Workshop

CPAP and sleep disorders

Fulvio Braido
Allergy and Respiratory Diseases Department
University of Genoa
To explain better the pathophysiology of obstructive sleep apnea and its consequences

To emphasize the magnitude of the clinical problem

To make familiar the practical aspects of the treatment techniques

To identify unmet needs
Classification of sleep-related breathing disorders

Central sleep apnea syndromes
- Primary central sleep apnea
- Other central sleep apnea due to a medical condition
  - Cheyne-Stokes breathing pattern
  - High altitude periodic breathing
  - Central sleep apnea due to a medical condition, not Cheyne-Stokes or high attitude
- Central sleep apnea due to a drug or substance
- Other sleep-related breathing disorder due to a drug or substance
- Primary sleep apnea in infancy (formally primary sleep apnea of the newborn)

Obstructive sleep apnea syndromes
- Obstructive sleep apnea, adult
- Obstructive sleep apnea, pediatric

Adapted from the International Classification of Sleep Disorders, 2nd edition: Diagnostic and coding manual [2].
Bickelmann AG, Burwell CS, Robin ED, Whaley RD.

Extreme obesity associated with alveolar hypoventilation. A pickwickian syndrome.


OSA

It is characterized by repetitive collapse of the upper airways during sleep.
Hypopnea: reduction in the airflow $\geq 30\%$
SO2 decrease $\geq 3\%$
at least 10 second
Apnoea: reduction in the airflow $\geq 90\%$ for at least $90\%$ of the event at least 10 second
Central Apnea: These are central apneas (2) with minimal oxygen desaturation. Notice the low SAO2 at the beginning of this tracing. This is associated with a previous apnea.
Both of these events between 13-16 seconds in duration.

No inspiratory effort
Obstructive Sleep Apnea (OSA)

- Snoring

- Recurrent episodes of upper airway obstruction during sleep (apneas, hypopneas)

- Arousals

- Excessive and disabling daytime sleepiness

- **OSA Syndrome** – features of OSA on sleep study + symptoms of daytime sleepiness
OSA ➔ OSAS

- Excessive daytime sleepiness
- Obstructive breathing events (more than 5 per hour of sleep)

OR

Two or more of the following:
1. Choking or gasping during sleep
2. Recurrent awakenings from sleep
3. Unrefreshing sleep
4. Daytime fatigue
5. Impaired concentration

K. Bonno, M.H. Kryger / Sleep Medicine 8 (2007) 400–426
OSAS

It is characterised by repetitive collapse of the upper airways during sleep.
Pathophysiology of Obstructive Sleep Apnea (continued)

Physiologic

- Decreased function of upper airway dilator muscles (more than 20 skeletal muscles normally involved)
- Decreased pharyngeal dilator reflex response
- Decreased chemoreceptor drive/central drive (mixed with central sleep apnea)
Impact of sleep on ventilation

- Fall in phrenic and hypoglossal activity
- Reduce response to hypercapnia and hypoxia
- Reduction in upper airways protective reflexes

- Minute ventilation fall (16%)
- $\text{PaCO}_2$ increase 4-6 mmHg
- $\text{PaO}_2$ decrease (So2 decrease 2%)
- Irregular breathing during light and fragmented sleep
- Upper airways caliber reduction
SLEEP

Cortical inputs

Chemoreceptor sensitivity

Respiratory motor neurones

Respiratory muscle contraction

Lung mechanics: Airflow resistance FRC V/Q relationships

Hypoventilation

Hypoxemia

Hypercapnia
Pathophysiology of Obstructive Sleep Apnea

**Mechanical**

- Short, thick neck
- Neck flexion, supine position
- Nasal obstruction, congestion, polyps
Pathophysiology of Obstructive Sleep Apnea (continued)

Anatomic

- Enlarged tonsils and adenoids (esp. ages 3-5), enlarged uvula
- MacroGLOSSIA
- Retrognathia, craniofacial abnormalities
- Compliant (floppy) pharynx, especially soft palate
- Fat deposition in lateral walls of pharynx, pharyngeal dilator muscles (obesity)
- Submucosal edema in lateral walls of pharynx
Gravity

Jaw and tongue are forward while awake.

While asleep, muscles relax and gravity can drop the tongue back and block off the airway.
The wider the beginning of the airway, the less risk for collapse of the airway.

The narrower the beginning of the airway, the greater the risk for collapse.
**Venturi Principle**

Air must pass through a small tube faster than through a large tube, if the volume of air and time to pass through are equal.
OSA pathophysiology

Upper airway volume

Pharyngeal collapsibility

UA muscle activity

Loss of UA protective reflex?

Instability of the control of breathing?

Alteration of chemosensitivity?
Craniofacial size and Obesity can influence upper airway caliber.

Watanabe et al, AJRCCM 165:260, 2002
Axial Upper Airway MR Images

Subjects with OSA have smaller upper airway in wakefulness
OSA Patients Have Elevated Activity of Their Genioglossus Muscle During Wakefulness (Lost during sleep)

Mezzanotte et al, JCI 89:1571, 1992
Chronic load and altered pattern of usage induce myopathic changes and following impaired ability to maintain pharyngeal patency.
Eupneic Inspiration
(Revised from Fig. 2-1 in Levitzky’s *Pulmonary Physiology*)

Atmospheric Pressure: 0 cm H$_2$O

No flow

Alveolar pressure: 0 cm H$_2$O

Outward recoil of chest wall

Inward recoil of alveoli

Intrapleural pressure: -5 cm H$_2$O

Transmural pressure =
0 cm H$_2$O - (-5 cm H$_2$O) = +5 cm H$_2$O

END EXPIRATION

Atmospheric Pressure: 0 cm H$_2$O

Flow in

Inspiratory force

Alveolar pressure: -1 cm H$_2$O

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END EXPIRATION

Atmospheric Pressure: 0 cm H$_2$O

Flow in

Inspiratory force

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Intrapleural pressure: -8 cm H$_2$O

Transmural pressure =
-1 cm H$_2$O - (-8 cm H$_2$O) = +7 cm H$_2$O

DURING INSPIRATION
Forced Inspiration

Atmospheric Pressure: 0 cm H₂O

No flow

Outward recoil of chest wall

Inward recoil of alveoli

Intralveolar pressure: 0 cm H₂O

Transmural pressure = 0 cmH₂O - (-5 cmH₂O) = +5 cmH₂O

Atmospheric Pressure: 0 cm H₂O

Flow in

Inspiratory force

Alveolar pressure: -23 cm H₂O

Intralveolar pressure: -30 cmH₂O

Transmural pressure = -23 cmH₂O - (-30 cmH₂O) = +7 cmH₂O

End Expiration

During Inspiration
Evidence for risk factors

Risk factors: strong evidence

• obesity
• snoring
• male sex
• middle age and older
• craniofacial abnormalities

Risk factors: some evidence

• Menopause
• Family member with OSAS
• Smoking
• Nasal congestion at night
Demographic pattern of occurrence

**Snoring** and **sleepiness** are the strongest predictor of OSA

60% of men and 40% women between ages 41-65 years abitually **snore**. Thus is diagnostic utility is limited.
Obesity is a very strong risk factor for OSA.

All measures of obesity - neck and waist girths, weight, skin folds - predict OSA.

An increase of 1 Kg/m² in BMI yields an estimated 30% increase in the odds of developing OSA.

An increase (decrease) of 1 kg/m² in BMI yields an estimated 9% increase (decrease) in AHI.

Male: female ratio for OSA prevalence is 2:1.
Obstructive Sleep Apnea

Upper airway anatomy

Hard Palate

Hyoid bone

Larynx

Soft Palate

Nasopharynx

Oropharynx

Epiglottis

Sites of obstruction during sleep apnea

Laryngopharynx
Fat Is Deposited in Tongue in Obese Subjects

A Obese Mouse sleeping upright to protect his upper airway

The standing sleeping mouse
<table>
<thead>
<tr>
<th>STRUCTURES</th>
<th>NZO (Fat)</th>
<th>NZW</th>
<th>P Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Soft palate, mm$^3$</td>
<td>5.86 ± 1.28</td>
<td>4.64 ± 0.88</td>
<td>0.021</td>
</tr>
<tr>
<td>Tongue, mm$^3$</td>
<td>137 ± 26.0</td>
<td>104 ± 18.0</td>
<td>0.003</td>
</tr>
<tr>
<td>Lateral pharyngeal walls, mm$^3$</td>
<td>111 ± 26.9</td>
<td>84.4 ± 16.2</td>
<td>0.014</td>
</tr>
<tr>
<td>Mandible width, mm</td>
<td>7.85 ± 0.37</td>
<td>7.9 ± 0.34</td>
<td>NS (P = 0.76)</td>
</tr>
<tr>
<td>Mandible A–P distance, mm</td>
<td>5.46 ± 0.34</td>
<td>5.2 ± 0.22</td>
<td>NS (P = 0.501)</td>
</tr>
</tbody>
</table>

Brennick et al, AJRCCM 179:158, 2009
OSA prevalence by age: 
Sleep Heart Health Study

Young T et al, Arch Intern Med 2002
By age 50 ys, incidence rates among men and women are similar.

Cleveland Family Study: interaction of age with gender and BMI

The effect of BMI decreases with age and may be irrelevant in elderly.

Tishler et al, JAMA 2003
Frequency ans severity of OSA in pre post menopausal women

![Bar chart showing frequency of OSA severity in pre and post-menopausal women.](chart.png)

*Chest 2001;120;151-155*
DOI 10.1378/chest.120.1.151
Clinical Features

- Loud snoring
- Excessive daytime somnolence
- Intellectual deterioration
- Personality changing
- Erectile disfunction
- Nocturnal enuresis
- Morning headaches

Guilleminault, et al 1978
Sleep Apnea Syndrome
<table>
<thead>
<tr>
<th>Sleepiness</th>
<th>Sleep-related obstructive breathing events (/h)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mild: Unwanted sleepiness or involuntary sleep episodes during activity requiring <em>little</em> attention (e.g., watching TV, reading)</td>
<td>5–15</td>
</tr>
<tr>
<td>Moderate: Unwanted sleepiness or involuntary sleep episodes during activity requiring <em>some</em> attention (e.g., meetings, concerts)</td>
<td>15–30</td>
</tr>
<tr>
<td>Severe: Unwanted sleepiness or involuntary sleep episodes during activity requiring <em>active</em> attention (e.g., eating during conversation, operating a motor vehicle)</td>
<td>&gt;30</td>
</tr>
</tbody>
</table>

*K. Banno, M.H. Kryger / Sleep Medicine 8 (2007) 400–426*
Explanation for Hypersomnolence or Excessive Daytime Sleepiness

- Repeated arousals (may be hundreds per night) interfere with sleep architecture, especially rapid eye movement sleep

- Abnormal sleep architecture leads to daytime somnolence, decreased attentiveness, blunted mentation, depression, personality changes
Sleep disruption

Disruption of restorative features of sleep

Disruption of cellular or chemical homeostasis

Prefrontal cortical dysfunction

Dysfunction of cognitive executive system

Behavioral inhibition
Set shifting
Self-regulation of affect and arousal
Working memory
Analysis/synthesis
Contextual memory

Adverse day time effects
Problems in mentally manipulating information
Poor planning and haphazard execution of plans
Disorganization
Poor judgement/decision-making
Rigid thinking
Difficulty in maintaining attention and motivation
Emotional lability ('mood swings')
Overactivity/impulsivity (especially in children)
Description of Sleep Apnea Event

- Upper airway Intermittent obstruction
- Decreased alveolar ventilation
- Decreased alveolar $\text{PO}_2$; increased alveolar $\text{PCO}_2$
- Decreased arterial $\text{PO}_2$; increased arterial $\text{PCO}_2$
- Stimulation of arterial and central chemoreceptors;
- Arousal - Secondary hyperventilation
Effects of Breathhold on Arterial $\text{PO}_2$ and $\text{PCO}_2$

- **O$_2$** decreases over time.
- **CO$_2$** increases over time.

All figures created by Betsy Giaimo.
Effects of Hematocrit on Human Blood Viscosity
Possible Explanation for Nocturia

- Increased blood viscosity and arterial hypertension
- Increased right ventricular afterload
- Increased right ventricular end diastolic pressure and volume
- Increased right atrial volume
- Increased secretion of atrial natriuretic peptide from atrial myocytes,
- Increases sodium excretion, and stretches receptors that suppress ADH secretion from the posterior pituitary gland
Effects of Arterial Po$_2$ and Pco$_2$ on Cerebral Blood Flow
Explanation for Morning Headaches

• Hypoxia and hypercapnia during obstruction cause dilatation of cerebral blood vessels
### CONDITIONS ASSOCIATED TO OSA

<table>
<thead>
<tr>
<th>Field</th>
<th>Associated disorders and conditions, presentation</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cardiology</td>
<td>Hypertension, left ventricular hypertrophy, angina pectoris, myocardial infarction, arrhythmia, heart failure, pulmonary hypertension, cor pulmonale, peripheral edema, sudden death</td>
</tr>
<tr>
<td>Respirology</td>
<td>Respiratory failure, nocturnal shortness of breath, postpolio syndrome</td>
</tr>
<tr>
<td>Endocrinology</td>
<td>Diabetes mellitus, insulin resistance, metabolic syndrome, hypothyroidism, acromegaly</td>
</tr>
<tr>
<td>Neurology</td>
<td>Stroke, epilepsy, impaired memory, cognitive dysfunction</td>
</tr>
<tr>
<td>Gastroenterology</td>
<td>Gastroesophageal reflux disease (GERD)</td>
</tr>
<tr>
<td>Hematology</td>
<td>Polythemia</td>
</tr>
<tr>
<td>Psychiatry</td>
<td>Depression, anxiety disorder, Schizophrenia</td>
</tr>
<tr>
<td>Urology</td>
<td>Nocturia, impotence, erectile dysfunction, reduced libido</td>
</tr>
<tr>
<td>Gynecology and obstetrics</td>
<td>Pregnancy, menopause, polycystic ovary syndrome (PCOS)</td>
</tr>
<tr>
<td>Otorhinolaryngology</td>
<td>Enlarged tonsils, adenoids, nasal obstruction</td>
</tr>
<tr>
<td>Ophthalmology</td>
<td>Glaucoma, non-arteritic ischemic optic neuropathy</td>
</tr>
<tr>
<td>Anesthesiology</td>
<td>Difficulty of intubation, prolonged apneic episodes after operation</td>
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<tr>
<td>Dentistry and Orthodontics</td>
<td>Retrognathia, micrognathia</td>
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Signs of Obstructive Sleep Apnea

- Systemic hypertension
- Pulmonary hypertension (right axis deviation on ECG)
- Polycythemia
- Cor pulmonale
- Bradycardia during apneic event
- Tachycardia after airflow restored
- Typically no respiratory abnormality while awake
Risk Factors

OSAS

Cardiovascular Pathologies

Sex
Age
Obesity
Smoking
Alcohol
Mechanisms for Cardiovascular Consequences of OSA

Arnardottir E et al, Sleep 32:447, 2009
Possible Explanation for Systemic Hypertension

- Repeated increases in sympathetic tone and systemic blood pressure during arousals may cause vascular remodeling and changes in endothelial function
Arterial hypertension is especially frequent in patients suffering from OSAS.

25 - 38%

Kryger M et al. WB Saunders 1989
OSAS = Independent risk for the Arterial Hypertension?

Carlson J et Al Am J Respir Crit Care Med 1994;150:72-77
Fischer J et Al Pneumologie 1993; 47 (Supl.1):151-154

Risk 2.1
Prospective Study of the Association between Sleep-Disordered Breathing and Hypertension

Paul E. Peppard, Ph.D., Terry Young, Ph.D., Mari Palta, Ph.D., and James Skatrud, M.D.

Volume 342:1378-1384 May 11, 2000 Number 19
**Table 3. Adjusted Odds Ratios for Hypertension at a Follow-up Sleep Study, According to the Apnea-Hypopnea Index at Base Line.**

<table>
<thead>
<tr>
<th></th>
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<tbody>
<tr>
<td>0 events/hr†</td>
<td>1.0</td>
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</tr>
<tr>
<td>0.1–4.9 events/hr</td>
<td>1.66 (1.35–2.03)</td>
<td>1.65 (1.33–2.04)</td>
<td>1.42 (1.14–1.78)</td>
<td>1.42 (1.13–1.78)</td>
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<tr>
<td>5.0–14.9 events/hr</td>
<td>2.74 (1.82–4.12)</td>
<td>2.71 (1.78–4.14)</td>
<td>2.03 (1.29–3.19)</td>
<td>2.03 (1.29–3.17)</td>
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<tr>
<td>≥15.0 events/hr</td>
<td>4.54 (2.46–8.36)</td>
<td>4.47 (2.37–8.43)</td>
<td>2.89 (1.47–5.69)</td>
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<tr>
<td>p for trend‡</td>
<td>&lt;0.001</td>
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<td>0.002</td>
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*Hypertension was defined as a blood pressure of at least 140/90 mm Hg or the use of antihypertensive medications. Data on 893 follow-up sleep studies from 709 participants were analyzed. The odds ratios and confidence intervals were adjusted for the fact that 184 participants completed two follow-up sleep studies. BMI denotes body-mass index.

†This category served as the reference group.

‡P values are for the linear trend of the logistic-regression coefficients (loge of the odds ratios).
Identifiable Causes of Hypertension

- Sleep apnea
- Drug-induced or drug-related
- Chronic kidney disease
- Primary aldosteronism
- Renovascular disease
- Chronic steroid therapy and Cushing syndrome
- Pheochromocytoma
- Coarctation of the aorta
- Thyroid or parathyroid disease

Obstructive Sleep Apnea
- Hypoxemia
- Reoxygenation
- Hypercapnia
- Intrathoracic Pressure Changes
- Arousals

Intermediary Mechanisms
- Sympathetic Activation
  - Vasoconstriction
  - Increased Catecholamines
  - Tachycardia
  - Impaired Cardiovascular Variability
- Endothelial Dysfunction
- Vascular Oxidative Stress
- Inflammation
- Increased Coagulation
- Metabolic Dysregulation
  - Leptin Resistance
  - Obesity
  - Insulin Resistance

Risk of Cardiovascular Disease
- Hypertension
- Congestive Heart Failure
  - Systolic Dysfunction
  - Diastolic Dysfunction
- Cardiac Arrhythmia
  - Bradycardia
  - A-V Block
  - Atrial Fibrillation
- Cardiac Ischemia
  - Coronary Artery Disease
  - Myocardial Infarction
  - Nocturnal ST-Segment Depression
  - Nocturnal Angina
- Cerebrovascular Disease

*JAMA. October 8, 2003;290:1906-1914.*
OSAS & Ischemic Cardiopathy

ECG ST elevation concomitant to apnoea induced Oxygen desaturation

Sleep Apnea and Cardiovascular Disease
Richard S. T. et Al
OSAS & Ischemic cardiopathy

Snorers/Normal -- > Probability risk 2.3


Apneics/Normal

Heart failure risk in OSAS  23.3 times compared to normal subjects

Hung J et Al. Lancet 1990;336:261-264
OSAS & Ischemic Cardiopathy

OSAS minimum prevalence among Coronaropathic subjects is about 16%

Andreas S et Al Coron Artery Dis 1996;7:541-545
During the follow-up period (post-myocardial infarction), cardiovascular death occurred in six of 16 OSA patients (37.5%) compared with 4 (9.3%) in the non-OSA group (p = 0.018)

OSA is represents a risk factor for death in post-myocardial infarction
The post-MI changes of cardiac function may predispose to the development of OSA, or may affect OSA severity.
Effects of Obstruction on Pulmonary Circulation and Right Ventricle

- Hypoxic and hypercapnic pulmonary vasoconstriction cause pulmonary hypertension
- Chronic nighttime hypoxia may cause erythropoiesis and polycythemia
- Increased hematocrit increases blood viscosity
- Hypoxic pulmonary vasoconstriction (HPV), increased blood viscosity, pulmonary hypertension increase right ventricular afterload
- Increased right ventricular afterload may lead to right ventricular hypertrophy and eventually cor pulmonale
Possible Explanations for Bradycardia During Obstruction, Tachycardia after Airflow Restored

• Stimulation of arterial chemoreceptors usually increases heart rate because it increases tidal volume (lung inflation reflex)

• Stimulation of arterial chemoreceptors without stretching the lungs causes bradycardia

• After arousal leads to restoration of airflow, large tidal volumes stretch lungs and cause tachycardia

• May hyperventilate immediately after arousal, then hypoventilate until CO₂ is restored
Artrial Fibrillation
Ventricular Extrasystole
Cardiaco arrest
Sinus bradycardia
Atrioventricular block

Benefit of Atrial Pacing in Sleep Apnea Syndrome
Stephane Garrigue, M.D. et Al
NEJM Volume 346:404-412 February 7, 2002 Number 6
Cardiac arrhythmias, snoring, and sleep apnea  
V Hoffstein and S Mateika  
Department of Medicine, St. Michael's Hospital, Toronto, Canada.

458 patients

82% of patients with mean nocturnal oxygen saturation < 90% had arrhythmias vs 40% of patients with mean nocturnal oxygen saturation > 90%

70% of patients with AHI ≥ 40 had arrhythmias vs 42% with AHI ≤ 40
Sleep-disordered breathing and cardiovascular disease: cross-sectional results of the Sleep Heart Health Study

*Am J Respir Crit Care Med*. 2001;163:19-25

“modest to moderate effects of sleep-disordered breathing on CVD manifestations within a range of the AHI that is typically considered "normal" or only mildly elevated (1-10 respiratory events per hour of sleep)"
Sleep-disordered breathing and cardiovascular disease: cross-sectional results of the Sleep Heart Health Study

Am J Respir Crit Care Med. 2001;163:19-25

modestly elevated risk coupled with a high prevalence of mild sleep-disordered breathing might have considerable public health implications
OSAS & Cerebral vascular pathology

Snorers/controls stroke risk = 1.7-3.4

Partinen et Al Lancet 1985;8468:1325-1326
Spriggs et Al Q J Med 1992;83: 555-562
Smirne S Eur et Al Respir J 1993; 6:1357-1361

However, it remains unclear whether sleep apnea is an independent risk factor for cerebrovascular disease.
Mortality and apnea index in obstructive sleep apnea.
Experience in 385 male patients.

385 male OSA patients

Probability of cumulative eight-year survival was 96 +/- 0.02 (SE) for HAI = less than 20 vs
63 +/- 0.17 for AHI greater than 20
EFFECTS of OSAS TREATMENT ON OSAS-RELATED CARDIAC CIRCULATORY DISEASES
Refractory hypertension and sleep apnoea: effect of CPAP on blood pressure and baroreflex.

Logan AG Et Al

11.0 +/- 4.4 mmHg reduction in 24-h systolic BP
7.8 +/- 3.0 mmHg reduction in nighttime diastolic BP


“several months of CPAP therapy resulted in a small but significant reduction of daytime blood pressure of between 1.3 and 5.3 mm Hg”
Nocturnal ischemic events in patients with obstructive sleep apnea syndrome and ischemic heart disease: effects of continuous positive air pressure treatment.

Peled N, Abinader EG, Pillar G, Sharif D, Lavie
Am Coll Cardiol. 1999 Nov 15;34(6):1744-9

Treatment with continuous positive airway pressure significantly ameliorated the nocturnal ST depression time from 78 min to 33 min (p<0.001)
The Cardiomyopathy of Obstructive Sleep Apnea
Robert Joseph Thomas, MD
Annals of Internal Medicine
1 September 1996 | Volume 125 Issue 5 | Page 425

<table>
<thead>
<tr>
<th>Patient</th>
<th>Age</th>
<th>Clinical Syndrome</th>
<th>Hypertension</th>
<th>Diabetes Mellitus</th>
<th>Ischemic Heart Disease</th>
<th>Ejection Fraction before CPAP</th>
<th>Ejection Fraction after CPAP</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>75</td>
<td>Recurrent hypercapnic respiratory failure and congestive cardiac failure</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>25</td>
<td>55</td>
</tr>
<tr>
<td>2</td>
<td>70</td>
<td>Recurrent pulmonary edema</td>
<td>+</td>
<td>−</td>
<td>−</td>
<td>35</td>
<td>60</td>
</tr>
<tr>
<td>3</td>
<td>55</td>
<td>Recurrent ventricular tachycardia and congestive cardiac failure</td>
<td>+</td>
<td>−</td>
<td>−</td>
<td>45</td>
<td>60</td>
</tr>
<tr>
<td>4</td>
<td>45</td>
<td>Idiopathic dilated cardiomyopathy</td>
<td>+</td>
<td>−</td>
<td>−</td>
<td>30</td>
<td>65</td>
</tr>
</tbody>
</table>

* CPAP = nasal continuous positive airway pressure.

Cardiovascular Effects of Continuous Positive Airway Pressure in Patients with Heart Failure and Obstructive Sleep Apnea
Kaneko et al. 348 (13): 1233 NEJM, March 27, 2003
<table>
<thead>
<tr>
<th>Disease</th>
<th>Prevalence</th>
</tr>
</thead>
<tbody>
<tr>
<td>Asthma</td>
<td>300 million</td>
</tr>
<tr>
<td>COPD</td>
<td>210 million</td>
</tr>
<tr>
<td>Allergic rhinitis</td>
<td>400 million</td>
</tr>
<tr>
<td>Sleep apnea</td>
<td>&gt;100 million</td>
</tr>
</tbody>
</table>
How common is OSA?

• US study in 1993: 24% of men and 9% of women had AHI>5 (age 30-60)

• 4% of men and 2% of women reported daytime sleepiness also (OSAS)

• UK study in 1991: 1% of men had OSAS

• Prevalence likely to increase as obesity level rises
Ferini-Strambbi L et al, 2004

- Minimally symptomatic or asymptomatic OSA is estimated to occur in 1 of 5 adults.

- OSA with daytime impairment (OSA syndrome) occur in 1 of 20 adults and is rarely recognized.
Symptoms attributed to sleep apnea ranked in order of frequency selected

<table>
<thead>
<tr>
<th>Symptom</th>
<th>Frequency Selected</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. Decreased energy</td>
<td>57%</td>
</tr>
<tr>
<td>2. Waking up in the morning feeling unrefreshed and tired</td>
<td>56%</td>
</tr>
<tr>
<td>3. Excessive fatigue</td>
<td>55%</td>
</tr>
<tr>
<td>4. Difficulty with a dry or sore mouth or throat upon awakening</td>
<td>29%</td>
</tr>
<tr>
<td>5. Waking up often (more than twice) during the night</td>
<td>29%</td>
</tr>
<tr>
<td>6. Feeling that ordinary activities require an extra effort to perform or complete</td>
<td>27%</td>
</tr>
<tr>
<td>7. Falling asleep if not stimulated or active</td>
<td>25%</td>
</tr>
<tr>
<td>8. Falling asleep at inappropriate times or places</td>
<td>22%</td>
</tr>
<tr>
<td>9. Waking up more than once per night (on average) to urinate</td>
<td>22%</td>
</tr>
<tr>
<td>10. Fighting the urge to fall asleep while driving</td>
<td>22%</td>
</tr>
</tbody>
</table>
Questionnaires

Epworth Sleepiness Scale

Table 6.1 Epworth Sleepiness Scale (a subjective measure of daytime sleepiness)

<table>
<thead>
<tr>
<th>Situation</th>
<th>Chance of Dozing</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sitting and reading</td>
<td></td>
</tr>
<tr>
<td>Watching TV</td>
<td></td>
</tr>
<tr>
<td>Sitting, inactive in a public place (e.g. theater or a meeting)</td>
<td></td>
</tr>
<tr>
<td>As a passenger in a car for an hour without a break</td>
<td></td>
</tr>
<tr>
<td>Lying down to rest in the afternoon when circumstances permit</td>
<td></td>
</tr>
<tr>
<td>Sitting and talking to someone</td>
<td></td>
</tr>
<tr>
<td>Sitting quietly after a lunch without alcohol</td>
<td></td>
</tr>
<tr>
<td>In a car, while stopped for a few minutes in the traffic</td>
<td></td>
</tr>
</tbody>
</table>

Limited utility of ESS and other sleepiness scales to predict the presence of OSAS
Questionnaires

**Berlin Questionnaire**


- Questions about snoring
- Questions about sleepiness
- Questions about hypertension
- Questions about age, weight, height, gender, neck circumference, ethnicity

Provides dichotomous outcome – high or low risk for OSA

High and low risk categories decided by consensus
Clinical Impressions

Upper airways abnormalities

- High-arched palate
- Large tongue
- Tonsillar hypertrophy
- Redundant soft palatal tissue
- Retrognathia
- Micrognathia
- Allergic Rhinitis Features

Morphometric measurements

- Measuring neck size
- Performing skin fold thickness measurements
Assessment of the Upper Airways

Direct visualisation
Endoscopy
Rhinometry
Rhinomanometry
Imaging
Sleep endoscopy

M. Barbieri, I. Zannis, M. Filidoro, A. Barbieri.
Clinical Impressions

History + Physical examination: sensitivity 50%

Hoffstein & Szalai Sleep 1993

Bed partner report of apnea an snoring:
  Sensitivity 78%
  Specificity 64%
  Positive predictive value 64%

Kapuniai et al Sleep 1988
Integration of Multiple Factors

- Witnessed apnea
- Snoring
- Nocturnal choking
- Excessive daytime sleepiness
- Motor vehicle accidents
- Male sex
- Obesity
- Hypertension

80%
<table>
<thead>
<tr>
<th>Level</th>
<th>Parameters</th>
<th>Body Position</th>
<th>Personnel</th>
<th>Interventions</th>
</tr>
</thead>
<tbody>
<tr>
<td>Level I</td>
<td>≥ 7, EEG, EOG, EMG chin, ECG, flow, respiratory effort, SaO2</td>
<td>Documented or objectively measured</td>
<td>In constant attendance</td>
<td>Possible</td>
</tr>
<tr>
<td>Level II</td>
<td>≥ 7, EEG, EOG, EMG chin, ECG, flow, respiratory effort, SaO2</td>
<td>May be objectively measured</td>
<td>Not in attendance</td>
<td>Not possible</td>
</tr>
<tr>
<td>Level III</td>
<td>≥ 4, including ventilation (at least two channels of respiratory activity)</td>
<td>May be objectively measured</td>
<td>Not in attendance</td>
<td>Not possible</td>
</tr>
<tr>
<td>Level IV</td>
<td>Minimum of one</td>
<td>Not measured</td>
<td>Not in attendance</td>
<td>Not possible</td>
</tr>
</tbody>
</table>
Ossimetria Holter
Flow evaluation
Flow evaluation
Flow evaluation

```
<table>
<thead>
<tr>
<th>Date</th>
<th>Start</th>
<th>End</th>
<th>Duration</th>
</tr>
</thead>
<tbody>
<tr>
<td>11/2/2004</td>
<td>10:08:39 PM</td>
<td>7:52:36 AM</td>
<td>9h 14min</td>
</tr>
</tbody>
</table>

**Evaluation period**

**Risk indicator**

```
<table>
<thead>
<tr>
<th>Normal range</th>
<th>Suspected pathological breathing disorder</th>
</tr>
</thead>
<tbody>
<tr>
<td>---------------</td>
<td>--------------------------------------------</td>
</tr>
</tbody>
</table>

**Analysis**

<table>
<thead>
<tr>
<th>Indices</th>
<th>Normal range</th>
<th>Results (during evaluation period)</th>
</tr>
</thead>
<tbody>
<tr>
<td>AHI **</td>
<td>&lt; 5 /h</td>
<td>3.006</td>
</tr>
<tr>
<td>FI **</td>
<td>&lt; 5 /h</td>
<td>152</td>
</tr>
<tr>
<td>Apnea index</td>
<td>&lt; 18 /h</td>
<td>Hypopnea:</td>
</tr>
<tr>
<td>Hypopnea index</td>
<td>&lt; 17 /h</td>
<td>142</td>
</tr>
<tr>
<td>Flow limited breaths without Sn (FS)</td>
<td>&lt; 22 %</td>
<td>Flow limited breaths without Sn (FL)</td>
</tr>
<tr>
<td>Flow limited breaths with Sn (FS)</td>
<td>&lt; 5 %</td>
<td>Flow limited breaths with Sn (FL)</td>
</tr>
<tr>
<td>Average breath frequency [bpm]</td>
<td>15-17 bpm</td>
<td>Breaths:</td>
</tr>
</tbody>
</table>

* AHI = Average number of apneas/hypopneas per hour during the evaluation period
* FI = Risk Indicator = score as a sum of AHI * score of FL/FS (details - see user's manual)

**Parameters used [MAP standard parameters]**

```

**Diagnosis**

Lo screening fisicostametrico evidenzia un quadro di sospetta OSAS che andrà indagato con monitoraggio
```
<table>
<thead>
<tr>
<th>Level I</th>
<th>Level II</th>
<th>Level III</th>
<th>Level IV</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Parameters</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>( \geq 7, \text{EEG, EOG, EMG chin, ECG, flow, respiratory effort, SaO}2 )</td>
<td>( \geq 7, \text{EEG, EOG, EMG chin, ECG, flow, respiratory effort, SaO}2 )</td>
<td>( \geq 4, \text{including ventilation (at least two channels of respiratory activity)} )</td>
<td>Minimum of one</td>
</tr>
<tr>
<td><strong>Body Position</strong></td>
<td>Documented or objectively measured</td>
<td>May be objectively measured</td>
<td>May be objectively measured</td>
</tr>
<tr>
<td><strong>Personnel</strong></td>
<td>In constant attendance</td>
<td>Not in attendance</td>
<td>Not in attendance</td>
</tr>
<tr>
<td><strong>Interventions</strong></td>
<td>Possible</td>
<td>Not possible</td>
<td>Not possible</td>
</tr>
</tbody>
</table>
### Apnea/Hypopnea Statistics

<table>
<thead>
<tr>
<th>Respiration</th>
<th>Number</th>
<th>%</th>
<th>A or H/h</th>
<th>Supine</th>
<th>Non-Supine</th>
<th>Mean [seconds]</th>
<th>Longest [seconds]</th>
</tr>
</thead>
<tbody>
<tr>
<td>Apnea</td>
<td>718</td>
<td>95</td>
<td>88,1</td>
<td>574</td>
<td>144</td>
<td>26,7</td>
<td>107,4</td>
</tr>
<tr>
<td>Obstructive</td>
<td>549</td>
<td>73</td>
<td>67,3</td>
<td>420</td>
<td>129</td>
<td>25,7</td>
<td>107,4</td>
</tr>
<tr>
<td>Central</td>
<td>8</td>
<td>1</td>
<td>1,0</td>
<td>2</td>
<td>6</td>
<td>13,7</td>
<td>18,5</td>
</tr>
<tr>
<td>Mixed</td>
<td>161</td>
<td>21</td>
<td>19,7</td>
<td>152</td>
<td>9</td>
<td>30,8</td>
<td>65,6</td>
</tr>
<tr>
<td>Hypopnea</td>
<td>38</td>
<td>5</td>
<td>4,7</td>
<td>11</td>
<td>27</td>
<td>17,4</td>
<td>48,0</td>
</tr>
<tr>
<td><strong>Total</strong></td>
<td>756</td>
<td>92,7</td>
<td>585</td>
<td>171</td>
<td>26,3</td>
<td>107,4</td>
<td></td>
</tr>
</tbody>
</table>

### Apnea-Desaturation Relation

<table>
<thead>
<tr>
<th>Desaturation</th>
<th>Apnea</th>
<th>Obstructive</th>
<th>Central</th>
<th>Mixed</th>
<th>Hypopnea</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td>&gt;90%</td>
<td>26</td>
<td>20</td>
<td>0</td>
<td>6</td>
<td>4</td>
<td>30</td>
</tr>
<tr>
<td>81-90%</td>
<td>402</td>
<td>360</td>
<td>5</td>
<td>37</td>
<td>18</td>
<td>420</td>
</tr>
<tr>
<td>71-80%</td>
<td>66</td>
<td>54</td>
<td>0</td>
<td>12</td>
<td>0</td>
<td>66</td>
</tr>
<tr>
<td>61-70%</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>51-60%</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>&lt;50%</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td><strong>Total</strong></td>
<td>494</td>
<td>434</td>
<td>5</td>
<td>55</td>
<td>22</td>
<td>516</td>
</tr>
</tbody>
</table>
Flow sensor (or PneumoFlow®)
ECG electrodes
Activity sensor
Oxygen saturation sensor
Body position sensor with integrated thoracic sensor
Microphone
Abdominal sensor
Pressure sensor
Activity sensor
VALIDATION STUDIES

- Esnaola S et al, Eur Respir J 1996 (MesamIV)
- Zucconi M et al, Eur Respir J 1996 (MicroDigitrapper)
- Ficker JH et al, Respiration 2001 (Somnocheck)

⇒ The diagnostic accuracy of manual analysis was found to be superior to that of automatic analysis
<table>
<thead>
<tr>
<th>Parameters</th>
<th>Level I</th>
<th>Level II</th>
<th>Level III</th>
<th>Level IV</th>
</tr>
</thead>
<tbody>
<tr>
<td>&gt; 7, EEG, EOG, EMG chin, ECG, flow, respiratory effort, SaO2</td>
<td>≥ 7, EEG, EOG, EMG chin, ECG, flow, respiratory effort, SaO2</td>
<td>≥ 4, including ventilation (at least two channels of respiratory activity)</td>
<td>Minimum of one</td>
<td></td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Body Position</th>
<th>Level I</th>
<th>Level II</th>
<th>Level III</th>
<th>Level IV</th>
</tr>
</thead>
<tbody>
<tr>
<td>Documented or objectively measured</td>
<td>May be objectively measured</td>
<td>May be objectively measured</td>
<td>Not measured</td>
<td></td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Personnel</th>
<th>Level I</th>
<th>Level II</th>
<th>Level III</th>
<th>Level IV</th>
</tr>
</thead>
<tbody>
<tr>
<td>In constant attendance</td>
<td>Not in attendance</td>
<td>Not in attendance</td>
<td>Not in attendance</td>
<td></td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Interventions</th>
<th>Level I</th>
<th>Level II</th>
<th>Level III</th>
<th>Level IV</th>
</tr>
</thead>
<tbody>
<tr>
<td>Possible</td>
<td>Not possible</td>
<td>Not possible</td>
<td>Not possible</td>
<td>Not possible</td>
</tr>
</tbody>
</table>
Management Options in OSAS

Subjective Indicators:
- Sleepiness.
- Quality of life
- Mood
- Symptoms
- Work

Objective Indicators:
- Comorbidity: (Cardiovascular and Respiratory diseases, Hypertension)
- Neuropsychiatric and behavioral complications
- AHI, ODI, RDI
CPAP for OSAS

- AHI ≥20 with or without symptoms
- AHI 5-19 with sleepiness, behavioral complications

Modified ACCP Statement
TREATMENT

- AHI > 25 and severe clinics: nCPAP + sleep hygiene
- AHI < 25 and mild clinics: Sleep hygiene and diet
- AHI < 25 and severe clinics: Sleep hygiene, diet and temporary nCPAP
- AHI > 25, mild clinics: Sleep hygiene and diet
- Alterations of soft tissues or of the skeleton: Surgery, especially in young and/or non-obese patients
Lack of efficacy for a cervicomandibular support collar in the management of obstructive sleep apnea

Skinner et al, *CHEST 2004*
AIM: To bring the mandible ahead
Management Options in OSAS

Nasal Continuous Positive Airway Pressure Therapy

CPAP provides a splint

Pharyngeal unfolding
Reduced compliance
Pharyngeal decompression
Upper airway dilator muscle activity

Splinting of upper airway

suction pressure

Hypoxemia

Diaphragm efficiency

Upper airway collapse

Excessive daytime sleepiness

Nasal CPAP

end expiratory lung volume

Upper airway mucosal Edema

Splinting of upper airway

Pharyngeal cross-sectional area

snoring
Placebo CPAP

Loredo et al., Chest 1999

awakening
Epworth Scale

**Jenkinson et al., Lancet 1999**

*Placebo vs. CPAP*

- Before
- After

**Statistical significance:**

- Placebo: 15
- CPAP: 5

Significant difference (p<0.01)
## Comparison of SF-36 scores before and after CPAP therapy in Patients (n = 45), and Irish Normative Data

<table>
<thead>
<tr>
<th>Variables</th>
<th>Before CPAP</th>
<th>After CPAP</th>
<th>p Value</th>
<th>Normative Values,</th>
</tr>
</thead>
<tbody>
<tr>
<td>Physical functioning</td>
<td>75</td>
<td>80</td>
<td>0.006</td>
<td>95</td>
</tr>
<tr>
<td>Role limitation (physical)</td>
<td>50</td>
<td>100</td>
<td>0.0004</td>
<td>100</td>
</tr>
<tr>
<td>Role limitation (emotional)</td>
<td>66.7</td>
<td>100</td>
<td>0.01</td>
<td>100</td>
</tr>
<tr>
<td>Social functioning</td>
<td>66.7</td>
<td>100</td>
<td>0.0004</td>
<td>100</td>
</tr>
<tr>
<td>Mental health</td>
<td>76</td>
<td>80</td>
<td>0.02</td>
<td>80</td>
</tr>
<tr>
<td>Energy/vitality</td>
<td>35</td>
<td>60</td>
<td>0.001</td>
<td>67</td>
</tr>
<tr>
<td>Pain</td>
<td>77.8</td>
<td>88</td>
<td>0.33</td>
<td>84</td>
</tr>
<tr>
<td>General health perception</td>
<td>67</td>
<td>67</td>
<td>0.05</td>
<td>77</td>
</tr>
</tbody>
</table>
** **

Jenkinson et al. Lancet 1999
Impact of Nasal Continuous Positive Airway Pressure Therapy on the Quality of Life of Bed Partners of Patients With Obstructive Sleep Apnea Syndrome

Doherty LS, Kiely JL, Lawless G, McNicholas WT.

Chest, 2003
## Comparison between Partners ESS scores before and after CPAP therapy (n=45)

<table>
<thead>
<tr>
<th>Variables</th>
<th>Before CPAP</th>
<th>After CPAP</th>
<th>p Value</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Patient</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>ESS</td>
<td>16 (11–20)</td>
<td>8 (4–15)</td>
<td>0.001</td>
</tr>
<tr>
<td>Anxiety</td>
<td>8 (5–9)</td>
<td>6 (3–8)</td>
<td>0.001</td>
</tr>
<tr>
<td>Depression</td>
<td>5 (3–8)</td>
<td>4 (2–6)</td>
<td>0.02</td>
</tr>
<tr>
<td><strong>Partner</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>ESS</td>
<td>4 (1–8.5)</td>
<td>2 (1–5)</td>
<td>0.007</td>
</tr>
<tr>
<td>Anxiety</td>
<td>7 (5–11)</td>
<td>7 (4–8)</td>
<td>0.02</td>
</tr>
<tr>
<td>Depression</td>
<td>4 (2–6)</td>
<td>4 (1–5)</td>
<td>0.1</td>
</tr>
</tbody>
</table>
Daytime Sleepiness

Nasal Continuous Positive Airway Pressure Therapy

Titration

Choice of Device: CPAP, Auto-CPAP

Mask Selection

Home monitoring
Figura 15. Dispositivi per l’erogazione di CPAP
AutoPAP

- Rationale – critical nCPAP level may vary depending on body position, sleep stage, alcohol consumption etc.
- Auto-titrating devices adjust pressure in response to
  - snoring
  - flow-limitation
  - apneas
  - hypopneas
  - leaks
**AutoPAP**

American Academy of Sleep Medicine Practice Parameters 2002

1) The diagnosis of OSA must be established by an acceptable method.

2) Auto-titrating PAP may be used during attended titration to identify a single effective pressure for use with standard CPAP.

3) Auto-titrating PAP may be used in self-adjusting mode for unattended treatment of OSA after an initial successful attended CPAP or auto-titrating PAP titration.
AutoPAP

- Auto-titrating PAP devices are NOT recommended for:
  - split-night studies
  - patients with CHF, significant lung disease (COPD), daytime hypoxemia, respiratory failure, or prominent nocturnal oxygen desaturation other than from OSA

- Auto-titrating PAP devices that rely on vibration or sound in the device’s algorithm should NOT be used in patients who snore.
Auto-Titrating Versus Standard Continuous Positive Airway Pressure for the Treatment of Obstructive Sleep Apnea: Results of a Meta-analysis

Najib T. Ayas, MD, MPH, Sanjay R. Patel, MD, Atul Malhotra, MD; Michael Schulzer, MD, PhD; Mark Malhotra; David Jung; John Fleetham, MD; David P. White, MD

Conclusions: Compared to standard CPAP, APAP is associated with a reduction in mean pressure. However, APAP and standard CPAP were similar in adherence and their ability to eliminate respiratory events and to improve subjective sleepiness. Given that APAP is more costly than standard CPAP, APAP should not be considered first-line chronic therapy in all patients with OSA. However, APAP may be useful in other situations (eg, home titrations, detection of mouth leak) or in certain subgroups of patients with OSA. Identifying circumstances in which APAP is a definite improvement over CPAP in terms of costs or effects should be the focus of future studies.

OSAS Treatment

• Nasal CPAP is the treatment of choice
• Results in successful treatment in 95%
• Not as costly as surgery
• Long term compliance rates of 60-70%
• Improved long term survival (vs. UPPP)
• Can re-titrate the pressure if the patient’s clinical condition changes
POTENTIAL BARRIERS TO CPAP ADHERENCE

- Mechanical
- Psychological
- Educational
- Physical
CPAP ADHERENCE:

MECHANICAL BARRIERS:

• Positive Airway Pressure System
• Interface System
• Humidification

• Nasal congestion
• Rhinorrhea
• Epistaxis
• Sinus discomfort
• Oronasal dryness
• Skin rash
• Abrasion
• Conjunctivitis
• Chest discomfort
• Difficulty exhaling
• Aerophagia
• Pneumothorax (rare)
• Pneumoencephaly (rare)
## Helpful hints for CPAP users (a)

<table>
<thead>
<tr>
<th>SITUATION:</th>
<th>TRY THIS:</th>
</tr>
</thead>
</table>
| Cold Nose  | - Increase temperature of room air  
            | - Place a blanket over your tubing to warm the air before it reaches you |
| Red, dry or sore eyes | - Readjust mask and/or headgear |
| Face redness around mask area | - Use a skin barrier product  
                              | - Try nasal “pillows”  
                              | - Avoid over-tightening of headgear  
                              | - Try mask “spacer” if available |
### Helpful hints for CPAP users (b)

<table>
<thead>
<tr>
<th>SITUATION</th>
<th>TRY THIS:</th>
</tr>
</thead>
<tbody>
<tr>
<td>Throat/Nose dryness</td>
<td>• Increase room humidity</td>
</tr>
<tr>
<td></td>
<td>• Consider a heated humidifier with your unit</td>
</tr>
<tr>
<td></td>
<td>• Utilize a saline nasal spray</td>
</tr>
<tr>
<td>Sneezing; nasal drainage</td>
<td>• Use a saline nasal spray</td>
</tr>
<tr>
<td></td>
<td>• Consider a heated humidifier or increase humidity settings</td>
</tr>
<tr>
<td></td>
<td>• Remove allergens from your room</td>
</tr>
<tr>
<td></td>
<td>• Use a HEPA air filter unit in your room</td>
</tr>
<tr>
<td></td>
<td>• Change pollen filters often</td>
</tr>
<tr>
<td></td>
<td>• Clean your mask</td>
</tr>
</tbody>
</table>
CPAP ADHERENCE:

PSYCHOLOGICAL BARRIERS

• Claustrophobic
• Embarrassment
• Vanity
• Personality Type Disorders
• Support System
CPAP ADHERENCE:

EDUCATIONAL BARRIERS

• Comprehension
• Patient’s knowledge of obstructive sleep apnea
• Patient’s knowledge of interface system
• Patient’s knowledge of delivery system
CPAP COMPLIANCE:

PHYSICAL BARRIERS

- Physical Handicaps
- Extreme Obesity
- Nasal Dryness
- Stomach Distention
- Retrognathic
- Under Treated
- Over Treated
- Sinus Problems
- Puffing
- Ear Discomfort
- Eye Irritation
- Material Sensitivity
To explain better the pathophysiology of obstructive sleep apnea and its consequences

To emphasize the magnitude of the clinical problem

To make familiar the practical aspects of the treatment techniques

To identify unmet needs
Workshop

CPAP and sleep disorders

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