل مرات تلاقى نفسك خايف وقلبك مقبوض بدون سبب، وحاسس كأن فيه مصيبة حتحصل ؟				
٣	هل بتحس بزغللة في عينيك، أو كأن الدنيا ضباب قدامك، وتبقى مش شايف كويس ؟				
٤	هل قلبك فجأة كدة يرفرف عليك ويدق جامد ؟				
٥	هل مرات تشم ريحة غريبة بدون سبب، يعنى مثلا تشم ريحة شياط أو بخور أو بلاستيك محروق من غير ما يكون فيه سبب؟				
٦	هل مرات تعمل حاجات ويقولولك إنك عملتها وتبقى مش فاكر إنك عملت الحاجات دى ؟				
٧	هل تلاقى نفسك بتسرح كتير، ومش عارف إنت سرحان في إيه ؟				
٨	هل مرات تحس إنك مكتئب أو متضايق وزهقان بدون ما يكون فيه سبب ؟				
٩	هل بيجيلك صداع شديد ، وكأن دماغك هتنفجر ؟				

١٠	هل مرات تلاقى نفسك جعان قوى، وتاكل كتيروتحس إنك مش شبعان ؟				
١١	هل مرات تحس إن الدنيا حواليك زى ما تكون أتغيرت وبقت غريبة شوية عليك ؟				
١٢	هل مرات تحس وإنت صاحي إن الحاجات اللى بتحصل حواليك كأنها حلم مش حقيقة ؟				
١٣	هل مرات وإنت بتتكلم في موضوع لأول مرة تحس إن الكلام ده زى ما يكون حصل قبل كده ؟				
١٤	هل مرات تحس إنك عايز تعيط أو تصرخ بصوت عالى غصب عنك وبدون سبب ؟				
١٥	هل مرات تحس برجفة في جسمك زى ما تكون أتكهربت فجأة ؟				
١٦	هل بطنك بتمغص عليك، أو تحس بكلبشة في فم المعدة ؟				
١٧	هل مرات تسمع صوت في ودانك زى الزنة، أوزى صوت الجرس من غير سبب ؟				
١٨	هل مرات يقولولك إنك بتتصرف تصرفات غريبة،وإنت مش واخد بالك إنك عملت التصرفات دى ؟				
١٩	هل بتلاقى صعوبة إنك تفتكر الحاجات العادية،يعنى تحس إن ذاكرتك ضعفت وبقيت تنسى ؟				
٢٠	هل بتحس إنك قلقان وعصبى زيادة عن اللازم من غير سبب ؟				
٢١	هل بتحس بتنميل أو شكشكة في أي حتة من جسمك أو حتى جسمك كله ؟				
٢٢	هل مرات تحس بضيق في التنفس، وتبقى مش قادر تاخد نفسك، أو إن نفسك مخنوق ؟				
٢٣	هل مرات تحس أن فيك حاجة غريبة، أو إنك أتغيرت، أو تحس إن أنت مش أنت ؟				
٢٤	هل مرات يغمى عليك، وتفوق من غير ما تعرف أيه اللى حصل ؟				
٢٥	هل مرات تحس إن الأماكن اللى تعرفها كويس كأنها غريبة عليك، أو كإنك بتشوفها لأول مرة؟				
٢٦	هل بتلاقى نفسك فجأة مبسوط وبتضحك وماتقدرش تمسك نفسك من الضحك ؟				
٢٧	هل مرات تحس بتنميل أو تقل في لسانك، وتحس إن كلامك إتلخبط شوية ؟				
٢٨	هل مرات تحس بالدوخة، أو زى ما تكون الدنيابتلف بيك ؟				

٢٩	هل مرات تلاقى الحاجات بتكبر أو بتصغر قدامك، أو كأنها بتبعد عنك ؟				
٣٠	هل مرات تحس فجأة إن فيه أفكار غريبة مالهاش معنى تيجى على بالك ، وتبقى مش قادر تتحكم فيها ؟				
	المجموع =				
	الدرجة =				
	الدرجة الكلية =				

يتكون المقياس من ٣٠ عبارة تقيس ٣٠ عرضا، هي الأكثر شيوعا في نوبات الصرع النفسي الحركي. ويمكن تصنيف هذه العبارات إلى فئات الأعراض التالية :

- أعراض اضطراب التركيز والذاكرة: وتشمل عبارات رقم ١، ٧، ١٣ ، ١٩ ، ٢٥ .
- الأعراض الوجدانية: وتشمل عبارات رقم ٢، ٨ ، ١٤ ، ٢٠ ، ٢٦.
- الأعراض الحسية: وتشمل عبارات رقم ٣ ، ٩ ، ١٥ ، ٢١ ، ٢٧.
- أعراض الجهاز العصبي الارادى: وتشمل عبارات رقم ٤ ، ١٠ ، ١٦ ، ٢٢ ، ٢٨ .
- أعراض اضطراب الإدراك: وتشمل عبارات رقم ٥ ، ١١ ، ١٧ ، ٢٣ ، ٢٩ .
- أعراض اضطراب التفكير والوعي : وتشمل عبارات رقم ٦ ، ١٢ ، ١٨ ، ٢٤ ، ٣٠.

الحركي شملت ٥ عبارات،وتم ويتبين من ذلك أن كل فئة من أعراض الصرع النفسيترتيبها بطريقة دائرية. ووضع الباحث للإجابة على كل عبارة ٤ احتمالات هي (غالبا) ، (أحيانا) ، (نادرا) ، (أبدا) . وعلى المفحوص اختيار إجابة واحدة من هذه البدائل الأربعة. وتعطى كل استجابة درجة واحدة من أربعة درجات هي ٣، ٢، ١، صفر، وذلك على التوالي. وبذلك تتراوح الدرجة الكلية على المقياس بين صفر - ٩٠ درجة . وتعتبر الدرجة ٤٥ فما فوق مؤشرا قويا لذلك .

V- Sexual dysfunction

Sexual dysfunction has been assessed by many scales such as,The International Index of Erectile Function (IIEF) Questionnaire ***(Rosen, 1999)***, The Female Sexual Functioning Index (FSFI) ***(Rosen et al., 2000)***, Questions to Guide Sexuality Assessment among Older Adult ***(Wallace, 2000)***.

The psychiatrists prefer to use the International Index of Erectile Function (IIEF) Questionnaire to evaluate erectile dysfunction in men.

The International Index of Erectile Function (IIEF) Questionnaire:*(Rosen, 1999)*

The questions are based on last 6 months (each answer assigned 1-5 score)

1. How do you rate your confidence that you could get and keep an erection?

1. Score 1: Very Low
2. Score 2: Low
3. Score 3: Moderate
4. Score 4: High
5. Score 5: Very High

2. When you had erections with sexual stimulation, how often were your erections hard enough for penetration?

1. Score 1: Never or almost never
2. Score 2: A few times
3. Score 3: Sometimes (about half the time)
4. Score 4: Most times
5. Score 5: Always or almost always

3. During sexual intercourse, how often were you able to maintain your erection after you had penetrated or entered your partner?

1. Score 1: Never or almost never
2. Score 2: A few times
3. Score 3: Sometimes (about half the time)
4. Score 4: Most times
5. Score 5: Always or almost always

4. During sexual intercourse, how difficult was it maintain your erection to completion of intercourse?

1. Score 1: Extremely difficult
2. Score 2: Very difficult
3. Score 3: Difficult
4. Score 4: Slightly difficult
5. Score 5: Not difficult

5. When you attempted sexual intercourse, how often was it satisfactory for you?

1. Score 1: Never or almost never
2. Score 2: A few times
3. Score 3: Sometimes (about half the time)
4. Score 4: Most times
5. Score 5: Always or almost always

Interpretation:

Based on total sum of the ordinal responses to the 5 items.

- Scores 22 to 25: Normal erectile function
- Scores 17 to 21: Mild erectile dysfunction
- Scores 12 to 16: Mild to moderate erectile dysfunction
- Scores 8 to 11: Moderate erectile dysfunction
- Scores 5 to 7: Severe erectile dysfunction

Female Sexual Function Index (FSFI)

The Female Sexual Function Index (FSFI), a 19-item questionnaire, has been developed as a brief, multidimensional self-report instrument for assessing thekey dimensions of sexual function in women***(Rosen et al., 2000).***

These questions ask about sexual feelings and responses during thepast 4 weeks. Responses will be kept completely confidential.In answering these questions the following definitions apply:

Sexual activity can include caressing, foreplay, masturbation, and vaginal intercourse.

Sexual intercourse is defined as penile penetration (entry) of the vagina.

Sexual stimulation includes situations like foreplay with a partner, self-stimulation(masturbation), or sexual fantasy.

Sexual desire or interest is a feeling thatincludes wanting to have a sexual experience,feeling receptive to a partner's sexualinitiation, and thinking or fantasizing abouthaving sex.

Sexual arousal is a feeling that includes both physical and mental aspects of sexual excitement. It may include feelings of warmth or

Tingling in the genitals, lubrication (wetness), or muscle contractions.

1. Over the past 4 weeks, how often didyou feel sexual desire or interest?

5 = Almost always or always

4 = Most times (more than half the time)

3 = Sometimes (about half the time)

2 = A few times (less than half the time)

1 = Almost never or never

2. Over the past 4 weeks, how wouldyou rate your level (degree) of sexualdesire or interest?

5 = Very high

4 = High

3 = Moderate

2 = Low

1 = Very low or none at all

3. Over the past 4 weeks, how often didyou feel sexually aroused ("turned on")during sexual activity or intercourse?

0 = No sexual activity

5 = Almost always or always

4 = Most times (more than half the time)

3 = Sometimes (about half the time)

2 = A few times (less than half the time)

1 = Almost never or never

4. Over the past 4 weeks, how would yourate your level of sexual arousal ("turnon") during sexual activity or intercourse?

0 = No sexual activity

5 = Very high

4 = High

3 = Moderate

2 = Low

1 = Very low or none at all

5. Over the past 4 weeks, how confident wereyou about becoming sexually aroused duringsexual activity or intercourse?

0 = No sexual activity

5 = Very high confidence

4 = High confidence

3 = Moderate confidence

2 = Low confidence

1 = Very low or no confidence

6. Over the past 4 weeks, how often haveyou been satisfied with your arousal(excitement) during sexual activity orintercourse?

0 = No sexual activity

5 = Almost always or always

4 = Most times (more than half the time)

3 = Sometimes (about half the time)

2 = A few times (less than half the time)

1 = Almost never or never

7. Over the past 4 weeks, how often didyou become lubricated ("wet") duringsexual activity or intercourse?

0 = No sexual activity

5 = Almost always or always

4 = Most times (more than half the time)

3 = Sometimes (about half the time)

2 = A few times (less than half the time)

1 = Almost never or never

8. Over the past 4 weeks, how difficultwas it to become lubricated ("wet")during sexual activity or intercourse?

0 = No sexual activity

1 =Extremely difficult or impossible

2 = Very difficult

3 = Difficult

4 =Slightly difficult

5 = Not difficult

9. Over the past 4 weeks, how oftendid you maintain your lubrication("wetness") until completion ofsexual activity or intercourse?

0 = No sexual activity

5 = Almost always or always

4 = Most times (more than half the time)

3 = Sometimes (about half the time)

2 = A few times (less than half the time)

1 = Almost never or never

10. Over the past 4 weeks, how difficultwas it to maintain your lubrication("wetness") until completion of sexualactivity or intercourse?

0 = No sexual activity

1 =Extremely difficult or impossible

2 = Very difficult

3 = Difficult

4 =Slightly difficult

5 = Not difficult

11. Over the past 4 weeks, when you hadsexual stimulation or intercourse, howoften did you reach orgasm (climax)?

0 = No sexual activity

5 = Almost always or always

4 = Most times (more than half the time)

3 = Sometimes (about half the time)

2 = A few times (less than half the time)

1 = Almost never or never

12. Over the past 4 weeks, when you had sexualstimulation or intercourse, how difficult wasit for you to reach orgasm (climax)?

0 = No sexual activity

1 =Extremely difficult or impossible

2 = Very difficult

3 = Difficult

4 =Slightly difficult

5 = Not difficult

13. Over the past 4 weeks, how satisfiedwere you with your ability to reachorgasm (climax) during sexual activityor intercourse?

0 = No sexual activity

5 = Very satisfied

4 =Moderately satisfied

3 =About equally satisfied and dissatisfied

2 =Moderately dissatisfied

1 = Very dissatisfied

14. Over the past 4 weeks, how satisfiedhave you been with the amount ofemotional closeness during sexualactivity between you and your partner?

0 = No sexual activity

5 = Very satisfied

4 =Moderately satisfied

3 =About equally satisfied and dissatisfied

2 =Moderately dissatisfied

1 = Very dissatisfied

15. Over the past 4 weeks, how satisfiedhave you been with your sexual relationshipwith your partner?

5 = Very satisfied

4 =Moderately satisfied

3 =About equally satisfied and dissatisfied

2 =Moderately dissatisfied

1 = Very dissatisfied

16. Over the past 4 weeks, how satisfiedhave you been with your overallsexual life?

5 = Very satisfied

4 =Moderately satisfied

3 =About equally satisfied and dissatisfied

2 =Moderately dissatisfied

1 = Very dissatisfied

17. Over the past 4 weeks, how often didyou experience discomfort or painduring vaginal penetration?

0 =Did not attempt intercourse

1 = Almost always or always

2 = Most times (more than half the time)

3 = Sometimes (about half the time)

4 = A few times (less than half the time)

5 = Almost never or never

18. Over the past 4 weeks, how oftendid you experience discomfort or painfollowing vaginal penetration?

0 =Did not attempt intercourse

1 = Almost always or always

2 = Most times (more than half the time)

3 = Sometimes (about half the time)

4 = A few times (less than half the time)

5 = Almost never or never

19. Over the past 4 weeks, how wouldyou rate your level (degree) ofdiscomfort or pain during orfollowing vaginal penetration?

0 = Did not attempt intercourse

1 = Very high

2 = High

3 = Moderate

4 = Low

5 = Very low or none at all

FSFI Domain Scores and Full Scale Score

The individual domain scores and full scale (overall) score of the FSFI can be derived from the computational formula outlined in the table below. For the individual domain scores, add the scores of the individual items that comprise the domain and multiply the sum by the domain factor. Add the six domain scores to obtain the full scale score. It should be noted that within the individual domains, a domain score of zero indicates that the subject reported having no sexual activity during the past month.

Domain	Questions	Score Range	Factor	Minimum Score	Maximum Score	Score
Desire	1,2	1 – 5	0.6	1.2	6.0	
Arousal	3, 4, 5, 6	0 – 5	0.3	0	6.0	
Lubrication	7, 8, 9, 10	0 – 5	0.3	0	6.0	
Orgasm	11, 12, 13	0 – 5	0.4	0	6.0	
Satisfaction	14, 15, 16	0 (or 1) – 5	0.4	0	6.0	
Pain	17, 18, 19	0 – 5	0.4	0	6.0	
Full Scale Score Range				1.2	36.0	

A score ≤26 is classified as female sexual dysfunction***(Rosen et al., 2000).***

VI. Health Related Quality Of Life (HRQOL)

The Functional Outcomes of Sleep Questionnaire (FOSQ)***(Weaver et al., 1997)*** is the gold-standard, disease-specific instrument designed to assess the impact of sleepiness on the ability to conduct daily activities, conceptually defined as functional status, a component of quality of life. The 30-item FOSQ has proven validity and reliability, performing well as an outcome measure in clinical trials ***(Hirshkowitz and Black, 2007)***. However, it may be too long to easily employ in large-scale studies and monitoring effortsas well as clinical practice ***(Ware et al., 1996)***.

The FOSQ-10 (Modified FOSQ) is a psychometrically strong instrument that performs similarly to the long version. The rapidly completed and easily scored FOSQ-10 shows promise for application in the clinical setting ***(Chasens et al., 2009)***

Modified F.O.S.Q.

Q1. Do you have difficulty concentrating on the things you do because you are sleepy or tired?

1. Yes, extreme

2. Yes, moderate

3. Yes, a little

4. No

Q2. Do you generally have difficulty remembering things becauseyou are sleepy or tired?

1. Yes, extreme

2. Yes, moderate

3. Yes, a little

4. No

Q3. Do you have difficulty operating a motor vehicle for shortdistances (less than 100 miles) because you become sleepy?

1. Yes, extreme
2. Yes, moderate
3. Yes, alittle
4. No

Q4. Do you have difficulty operating a motor vehicle for longdistances (greater than 100 miles) because you become sleepy?

1. Yes, extreme
2. Yes, moderate
3. Yes, a little
4. No

Q5. Do you have difficulty visiting your family or friends in their home because you become sleepy or tired?

1. Yes, extreme
2. Yes, moderate
3. Yes, a little
4. No

Q6. Has your relationship with family, friends or work colleagues been affected because you are sleepy or tired?

1. Yes, extreme
2. Yes, moderate
3. Yes, a little
4. No

Q7. Do you have difficulty watching a movie or video because you become sleepy or tired?

1. Yes, extreme
2. Yes, moderate
3. Yes, a little
4. No

Q8. Do you have difficulty being as active as you want to be in the evening because you are sleepy or tired?

1. Yes, extreme
2. Yes, moderate
3. Yes, a little
4. No

Q9. Do you have difficulty being as active as you want to be in the morning because you are sleepy or tired?

1. Yes, extreme

2. Yes, moderate

3. Yes, a little

4. No

Q10. Has your mood been affected because you are sleepy or tired?

1. Yes, extreme

2. Yes, moderate

3. Yes, a little

4. No

SUMMARY AND CONCLUSION

The aims of this Essay are to study the symptoms and signs of psychiatric manifestations in OSAS for early diagnosis of these common confounding associations for better outcome.

Obstructive sleep apnea syndrome (OSAS) is a common chronic disorder that often requires lifelong care. It is estimated that 26 percent of adults are at high risk for OSAS. The prevalence of OSAS in the general population is approximately 20 percent if defined as an apnea hypopnea index (AHI) > five 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 OSAS. 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 psychiatric disorders such as depression, anxiety, epilepsy, sexual dysfunction and cognitive impairment such as (short memory impairment, attention, vigilance, and delirium). In addition, a high prevalence of other psychiatric symptoms such as somatization, attension 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 reportedin 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 OSAS. It involves monitoring the patient during a full night's sleep. Patients who are diagnosed with OSAS 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 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.

For patients with mild or moderate OSAS 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.

Depression in the general population is approximately 3%. Among OSAS patients it varies from 7-63%which may be caused by OSAS pathology or its symptoms leads to increase incidence of suicidal attempts between OSAS patients. Depression and OSAS are bidirectional interactive.

Depression is associated with low adherence to CPAP treatment, increased sensitivity to symptoms and daytime fatigue similar to that caused by OSAS. It should be noticed that some hypnotics used as adjunct therapy in patients with depression, which may make OSAS more severe and change sleep staging, are prescribed extensively. Though there has been little research on the impact of depression in OSAS, patients who have depression as well as OSAS also appear worse off than their counterparts with OSAS only. OSAS patients with high levels of depression are those with most daytime sleepiness, fatigue, and lowest quality of life scores.

There is a lot of scales for diagnosis of depression for example; The Beck Depression Inventory (BDI-I, BDI-II), created by ***Dr. Aaron T. Beck***, the three versions of the BDI—the original BDI, first published in 1961 and later revised in 1978 as the BDI-I, and the BDI-II, published in 1996.

Other scales for diagnosis of depression include: Beck Hopelessness Scale***(Beck*, *1988),***Edinburgh Postnatal Depression Scale (EPDS)***(Cox et al., 1987)***, Geriatric Depression Scale (GDS)***(Yesavage et al., 1982-1983)***, Hamilton Rating Scale for Depression (HAM-D)created by ***Dr. Max Hamilton*** who originally published the scale in 1960 and revised it in 1966, 1967,1969, and 1980. Kutcher Adolescent Depression Scale

(KADS)***(Kutcher, 2003)***, Major Depression Inventory (MDI) ***(Bech et al., 2001)***, Montgomery-ÅsbergDepression Rating Scale (MADRS) (***Montgomery and Asberg, 1979)***, and Zung Self-Rating Depression Scale ***(Zung, 1965)***.

Almost all suicides are associated with and preceded by major depression. This medical depression results from altered brain chemistry and function due to the cumulative stress of ongoing inadequate sleep (either insufficient quantity or poor quality or both). Persons who get adequate sleep feel better and handle the stresses of everyday life better. It seems logical that restoration of an adequate quantity or quality of sleep would improve or even resolve depression and that a reduction in suicides would follow.

On the other hand, the prevalence of anxiety in the general population is approximately 20%. Among OSAS patients it varies from 11-70%.Anxiety has been assessed by many scales such as Beck Anxiety Inventory***(Beck and Steer, 1993)***, Hamilton Anxiety Scale (HAM-A) ***(Maier et al., 1988),*** Hospital Anxiety and Depression Scale***(Zigmond and Snaith, 1983)***, Generalized Anxiety Disorder 7 (GAD-7)***(Spitzer etal., 2006)***, Panic and Agoraphobia Scale (PAS)***(Bandelow, 1995),*** Panic Disorder Severity Scale (PDSS)***(Shear et al., 1997),*** PTSD Symptom Scale – Self-Report Version***(Foa et al., 1997),*** Social Phobia Inventory (SPIN)***(Connor et al., 2000),*** Trauma Screening Questionnaire***(Brewin et al.,2002),*** Yale–Brown Obsessive Compulsive Scale (Y-BOCS)***(Goodman etal., 1989),*** and Zung Self-Rating Anxiety Scale ***(Zung, 1971)***.

Some patients prescribed positive airway pressure (PAP) therapy for OSAS experience claustrophobia, anxiety, or panic symptoms related to wearing the mask (feeling restricted) and/or tolerating the air pressure (feeling suffocated) 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.

Another important finding in OSAS is the health-related quality of life (HRQOL), The findings that the HRQOL may play an important role in anxious or depressive state is reasonable because this reduced HRQOL reflects deficits in functioning across a number of areas. Such difficulties may be so severe that job performances and family life may be affected, leading in turn to emotional disturbances and personality changes. These findings support the hypothesis that some of the psychopathological changes described in patients with OSAS are likely to reflect the reduced alertness and the reduced HRQOL related to breathing disorders.

Moreover, the reduced HRQOL present in OSAS patients may affect their general health perception and their functional and emotional well-being inducing, in turn, personality changes such as aggressiveness, irritability, anxiety or depression, all expressing adaptation of the patients to their worsening life condition.

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 OSAS patients. 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.

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

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

Excessive daytime sleepiness (EDS) is among the most common complaints of people with epilepsy, reported by 18–50% of adult patients. Excessive daytime sleepiness is often uniquely attributed to antiepileptic drugs (AEDs) and seizures. However, EDS is also one of the most common symptoms of obstructive sleep apnea syndrome (OSAS). By itself, OSAS is a highly prevalent disorder, affecting 24% of men and 9% of women. Small series suggest that nocturnal positive airway pressure (nPAP) therapy for OSAS reduces seizures in some cases. The Psychomotor Epilepsy Scale is used for diagnosis of epilepsy (***Ali, 1994)***.

About 48% of men with OSAS had sexual dysfunction. More recently, ED has been reported to occur in 30%-50% of men with OSAS. Prevalence of sexual dysfunction is high among women with OSAS than men with OSAS. OSAS patients were impaired in intimate and sexual relationships. Following treatment, patients were significantly more alert and had reported improved intimate and sexual relationships, with the greatest change occurring in those with the most disease severity which means that OSAS has an adverse impact on intimate and sexual relationships that is related to subjective sleepiness and improved with CPAP treatment. Sexual dysfunction has been assessed by many scales such as the International Index of Erectile Function (IIEF) Questionnaire, The Female Sexual Functioning Index (FSFI), Questionsto Guide Sexuality Assessment among Older Adult.

The other side of the coin was the psychiatric disorders associated with respiratory diseases such as bronchial asthma, COPD …

Psychiatric disorders associated with bronchial asthma such as depression is about 9% and ranges from 6% to 42% in COPD patients

and rises to 46% in TB patients. On the other hand the prevalence of anxiety disorders in the general population is approximately 20%. Among asthmatic patients it is about 30%, the prevalence of anxiety disorders also varies widely in COPD patients (from 21% to 96%) and rises to 47% in TB patients.

The association of psychiatric disorders with respiratory diseases leads to difficulty in treatment, repeated hospitalization, greater utilization of health care resources, and poorer health-related quality of life.

Conclusion and Recommendations

- Psychiatric manifestations are common in OSAS patients than in general population as it occur in 7%-63% for depression, 11%-70% for anxiety, 60% for impaired cognition, 24% for epilepsy in men and 9% for epilepsy in women,30%-50% for erectile dysfunction.

- Sleep specialists must be cognizant to the symptoms and signs of psychiatric manifestations for early diagnosis of these common confounding associations.

- Treatment of psychiatric manifestations associated with OSAS will lead to better control of OSAS and better quality of life.

- Psychiatrists and neurologists must be cognizant that OSAS may be a cause of epilepsy and depression that are resistant to treatment.

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الملخص العربى

الهدف من العمل :

الهدف من هذا العمل هو دراسة الإضطرابات النفسية المصاحبة لمتلازمة توقف التنفس الإنسدادى أثناء النوم و المقاييس المختلفة المستخدمة فى تشخيص هذه الإضطرابات النفسية, و من ثم, سوف يتم استخدام هذه المقاييس النفسية للمرضى المرتادين لعيادات اضطرابات التنفس أثناء النوم بالمنصورة .

الملخص العربي

تعد متلازمة توقف التنفس الانسدادى 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.ويعتبرمعدل انتشارالمتلازمة بين عموم السكان ما يقرب من عشرون في المائة في حالة تعريفه على أن مؤشر توقف التنفس أثناء النوم أكثر من خمس أحداث في الساعة،حيث يصيب أكثرمن اثنان بالمائة من الإناث البالغات، و أربعة بالمائة من الذكور .وعلاوة على ذلك، تزداد معدلات الانتشارمع تقدم العمر، و التى تتراوح بين ثلاثين إلى ثمانين بالمائة من السكان المسنين.

تشتمل السمات الأساسية لمتلازمة توقف التنفس الانسدادى أثناء النوم في البالغين على توقف و ضعف التنفس الانسدادي، الاستيقاظ المتكرر المصاحب للمجهود التنفسي وكذلك 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 توقف التنفس التي تنتهي بالشخير بصوت عال، وضعف التركيز، والذبحة الصدرية الليلية،والاستيقاظ مع الإحساس بالاختناق واللهاث والموت 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، الجسدنة، واضطراب نقص الانتباه المصاحب فرط النشاط، وأعراض الوسواس القهري في هؤلاء المرضى .كما أشارت أيضا إلي إمكانية حدوث نوبات الذعر الليلية، خطل نومي ، هذيان، والذهان، ونوبات عنف و تغير في شخصية بعض المرضى.

ويستخدم استبيان برلين للكشف عن المتلازمة . كما يستخدم مقياس ايبوورث 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.

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

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

إن الإضطرابات النفسية شائعة لدى المصابين بأمراض مزمنة مثل أمراض الجهاز التنفسى كما أن اكتشافها وعلاجها له أهمية كبيرة. إن من أشهر أمراض الجهاز التنفسى التى يصاحبها إضطرابات نفسية , متلازمة توقف التنفس الإنسدادى أثناء النوم والتى وجد أن الإكتئاب المصاحب لها يمثل حوالى من ٧ %إلى ٦٣% والتى تؤدى إلى إرتفاع نسبة محاولات الإنتحار بين مرضى متلازمة توقف التنفس الإنسدادى أثناء النوم. كما وجد أن نسبة القلق بين مرضى متلازمة توقف التنفس الإنسدادى أثناء النوم قد تصل إلى من

١١% إلى ٧٠ % والتى بدورها تقلل من جودة الحياة الصحية لمرض متلازمة توقف التنفس الإنسدادى أثناء النوم. يستخدم علاج التعرض للأفراد المصابين بتوقف التنفس أثناء النوم غير القادرين على تحمل أجهزة الضغط الهوائي الايجابي المستمربسبب 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 بالقيد) و / أو التغاضي عن ضغط الهواء (الشعور بالاختناق). علاج التعرض لحالات الخوف من الأماكن المغلقة المرتبطة باستخدام جهاز الضغط الهوائي الايجابي المستمرهو التدخل السلوكي على المدى القصير الذي عادة يمكن تنفيذه على نحو فعال في جلسةحتى ستة جلسات على مدى شهر إلي ثلاثة أشهر.

إن الإضطرابات النفسية المصاحبة لمتلازمة توقف التنفس الإنسدادى أثناء النوم لا تقتصر فقط على الإكتئاب والقلق ,ولكنها تشمل أيضا قلة القدرة على الإدراك , ضعف الذاكرة قصيرة المدى وطويلة المدى , وقد تؤدى أيضا إلى قلة الوعى بالبيئة المحيطة بمريض متلازمة التنفس الإنسدادى أثناء النوم. كما وجد أنهم يعانون من قلة القدرة الجنسية عند الإناث أكثر من الذكور.

ومن الأمراض الصدرية المزمنة التى يصاحبها إضطرابات نفسية مرض الربو الشعبى, فقد أوضحت الدراسات أن ٣٠% من هؤلاء المرضى يعانون من القلق و ٩% منهم معرضون للإكتئاب . ومن المثير أن إحدى الدراسات فى عام ٢٠٠٠ تشير إلى أن مرضى الربو الشعبى والإضطرابات النفسية معا معرضون إلى تفاقم أزمات الربو بحوالى الضعف أكثر من مرضى الربو الشعبى بدون إضطراب نفسى كما إن إحتمال دخولهم المستشفى بسبب أزمة الربو تزيد خمسة أضعاف. وتجدر الإشارة إلى أن مثل تلك الإضطرابات النفسية تؤدى إلى صعوبة التحكم بالربو وبالتالى إستخدام جرعات كبيرة من الدواء وإستنفاذ موارد الرعاية الصحية وقلة جودة الحياة الصحية .

وعندما يأتى الحديث لذكر مرض السدة الرئوية المزمنة نجد أن الإكتئاب يصيب من ٦% -٤٢%من هؤلاء المرضى كما يشيع القلق بينهم بنسبة تصل إلى ٢١% -٩٦ %. وحيث أن التدخين عامل هام فى إحداث مرض السدة الرئوية المزمنة فمن الضرورى الإشارة إلى أن الأبحاث المتعلقة أوضحت أن المدخنين أكثر عرضة للإصابة بالقلق وهو عامل مؤثر فى صعوبة الإقلاع عن التدخين. ومرة أخرى نجد أن الإضطرابات النفسية لها تأثير سلبى على حياة هؤلاء المرضي .