Obstructive sleep apnoea-hypopnoea syndrome

The beginning of health is sleep – Irish proverb

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Obstructive sleep apnoea is a subject that has been extensively researched in recent years by sleep specialists. Sleep specialists come from a diverse group of clinicians, including ENT specialists, pulmonologists, cardiologists, paediatricians and psychiatrists. The term *obstructive sleep apnoea-hypopnoea syndrome* (OSAHS) is a more apt description and will be used as such in this article.

Definitions

- *Snoring* is a sound originating from the upper airway due to obstruction.
- *Apnoea* is the cessation of airflow at the mouth and/or nostrils for a minimum of 10 seconds.
- *Hypopnoea* is a 50 % reduction in airflow (for a minimum of 10 seconds), terminated by an arousal or followed by a minimum of 2% reduction in oxygen saturation.
- *Obstructive sleep apnoea-hypopnoea syndrome (OSAHS)* is defined as excessive daytime sleepiness (EDS) and an apnoea-hypopnoea index (AHI) of at least 5 apnoeas plus hypopnoea per hour of sleep.
- *Upper airway resistance syndrome (UARS)* is a sleep breathing disorder in which there are increased breathing efforts during periods of increased upper airway resistance, but in the absence of apnoea or hypopnoea. Multiple micro-arousals precipitate cardiovascular strain and EDS. Oxygen saturation is maintained at >90%. UARS may be present in the absence of snoring.

Snoring and OSAHS are progressive conditions

The mean apnoea index (AI) in normal subjects is 1 in males and 0.3 in females.

Pathophysiology

Hypoxia:

- Increases sympathetic activity, causing peripheral vasoconstriction and therefore pulmonary and systemic hypertension.
- Dysrythmias are more prevalent.

The increase in inspiratory efforts during obstruction stimulates vagal activity, resulting in bradycardia.

Obesity leads to:

- Compression of the pharyngeal airway by the excess cervical adipose tissue
- Decreased lung volumes
- Compromised pulmonary and pharyngeal reflexes.

Heavy snoring, *even without recorded apnoeas*, may affect pulmonary artery pressure. It may even cause EDS and have some health consequences.

Risk factors for OSAHS

Major independent risk factors for snoring include:

- Male gender
- Age 40 – 64 years
- Obesity (BMI>40 kg/m²)
- Cigarette smoking (>40/day)

Snoring and OSAHS are progressive conditions. A person usually snores at the age of 30 and progresses to OSAHS at the age 45.

Other risk factors for OSAHS include:

- Family history of snoring
- Neck circumference (NC) measured on the level of the thyrohyoid membrane: male≥43 cm; female≥40 cm
- Nasal obstruction
- Medical conditions, e.g. diabetes, cardiac failure, Parkinson's disease
- Endocrine disorders, e.g. hypothyroidism, acromegaly

- Hypertrophied tonsils and adenoids
- Hypoplasia of mandible/maxilla (Marfan, Pierre-Robin and Down syndromes)
- Neuromuscular diseases.

Prevalence

In an epidemiological study done by Madison in Wisconsin (n=602):

- 9% of females and 24% of males are estimated to have OSAHS (AI>5 and EDS).
- OSAHS among patients with essential hypertension was found to be in excess of 25%.
- The minimum prevalence of clinically significant OSAHS is 1%.

There is a significantly higher prevalence rate of OSAHS among obese subjects and hypertensives.

Obesity as a risk factor for snoring and OSAHS

A body mass index (BMI)≥28 kg/m² is present in 60 – 90 % of patients with OSAHS.

With a BMI≤28 kg/m², central obesity measurements and neck circumference exhibit a better correlation with OSAHS than does BMI.

Katz reported neck size to be more closely related to the severity of OSAHS than the BMI. Neck size is a useful indicator of upper body obesity.

Arterial hypertension

Several cross-sectional studies in Italy, Canada, and Scandinavia have shown an association between snoring and hypertension. Snoring may influence blood pressure via changes in intra-thoracic pressure, OSAHS and nocturnal hypoxemia.

According to Carlson, the combined effect of obesity and OSAHS resulted in a 3.9-fold increase in the prevalence of hypertension.

Snoring, OSAHS and heart disease

Several studies have found a significant association of OSAHS with myocardial infarction. This was independent of age, BMI, arterial hypertension, smoking and cholesterol levels (Peker).

In patients with coronary artery disease, the odds ratio for OSAHS was similar to the odds ratios of other risk factors such as current smoking, diabetes and hypertension.[1]

Men with an AHI of >5.3 had a 23.3-fold risk for myocardial infarction (MI) compared to men with an AHI of <0.4.

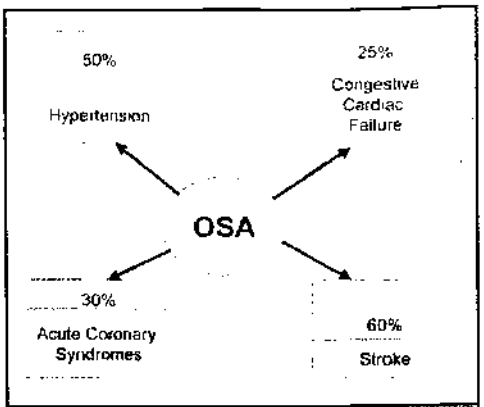

Figure 1. Prevalence of obstructive sleep apnoea (OSA) in patients with cardiovascular and cerebrovascular disease

* The figures used are approximations from published data, and are unadjusted for baseline variables (1 to 4):

1. Parati G, Ongaro G, Bonsignore SIR, Glavina F, Di Rienzo M, Mancia G. Sleep apnoea and hypertension. *Curr Opin Nephrol Hypertens* 2002;**11**:201-14.
2. Sin DD, Fitzgerald F, Parker JD, Newton G, Floras JS, Bradley TD. Risk factors for central and obstructive sleep apnea in 450 men and women with congestive heart failure. *Am J Respir Crit Care Med*.1999;**160**:1101-6;
3. Bassetti C, Alrich, MS. Sleep apnea in acute cerebrovascular diseases: final report on 128 patients. *Sleep* 999;**22**:217-23
4. Peker Y. and others. An independent association between obstructive sleep apnea and coronary artery disease. *Eur. Respir. J* 1999;**14**:179-84.

Patients with nocturnal angina pectoris may be found to have obstructive apnoea. Some may be referred for asymptomatic bradyarrhythmia.

Snoring and brain infarction

The prevalence of habitual snoring was 23.3% among patients with stroke and 8.3% among controls.

Even after adjustment for age, sex, arterial hypertension, cardiac arrhythmias and obesity, the odds ratio of habitual snoring for stroke remained statistically significant.

Routine investigation of stroke patients for OSAHS cannot be recommended.

Snoring and sudden death

Cardiovascular cause of death was more common among habitual snorers and often snorers, than among occasional or never snorers. Habitual snorers died more often while sleeping.

Table I.

Night time:	Daytime:
Snoring	Sleepiness
Witnessed apnoea	Fatigue
Choking	Morning headaches
Dyspnoea	Poor concentration
Restlessness	Decreased libido or impotence
Nocturia	Decreased attention
Diaphoresis (especially upper body)	Depression
Reflux	Decreased dexterity
Drooling	Personality changes

Table II.

0 = Would never doze
1 = Slight chance of dozing
2 = Moderate chance of dozing
3 = High chance of dozing

CHANCE OF DOZING	SITUATION 0	1	2	3
Sitting and reading				
Watching TV				
Sitting inactive in a public place (e.g., a theatre or a meeting)				
As a passenger in a car for an hour without a break				
Lying down to rest in the afternoon when circumstances permit				
Sitting and talking to someone				
Sitting quietly after lunch without alcohol				
In a car, while stopping for a few minutes in traffic				

Clinical symptoms of OSAHS *(Table I)*

Although up to 30% of pregnant women snore, overt OSAHS is uncommon.

Some patients may present with polycythemia, proteinuria or a nephrotic syndrome.

OSAHS patients may also have seizures during sleep or may be found to have a patent foramen ovale. Postanaesthetic respiratory failure may be caused by OSAHS.

Oesophageal reflux results from increased gastric pressure during episodes of upper airway obstruction and a subsequent increase in breathing effort and abdominal pressure. Patients often report awakening with heartburn.

Nocturia is common. Studies showed up to 28% of patients making 4-7 nightly trips to the bathroom. Increased intra-abdominal pressures, confusion associated with arousals, and increased secretion of atrial natriuretic peptide have been postulated to contribute.

Dry mouth (74%) and **drooling** (36%) are common symptoms with OSAHS.

Daytime symptoms

Excessive daytime sleepiness (EDS) is the most common symptom. EDS is seldom experienced with an RDI<20. There is an increased risk of car accidents with an AHI>15.

The Epworth Sleepiness Scale is used to determine the severity of EDS. It may be subtle, such as mid-afternoon drowsiness during a group meeting or occasional naps, or severe, such as falling asleep while driving.

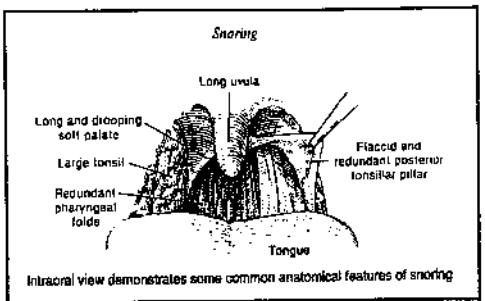

Figure 2.

THE EPWORTH SLEEPINESS SCALE *(Table II)*

The average score is 7. An Epworth score above 15 strongly suggests OSAHS.

Personality changes such as aggressiveness, irritability, anxiety or depression and even psychosis may be observed. Family and social life may suffer considerably. Alienation may lead to depression.

A third of patients report **decreased libido** or **impotence.**

Morning or nocturnal headaches may result and usually last 1–2 hours.

Clinical examination

Obesity and neck circumference (NC):

BMI and NC should be measured. NC is measured at the level of the thyrohyroid membrane with the patient in the upright position.

In patients *with* OSAHS the average NC was 43.7 ± 4.5cm and patients *without* OSAHS the average NC was 39.6 ± 4.5cm.

Kushida found the cut-off level of 40 cm as having a sensitivity of 61% and a specificity of 93% for OSAHS regardless of sex.

Upper airway:

- Examine the patient seated and supine.
- Retrognathia and dental overbite should be identified.
- Tongue–macroglossia is a predisposing factor.
- Uvula and velum – look at the size, length and height. Oedema, hyperaemia and mucosal folds (telescoping of uvula) are also indicative of possible OSAHS.
- Tonsillar hypertrophy and the size of the tonsillar pillars must be evaluated.
- Make a visual estimate of the retroglossal area.
- Nose: examine the septum and turbinates for deviation, polyps, etc.
- Nasal obstruction itself cannot be a main factor causing OSAHS, but a co-factor of OSAHS during sleep.[2]
- Nasal obstruction may cause habitual snoring, sleep fragmentation, sleep deprivation and daytime tiredness.

Lower airway

Floppy redundant supraglottic mucosa may also contribute to OSAHS. A fibre-optic nasopharyngolaryngoscopy is essential to adequately assess the abovementioned anatomical areas.

The Muller manoeuvre is a subjective examination done with the nasopharyngoscope. This demonstrates lateral pharyngeal collapse at the retropalatal and retrolingual levels. The breath is held at the end of expiration and the size of the airway is classified from 1 to 4.

Sleep-endoscopy (jaw lift) and sedation-endoscopy are also utilized to assess the upper airway.

Special investigations:

Blood

- Haematocrit
- Thyroid function test
- Blood gas analysis

Pulmonary function test

- Lung function testing is only indicated when conditions other than OSAHS are suspected.

Lateral cephalometric radiographs

In patients with severe OSAHS and hypoplasia of the maxilla and/or retrognathia,

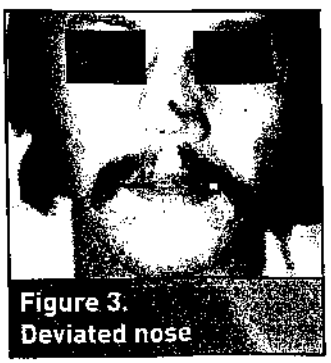

Figure 3. Deviated nose

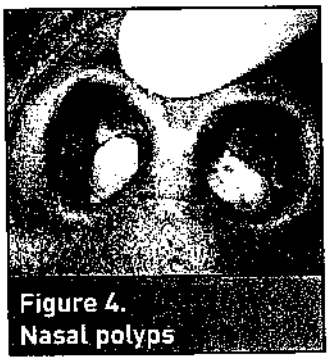

Figure 4. Nasal polyps

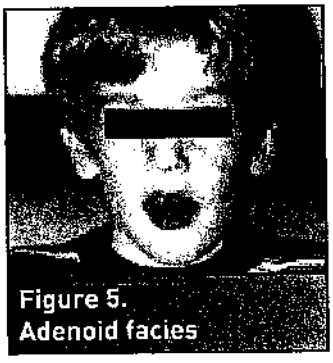

Figure 5. Adenoid facies

cephalometric measurements are indicated to identify those who might need facial skeletal surgery.

Overuse of this technique by itself to determine the exact surgical intervention is not recommended.

CT and MRI scanning are reserved as investigational tools.

The history and physical examination can predict OSAHS in only about 50% of patients. Definitive diagnosis requires a polysomnogram (PSG).

Polysomnogram (PSG)

A negative PSG most often will rule out OSAHS. If the history is strongly suggestive of OSAHS (snoring, observed apnoea and EDS) and the PSG is negative, the test may have to be repeated.

AHI≥5 along with EDS define OSAHS. Several laboratories use AHI≥10. The severity should be based not only on the RDI, but also on the degree of daytime sleepiness and other PSG features, such as the degree of sleep fragmentation, oxygen desaturation and the presence of cardiac arrhythmias.

Overnight pulse oximetry is **not** a substitute for PSG.

Treatment

Medical therapy

At Stanford University, approximately 85% of patients with OSAHS are treated medically. Data indicate that the presence of >20 apnoeas per hour of sleep may predict early mortality.

Obesity:

It has been well documented that either medical or surgical weight reduction often has a substantial ameliorative impact on this disorder. Insulin resistance is often associated with OSAHS and treatment of OSAHS results in improved glucose metabolism.

Sleep deprivation:

Absolute deprivation as well as repetitive sleep disruption may also predispose or worsen existing OSAHS. Patients should be encouraged to maintain good sleep hygiene. The average adult should sleep 7 to 8 hours per night.

Modification of body position:

A bed partner's prompting constitutes one of the oldest interventions for snoring and OSAHS. Upper airway dysfunction is particularly notable during sleep in the supine position, owing to the effect of gravity on the tongue, which tends to relapse posteriorly and come into apposition with the posterior pharyngeal wall.

Pharmacological agents that adversely affect upper airway function during sleep:

- Alcohol
- Benzodiasepines
- Narcotics and anaesthetics
- Barbiturates

These agents selectively reduce the neural output via the hypoglossal nerve, reduce the tone of the upper airway dilation muscles, and predispose to upper airway occlusion during sleep.

Some beta-blockers and alpha-methyldopa act centrally and may worsen the apnoeas. Consider changing to alternatives like ACE-inhibitors.

Diuretic use results in a metabolic alkalosis that aggravates hypoventilation.

Mechanical devices and techniques for maintenance of upper airway patency during sleep (exclusive of continuous positive airway pressure (CPAP)):

Oral appliances fall into two main categories: those that hold the tongue forward and those that reposition the mandible (and attached tongue) forward during sleep. These devices may prove to be successful in up to 60% of carefully selected patients.

A nasopharyngeal airway:

This method has limited therapeutic utility and is not well tolerated.

Electrical stimulation of the UA:

Limited available data suggest that this improves, but may not eliminate OSAHS.

Nasal dilators:

These may offer some benefit for a few selected patients, but are of no proven value for most OSAHS patients.

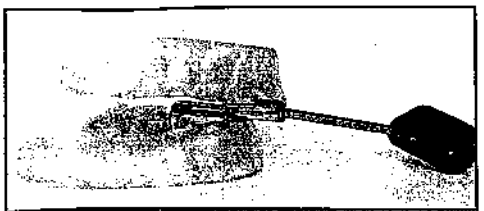

Figure 6. Mandibular advancement device.

Pharmacological therapy:

- Protripteline
  There is a lack of clinically significant therapeutic impact and it poses some troublesome side effects.
- Oxygen
  Providing supplemental oxygen is not effective in reducing the frequency of apnoeas and increasing daytime alertness.
- Agents with an uncertain or limited therapeutic role:
  Stimulants: Some patients remain unacceptably and perhaps dangerously sleepy or inattentive. Stimulants may provide symptomatic benefit.

CPAP:

This is an established form of treatment for OSAHS and for some forms of central apnoea.

Nasal CPAP was first described in 1981 in Sydney, Australia. It acts as a pneumatic splint to prevent collapse of the pharyngeal airway. MRI studies confirmed the pneumatic splint of CPAP and its ability to reduce upper airway oedema secondary to chronic vibration and occlusion of the airway.

With correct CPAP levels, there is often "rebound" slow-wave and REM sleep, lasting about a week.

Inadequate CPAP levels are diagnosed when there are continued arousals and continued snoring.

Surgical therapy for OSAHS

Surgery for obesity (bariatric surgery):

This is only considered in patients with a BMI>40 kg/m² or a BMI>35kg/m² in combination with other co-morbidity including life-threatening cardiopulmonary problems, severe diabetes mellitus, obesity-related pulmonary hypertension and degenerated joint disease.

Surgery involving the upper airway:

Fujita classification of obstructive regions:
- type I : palate (normal base of tongue)
- type II : palate plus base of tongue
- type III : base of tongue (normal palate)

In a group of patients with severe OSAHS, pharyngeal manometry demonstrated that 35–50% of patients have tongue base or hypopharyngeal obstruction. Endoscopic studies during sleep suggested that 30–40% of cases obstruct at the tongue base or hypopharynx.

Nasal reconstruction

This is suggested for any patient with notable obstruction of the nasal airway.

A patent nasal airway is also a prerequisite for compliant CPAP therapy.

Palatal procedures

- **Uvulopalatopharyngoplasty (UPPP)**
  This was proposed by Ikematsu in 1964 for the treatment of habitual snoring. Fujita (1981) adopted the UPPP for the treatment of OSAHS as well as snoring.
  The response rate of UPPP in severe OSAHS (RDI>20) varies from 0–50%. A meta-analysis by Sher *et al.* reported a 39% success rate of UPPP for correcting OSAHS. UPPP has seldom been credited with curing moderate or severe OSAHS. Woodson advocates careful selection of patients.
- **Even the best palatal surgery cannot stand against severe retrolingual obstruction.**[2] Although there is often an improvement in sleep-related breathing following UPPP, the degree of improvement is frequently insufficient to define surgical success. The incomplete response to UPPP is almost always due to the fact that other regions of obstruction exist. UPPP fails in 50–70% to decrease the RDI<20.
- **Uvulopalatal flap (UPF)**
  A modification of UPPP is the uvulopalatal flap by Riley and Powell. Compared to UPPP, the UPF has a decreased risk of nasopharyngeal incompetence and nasal stenosis. UPF is also less painful and potentially reversible.
- **Laser-assisted uvuloplasty (LAUP)**
  Introduced by Kamami in 1990. This progressively shortens and tightens the uvula through a series of CO_2 laser incisions and vaporizations.
- **Z-palatoplasty**
  Friedman proposed this procedure for patients without tonsils. The direction of scar contraction is opposite to that of the UPPP, thus achieving a better objective success rate compared to UPPP.
- **Transpalatal advancement procedure (TPA)**
  TPA increases the oropharyngeal size by palatal advancement and not by soft tissue excision. This procedure is indicated when the soft palate is of normal length, but with a narrow retropalatal space, for mild to moderate disease. Good lateral wall movement is a prerequisite for TPA and UPPP.
  Results: the retropalatal area increased by 321% in patients following TPA and the closing pressures decreased by an average of 8.4 cm H_2O.[3] In a study it was found that TPA alters the retropalatal airway characteristics more than UPPP alone.
- **Palatal stiffening procedures**
  These procedures are mostly indicated for habitual snorers and mild sleep apnoea in carefully selected patients:

- Injection snoreplasty using sclerotherapy
- Somnoplasty using radio frequency (temperature-controlled radiofrequency tissue ablation: TCRFTA)
- Palatal implants.

The future of effective palatal surgery for OSAHS rests with the Z-palatoplasty and the transpalatal advancement procedures.

Procedures involving the hypopharynx and base of tongue

- **Genioglossus advancement (GA)**
  The genial tubercle and genioglossus–hyoid complex are advanced. An osteotomy is performed at the genial tubercle. This advancement places tension on the tongue musculature and thereby limits posterior displacement during sleep. No additional room is anatomically created for the tongue.
- **Hyoid advancement (HA)**
  The HA was first described in 1984. Forward movement of the hyoid improves the posterior airway space down to vallecular level. The lower tongue base (level of mandibular plane) is addressed with the HA procedure.
  Numerous reports have supported the concept that surgical intervention at the hyoid complex level improves the hypopharyngeal airway. Kaya is credited with the first report, followed by Patton. Indications: Fujita type II and III obstruction.
  In case of a "toilet lid" (omega shaped and retro-displaced) epiglottis, the HA is very effective. The HA procedure is totally reversible. Tongue volume reduction procedures may be combined with the HA.
- **Radiofrequency volume reduction (RFTVR) of tongue base**
  This is a minor procedure directed at reducing the tongue base volume to treat obstructive sleep apnoea. Radiofrequency energy is delivered submucosally to the base of tongue, coagulating the tissue beneath the surface. This leads to reduction in volume and stiffening.
  In mild obstructive sleep apnoea, treatment of symptomatic outcomes with RFTVR may be an alternative to CPAP.[4]
- **Partial midline glossectomy**
  Macroglossia is defined by a posterior airspace (PAS) of <10mm, without retrognathia (sella-nasion-supramentale [SNB]>76°). The PAS is calculated as the narrowest distance from the tongue base to the posterior pharyngeal wall, parallel to the Frankfurt horizontal plane.[5]
  The coblation technique (applied at numerous sleep centres in the USA, EU and Australia) has brought a new dimension to the concept of tongue volume reduction. Previously, this was done by electrocauterization or laser, causing significant thermal insult to the tissue. The resultant oedema necessitated a tracheotomy to protect the airway, therefore increasing the morbidity and mortality of the procedure. The coblation wand operates at 60 degrees centigrade, therefore reducing the thermal damage to the tissue significantly. Intra- and post-operative haemorrhage is not significant with the coblation wand.
  The procedure is performed through a midline incision, using the coblation Evac 70™ wand. Coblation of tissue is largely restricted to the middle third of the tongue base as a result of the location of the neurovascular bundle entering the tongue base laterally. Localization of this structure (which has a variable position) by ultrasound enables more extensive dissection laterally with minimal risk of neurovascular damage.[5]

The hyoid advancement, RFTVR and partial glossectomy of the tongue base are the procedures of choice for hypopharyngeal obstruction in OSAHS.

Orthognatic surgery

The practice of maxillo-mandibular advancement as primary treatment modality is generally not advocated due to acceptable cure rates with the less invasive phase I surgery.
Types of surgery include:

- Mandibular advancement osteotomy
- Distraction osteogenesis
- Maxillo-mandibular osteotomy

Tracheotomy/tracheostoma

Tracheotomy is done in cases of severe life-threatening sleep apnoea, i.e. an RDI>60 and SaO_2<60%, where patients are intolerant of CPAP.

Conclusion

Obstructive sleep apnoea is one of the most prevalent conditions, affecting almost any age group from neonates to the geriatric population.

The syndrome is often not recognized by medical practitioners or medical specialists.

Patients very seldom realize that they are affected; therefore awareness about OSA is of vital importance.

Management of OSA requires a multidisciplinary approach.

References available on request.