Obstructive Sleep Apnea for the Primary Care Physician

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Agenda / Topics

• What is Obstructive Sleep Apnea?
• Evaluation of OSA
• Diagnosing OSA
• Management of OSA
• Follow up of OSA
• Q/A Session
What is Obstructive Sleep Apnea?
History of Sleep Apnea

• In 400 BCE, Hippocrates in one of his medical works, *On the Sacred Disease*, recounted one of the first descriptions of sleep apnea:

  “I have known many persons in sleep groaning and crying out, some in a state of suffocation, some jumping up and fleeing out of doors, and deprived of their reason until they awaken, and afterward becoming well and rational as before, although they be pale and weak; and this will happen not once but frequently.”
In 360 BCE, Dionysius of Heraclea was known in history as an unusually obese man. Historical documents describe his physicians may have used one of the first treatments of apnea: sticking needles through his skin to arouse him from sleep.

“Hence, because of his obesity, he was afflicted with shortness of breath and fits of choking. So the physicians prescribed that he should get some fine needles, exceedingly long, which they thrust through his ribs and belly whenever he happened to fall into a very deep sleep.”
In Charles Dickens’ 1836 novel, one of the characters featured, Joe, is an obese boy who is constantly hungry and always falling asleep in the middle of tasks—symptoms suggestive of obesity hypoventilation syndrome, likely in the context of obstructive sleep apnea:

"'Damn that boy,' said the old gentleman, 'he's gone to sleep again.'
'Very extraordinary boy, that,' said Mr. Pickwick; 'does he always sleep in this way?'
'Sleep!' said the old gentleman, 'he's always asleep. Goes on errands fast asleep, and snores as he waits at table.'"
Awareness in the 19th Century

In 1887, W.H. Broadbent, a doctor from St. Mary’s Hospital in London published an article in *The Lancelet* on Cheyne-Stokes breathing where he described a case of sleep disordered breathing that was “similar to Cheyne-Stokes breathing”:

“When a person, especially advanced in years, is lying on his back in heavy sleep and snoring loudly, it is very commonly happens that every now and then the inspiration fails to overcome the resistance in the pharynx of which stertor or snoring is the audible sign, and there will be perfect silence through two, three, or four respiratory periods, in which there are ineffectual chest movements; finally air enters with a loud snort, after which there are several compensatory deep inspirations before the breathing settles down to usual rhythm[...]the snoring ceased at regular intervals, and the pause was so long as to excite attention, and indeed alarm.”
In 1889, Richard Caton presented to the clinical society of London a case that he termed, “A case of Narcolepsy.”

However the description of the unusual features of sleep in Caton’s patient leave little doubt as to the actual nature of his disorder:

“When in sound asleep a very peculiar state of the glottis is observed, a spasmodic closure entirely suspending respiration. The thorax and abdomen are seen to heave from fruitless contractions of inspiratory and expiratory muscles; their efforts increase in violence for about a minute or a minute and a half, the skin meantime becoming more and more cyanosed, until at last, when the condition to the onlooker is most alarming, the glottic obstruction yields, a series of long inspirations and expirations follows, and the cyanosis disappears.”
Progress in the 20th Century

• In 1924, Berger, a psychiatrist, recorded the first human electroencephalograms and published a paper, “On the EEG in Humans” in 1929.

• In the 1930s, Alfred Loomis (attorney, investment banker, scientist), studied sleep in humans with EEG technology and showed that EEG patterns in humans changed dramatically during a night’s sleep.

• In 1960, Werner Gerady and his colleagues conducted studies on ‘Pickwickian’ patients and were the first to report on apneic events during sleep in medical literature.

• In 1961, Drachman and Gumnit of the NIH reported on a patient they observed who’s breathing ceased and arterial oxygen saturation level dropped as soon as her EEG showed signs of sleep— the patient would “awaken” half a minute later and start breathing and raise her oxygen saturation level, only to stop breathing again after a few seconds and repeat the cycle.
Advent of Sleep Apnea in the US

• In 1970, the first sleep clinic was opened at Stanford.
• However the first patients seen were primarily insomniacs and narcoleptics, with little interest in patients with sleep-disordered breathing.
• In 1973 they demonstrated that insomnia associated with upper airway obstruction during sleep can occur in people with normal weight in a paper titled, “Insomnia with sleep apnea: a new syndrome.”
• In the paper they used the term “Sleep Apnea Syndrome” for the first time and defined it as when at least 30 apneas of a minimum duration of 10 seconds each were detected during sleep.
Obstructive Sleep Apnea (OSA)

• A disorder of obstructed breathing that occurs during sleep.
• Is the most common sleep-related breathing disorder.
• More specifically, abnormal breathing during sleep that causes recurrent arousals, sleep fragmentation, and nocturnal hypoxemia.
• This leads to daytime symptoms attributable to disrupted sleep, such as excessive sleepiness, fatigue, or poor concentration.
Societal Impact of OSA

• Of approximately 75,000 patients seen annually in sleep disorder centers, roughly 75% are diagnosed with OSA.

• Large medical cost burden of untreated moderate/severe OSA:
  ▪ 1990 - $275 million dollars
  ▪ 1999 - $3.4 billion dollars
Societal Impact of OSA (cont)

• OSA has been associated with higher rates of unintentional injury, including motor vehicle collisions and work-related injuries, which add to the public health burden.

• Traffic accidents:
  ▪ Sleep apnea sufferers are 7 times more likely to be in an automobile accident.
  ▪ People with sleep disordered breathing are 100x more likely to drive off the road.
  ▪ Reaction times of someone with OSA is equivalent to a normal control who is legally intoxicated (ABL >0.8).
Prevalence

• Approximately 20 to 30 percent of males and 10 to 15 percent of females in North America have OSA.

• Men are three times more likely than women to have OSA.
  § OSA is uncommon in non-obese, premenopausal women.
  § However, rates of OSA in postmenopausal women not on hormone therapy approach the rates of OSA in men of a similar age and body mass index.

• Prevalence of OSA increases with age, especially in persons older than 60 years.

• Higher prevalence in African Americans who are younger than 35 years old in comparison with Caucasians of the same age group.
Prevalence (cont)

- NCSDR-1994
  40 million Americans with chronic sleep disorder
  20 million Americans with occasional sleep disorder

- Wisconsin Cohort Study
  *Sleep Disordered Breathing (REI >5):* 24% middle aged males, 9% middle aged females
  *Obstructive Sleep Apnea (AHI >15/hour):* 4% middle aged males, 2% middle aged females
Risk Factors

• Obesity
  ▪ The strongest risk factor in both males and females.
  ▪ In a prospective study of nearly 700 adults with four-year longitudinal follow-up, a 10% increase in weight was associated with a six-fold increase in risk of incident OSA.
  ▪ In a population-based study of over 1000 adults in Sao Paulo who underwent polysomnography, moderate to severe OSA (AHI ≥15 events/hour) was present in:
    11% of men and 3% of women who were normal weight
    21% of men and 9% of women who were overweight (BMI 25 to 30 kg/m²)
    63% of men and 22% of women who were obese (BMI >30 kg/m²)
Risk Factors (cont)

• Disorders that predispose to narrowing of the upper airway or reduction in its stability increase the likelihood of having or developing OSA.

• Higher recognized risk factor than obesity in Asian patients.
  ▪ Abnormal maxillary or short mandibular size
  ▪ Wide craniofacial base
  ▪ Tonsillar hypertrophy
  ▪ Adenoid hypertrophy

• Smoking appears to increase the risk of OSA or perhaps aggravate preexisting symptoms.
  ▪ In one study, current smokers were nearly three times more likely to have OSA than former or never smokers.
Family History

• Patients with OSA may often report a family history of snoring or OSA.

• It has been suggested that about one-fourth of the prevalence of OSA as a disease has a genetic basis.
  - May be due to shared behavioral factors related to obesity.
  - Also a possibility of a genetic predisposition to OSA through factors such as craniofacial structure.
Pathophysiology of OSA
OSA-mediated Health Related Morbidity

**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

## Associated Conditions

<table>
<thead>
<tr>
<th>Condition</th>
<th>Odds ratio*</th>
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</thead>
<tbody>
<tr>
<td>Atrial fibrillation</td>
<td>4.0</td>
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<tr>
<td>Depression</td>
<td>2.6</td>
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<tr>
<td>Congestive heart failure</td>
<td>2.4</td>
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<tr>
<td>Stroke</td>
<td>1.6 to 4.3</td>
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<tr>
<td>Hypertension</td>
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<tr>
<td>Coronary artery disease</td>
<td>1.3</td>
</tr>
<tr>
<td>Diabetes mellitus</td>
<td>1.2 to 2.6</td>
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Evaluation
Symptoms prompting consideration for OSA

• Witnessed episodes of apnea
• **Loud, frequent, or bothersome snoring**
• Choking/gasping during sleep
• **Excessive daytime sleepiness**
• Drowsy driving
• Unrefreshing sleep, sleep fragmentation
• Insomnia
• Nocturia
• Nighttime awakenings
• **Morning headaches**
• Decreased concentration, memory loss
• Decreased libido
Sleep History

• Specific symptoms or complaints pertaining to sleep?
• Length and severity of symptoms?
• Reported history from family members, spouses, significant others, friends?
• Feelings of tiredness, sleepiness, or refreshed upon waking up in the morning?
• Presence of daytime naps and length?
• Sleep schedule on weekdays and weekends?
• Sleep habits, subjective time of sleep per night?
• Bed partners, sleeping environment?
• Previous evaluation or treatment for sleep disorders; oral appliances?
Sleep History (cont)

• Past Medical History
  ▪ Type 2 diabetes mellitus
  ▪ Polycystic ovary syndrome
  ▪ GERD
  ▪ Elevated blood pressure/HTN
  ▪ Asthma, COPD, pulmonary hypertension

• Social History
  ▪ Occupation, work hours, shift work
  ▪ EtOH use, substance use
  ▪ Tobacco history
  ▪ Caffeine use
Sleep History (cont)

- Medications
  - Opioids
  - Benzodiazepines
  - Antihistamines
  - Barbiturates
Screening Questionnaires

• The high false positive rate of questionnaires diminish their diagnostic value.
  ▪ When the score is high, patients do not always have OSA.
  ▪ In contrast, when the score is low, patients are unlikely to have OSA.

• Another major limitation to their use is that most scores have been derived from sleep center or preoperative referral-based populations.
  ▪ May reflect a higher overall prevalence of OSA.
  ▪ Likelihood of the sensitivity and specificity of such instruments to be overestimated.
STOP-Bang Screening Test

• An eight-item survey that incorporates information on snoring, observed apneas, blood pressure, BMI, age, neck circumference, and gender.

• A score of three or higher has a sensitivity and specificity of 84 and 56 percent for the diagnosis of OSA when defined as an AHI ≥5 events per hour.

• Sensitivity and specificity of 93 and 43 percent for an AHI >15 events/hour.
STOP-Bang

THE STOP-Bang Questionnaire
A Tool to Screen Patients for Obstructive Sleep Apnea (OSA)

Name: ___________________________ Age: ___ DOB: ________

1. S (Snoring): Do you Snore loudly? Yes ____, No ___
2. T (Tired): Do you often feel Tired or sleepy? Yes ____, No ___
3. O (Observed): Has anyone Observed you stop breathing during your sleep? Yes ____, No ___
4. P (Blood Pressure): Do you have or are you being treated for high Blood Pressure? Yes ____, No ___
5. B (BMI): Body Mass Index > 35 kg/m²? Yes ____, No ___
6. A (Age): Age over 50 years old? Yes ____, No ___
8. G (Gender): Gender male? Yes ____, No ___

High Risk for OSA: Answering “Yes” to 3 or more items.
Evaluation by a sleep specialist may be warranted.
Berlin Questionnaire

• Consists of 10 items relating to snoring, non-restorative sleep, sleepiness while driving, apneas during sleep, hypertension, and BMI.

• A high risk score is associated with a sensitivity and specificity of 80 and 46 percent when OSA is defined as an AHI ≥5 events per hour.

• Sensitivity and specificity of 91 and 37 percent when OSA is defined as an AHI ≥15 events per hour.
Epworth Sleepiness Scale

• A self-administrated questionnaire that asks subjects to rate how likely they would fall asleep in 8 specific situations or activities that are commonly met in daily life.

• The chance of falling asleep is rated on a scale of 0–3 (0 = would never dose, 1 = slight chance of dozing, 2 = moderate chance of dozing, and 3 = high chance of dozing)

• The total ESS score is the sum of 8-items scores and can range between 0 and 24.

• The higher the score, the higher the person’s level of daytime sleepiness.
  ▪ Categorized into <11 (low risk for sleepiness) and ≥11 (high risk for sleepiness).

• Highest specificity to predict OSA, moderate-to-severe OSA and severe OSA (75%, 48.15% and 46.43%, respectively) but with the lowest sensitivity (72.55%, 75.71% and 79.73%, respectively)
Physical Examination

• Height and Weight
  ▪ Over 90% of individuals with a BMI of 40 or greater have OSA requiring treatment.
  ▪ Up to 30% of individuals with OSA have a BMI less than 30.

• Neck Size
  ▪ Stronger correlation between OSA and increased neck size or waist circumference than general obesity.
  ▪ OSA is particularly prominent among men who have a collar size greater than 17 inches and women who have a collar size greater than 16 inches.

• Peripheral Edema, JVD
  ▪ Pulmonary hypertension and cor pulmonale are common sequelae when OSA coexists with either obesity hypoventilation syndrome or an alternative cause of daytime hypoxemia (ie, chronic lung disease)
ENT Examination

• Head/Face Exam
  ▪ Retrognathia

• Nose
  ▪ External nose deformities
  ▪ Deviated or perforated nasal septum
  ▪ Nasal turbinate size

• Oral Cavity
  ▪ Dentition, signs of bruxism
  ▪ Overbite, overjet
  ▪ Tongue enlargement, scalloping
ENT Examination (cont)

• Oropharynx
  ▪ Soft palate appearance
  ▪ High-arched hard palate
  ▪ Uvula position and size
  ▪ Tonsillar size
  ▪ Mallampati score
Tonsil Size Scoring

0: Surgically removed tonsils
1: Tonsils hidden within tonsil pillars
2: Tonsils extending to the pillars
3: Tonsils are beyond the pillars
4: Tonsils extend to midline
Modified Mallampati Score

• The modified Mallampati classification is commonly used to quantify airway narrowing.
• Classes 3 and 4 considered positive for airway narrowing
Diagnosis
Diagnostic Testing

- Objective testing is necessary to diagnosis OSA because clinical features are non-specific and the diagnostic accuracy of clinical impression alone is poor.
- In-laboratory polysomnography is the first-line diagnostic study when OSA is suspected.
- Home sleep apnea testing may be an acceptable alternative for patients who are strongly suspected of having OSA and who do not have significant medical comorbidities.
The Polysomnogram
Terms

• **Apnea**: Cessation of breathing with respiratory effort lasting >10 seconds

• **Hypopnea**:
  - Decreased airflow of >70%
  - Any decreased airflow with desaturation <90%

• **Apnea-Hypopnea Index (AHI)**: Number of apnea and hypopnea events per hour of sleep.

• **EEG Arousal**: An abrupt shift in EEG frequency, which may include theta, alpha, and/or frequencies greater than 16 Hz but not spindles.

• **Respiratory-effort related arousals (RERAs)**: Arousals from sleep that do not technically meet the definitions of apneas or hypopneas, but do disrupt sleep.
Normal vs. OSA

Normal Polysomnograph

Obstructive Sleep Apnea

EEG
EMG
ECG
BP
Abd
Chest
Vt (air flow)

100
Pulse Oxygen Saturation

75

20 sec
Time (minutes)

EEG
ECG
BP
Abd
Chest
Vt (air flow)

100
Pulse Oxygen Saturation

75

20 sec
Time (minutes)
Home Sleep Study

• Diagnostic test used to diagnose obstructive sleep apnea outside of the clinical setting.

• Advantages include convenience, greater patient acceptance, and its potential lower costs relative to an in-lab polysomnogram.

• Disadvantage is that for most of these devices, fewer physiologic variables are measured than with a NPSG, which can lead to misinterpretation or underestimation of results.
Diagnostic Criteria

• The criteria for a diagnosis of OSA are traditionally classified on the basis of symptoms and the apnea-hypopnea index (AHI).

• AHI is the number of apneas or hypopneas recorded during the study per hour of sleep. It is generally expressed as the number of events per hour.

• Based on the AHI, the severity of OSA can be classified as:
  ▪ None/Minimal: AHI < 5 per hour
  ▪ Mild: AHI ≥ 5, but < 15 per hour
  ▪ Moderate: AHI ≥ 15, but < 30 per hour
  ▪ Severe: AHI ≥ 30 per hour
Management of OSA
Goals of Management

• OSA is a chronic disease that requires long-term, multidisciplinary management.
• The goals of therapy is to reduce or eliminate apneas, hypopneas, and oxyhemoglobin desaturation during sleep and thereby improve sleep quality and daytime function.
Behavioral Modifications

• Weight loss and exercise should be recommended to all patients with OSA who are overweight or obese.

• Effects of weight loss on OSA was demonstrated in a trial consisting of 72 consecutive overweight patients with a mean BMI of 32 kg/m² and mild OSA (mean AHI of 10 events/hour sleep)
  ▪ Patients were randomly assigned to receive a single session of general nutrition or an intensive program consisting of a low-calorie diet for 3 months plus nutrition and exercise counseling for one year.
  ▪ Latter group had significantly greater weight loss, reduction in AHI, and improvement in QOL compared with the control group.

• The effect of weight loss achieved via bariatric surgery on OSA appears to be similar, with reductions in AHI proportional to weight loss.
Behavioral Modifications (cont)

• Avoidance of alcohol or other sedating agents.
  ▪ Can depress the central nervous system and exacerbate OSA, worsen sleepiness, and promote weight gain.

• Treatment of disorders that reduce nasal airflow, such as chronic rhinitis, nasal polyps, or septal deviation.
Positional Therapy

- Patients with more milder OSA who show a documented higher AHI in specific positions (such as supine) may consider altering sleep position.

- Sleeping in a non-supine position may correct or improve OSA in such patients and should be encouraged but is not generally relied upon as the sole therapy.

- Methods for adherence include:
  - Tennis ball strapped to the back while sleeping to discourage supine position.
  - Positional pillows to prevent rolling into specific positions.
  - Wearable positional avoidance devices.
  - Raising head of bed to avoid supine position.
Positive Airway Pressure Therapy

• “Gold standard” in the treatment of adults with OSA.
• Most consistently successful and extensively studied treatment for obstructive sleep apnea
• The positive pressure ventilation functions as a pneumatic splint that keeps the pharyngeal airway open.
Positive Airway Pressure Therapy (cont)

• In a meta-analysis of 22 randomized trials (1160 patients):
  ▪ Nocturnal CPAP was compared with a control (sham CPAP, placebo tablets, or conservative management).
  ▪ Subjective and objective sleepiness, quality of life, cognitive function, and depression were all improved with nocturnal CPAP therapy.

• In a meta-analysis of 35 randomized trials, CPAP was compared with sham showed:
  ▪ Significant reduction in the apnea-hypopnea index (AHI; mean difference -33.8 events/hour).
  ▪ Improved daytime sleepiness as assessed by the Epworth Sleepiness Scale (mean difference -2 points).
  ▪ Improved systolic and diastolic blood pressure, and sleep-related quality of life.
CPAP Compliance

• In a prospective study of 233 OSA (AHI >10) patients and 36 non-apneic snorers:
  ▪ 214 OSA patients accepted CPAP
  ▪ 181 continued to be on treatment (77%)
  ▪ Mean of 5.6 hours/night
  ▪ 77-89% compliance rate

• CMS has defined “adequate” CPAP adherence as use of ≥ 4 hours per night on 70% of nights.

• CMS along with an increasing number of third-party payers requires documentation of this level of adherence during a consecutive 30-day period within the first 90 days of therapy to continue reimbursement.
CPAP Machine
Mask Types

Nasal

Nasal Pillow

Full Face
Mask Types (cont)

• No single mask type is superior to another.
• Select the mask that will best maximize patient comfort.
• Oronasal (“full face”) masks:
  ▪ Patients who sleep with their mouth open.
• Nasal masks:
  ▪ Better tolerated with claustrophobia.
• Nasal pillows (sit under the nose and fit in the nares)
  ▪ Also better tolerated with claustrophobia.
  ▪ For patients with unusual nasal bridge anatomy, facial hair, or absent dentition.
Mandibular Advancement Devices

• Reasonable alternative for patients with mild or moderate OSA who decline or fail to adhere to positive airway pressure therapy.

• Variable efficacy in patients with severe OSA and/or significant sleep-related hypoxemia; such patients are not good candidates for an oral appliance as first-line therapy.
Mandibular Advancement Devices (cont)

• Less effective than CPAP for normalizing the AHI:
  ▪ 67 studies comprising 6873 patients in a meta-analysis.
  ▪ Compared with an inactive control, continuous positive airway pressure was associated with a reduction in ESS score of 2-5 points (95% CI 2.0-2.9).
  ▪ Mandibular advancement devices demonstrated a reduction of 1-7 points (95% CI 1.1-2.3).
Mandibular Advancement Devices (cont)

- Referral to a certified AADSM dentist recommended.
- Insurance companies generally do not pay for oral appliances.
- Follow-up sleep study needed to document adequacy.
Mechanism of MAD

Without MAD

With MAD
Upper Airway Surgery

• Usually reserved as a second-line therapy for obstructive sleep apnea (OSA).

• Either as salvage therapy in patients with OSA who cannot adhere to CPAP, or as adjunctive therapy along with CPAP or an oral appliance.
  ▪ Surgery is rarely indicated as primary therapy in adults.

• Drug-induced sleep endoscopy can be used to study the dynamic airway in a patient with OSA, allowing the ENT surgeon to tailor the operative procedure to the patient.
Uvulopalatopharyngoplasty (UPPP)

- Currently the most common surgery performed for adults with obstructive sleep apnea in the United States.
- A surgical or laser procedure that removes part of the uvula and soft palate in an attempt to alleviate snoring and sleep-disordered breathing.
- Small reduction in symptoms.
- Fewer than half of patients have reduction in severity of OSA.
Tonsillectomy

• Not as effective in adults as in children for treatment of OSA.
• Limited studies show substantial tonsillar hypertrophy can rarely cause OSA in adults.
• However selected patients with enlarged tonsils and otherwise favorable anatomy (eg, small tongue) may experience cure with simple tonsillectomy.
• Even if not cured, selected patients who undergo tonsillectomy often experience significant reduction in the high CPAP pressure that was required to splint the airway adequately around noncompliant tonsillar tissue.
Maxillomandibular Advancement

• Patients with maxillomandibular anatomy that predisposes them to airway obstruction may be considered for jaw surgery.

• Invasive procedure with prolonged postop recovery.

• Cure rate >90%, particularly in non-obese patients with retrognathia.
Tracheostomy

• Last-resort measure employed in life-threatening situations, such as acute respiratory failure or for patients who are severely apneic and noncompliant.

• Bypasses the entire upper airway and can eliminate or dramatically reduce OSA.

• A smaller cannula tube is used which is more comfortable and easier to manage.

• Patients can typically eat and speak normally with the cannula capped during waking hours and can breathe easily with the cannula open during sleeping hours.

• Requires vigilant maintenance to minimize complications.

• Generally is not well accepted by patients as a long-term treatment.
Other Interventions

• Provent Therapy
  ▪ Proprietary valve design that attaches over the nostrils and is secured in place with hypoallergenic adhesive.
  ▪ During inhalation, the valve opens, allowing the user to breathe in freely.
  ▪ When exhaling, the valve closes and air passing through the nose is directed through two small air channels.
  ▪ This increases the pressure in the airway and helps to keep it open.
  ▪ 81% of obstructive sleep apnea sufferers were reported successfully treated.
Other Interventions (cont)

• Inspire Therapy
  ▪ Upper airway stimulation via an implantable neurostimulator device.
  ▪ Activates the protrusion muscles of the tongue via the hypoglossal nerve to open the lower pharyngeal airway.
  ▪ Also improves the upper pharyngeal airway by physiological and anatomical coupling of the tongue to the palate.
Patient Follow-up
Adherence

• While CPAP is the most effective therapy for OSA, adherence is suboptimal.

• It is estimated that 29 to 83 percent of patients are non-adherent, when non-adherence is defined as a mean of ≤4 hours of use per night.

• Patients generally make the decision to adhere to CPAP therapy early during the first week of therapy, usually by the second to fourth day.

• Even one night without CPAP may mitigate the benefits of CPAP therapy.
  - Including fewer apneas and hypopneas, reduced daytime sleepiness, and improvements in sleep architecture, daily activity, quality of life, hypertension, and neurobehavioral performance.
Interventions

• Several behavioral interventions have been shown to improve CPAP adherence.
  ▪ Cognitive behavioral therapy plus education to help reinforce good behaviors regarding CPAP therapy.
  ▪ Motivational interviewing including interaction with the patient utilizing questions to identify perceptions, behaviors, and volitions that are barriers to engaging in the treatment.
  ▪ Frequent contact and follow-up with a health care provider who has expertise in treating sleep disorders is especially important during the first week of therapy.
  ▪ Troubleshooting mask leaks, addressing fit issues, determining best mask type, adjusting pressure settings on CPAP machine, changing humidifier and air temperature settings, etc.
Compliance

• Usually required by insurance companies within the first 90 days of starting therapy.
• Data can be downloaded from the actual machine, or accessed online (AirView).
• Patients can access their compliance data via phone apps as well to help encourage them to stick to CPAP therapy.
In Conclusion...

• Obstructive sleep apnea (OSA) is a common disorder in adults.
• The prevalence of OSA in the general adult population is approximately 20 to 30 percent in males and 10 to 15 percent in females.
• A detailed sleep history looking for symptoms in conjunction with a screening tool can help determine patients who are more likely to have underlying OSA.
• In-lab polysomnography is the preferred diagnostic study when OSA is suspected, but home sleep apnea testing is an acceptable alternative.
• Gold standard for treatment of OSA is CPAP therapy, with other lesser options available for those who may not tolerate CPAP.
• Early intervention is important in maintaining compliance in patients.
• Role of primary care physicians in following up OSA and performing direct studies.
Fin
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