

# OBSTRUCTIVE SLEEP APNOEA

## WARNING SIGNS OF THE SLEEPY SNORER

### Definition of Obstructive Sleep Apnoea

Seven names - 1 condition! The definition of sleep apnoea can be confusing because it is described in research literature under many guises:

Obstructive Sleep apnoea (OSA)

Sleep Apnoea (SA)

Obstructive Sleep Apnoea Syndrome (OSAS)

Sleep Apnoea/Hypopnoea Syndrome (SAHS)

Obstructive Sleep Apnoea/Hypopnoea Syndrome (OSA/H)

and of late, a new condition has been introduced Upper Airway Resistance Syndrome (UARS).

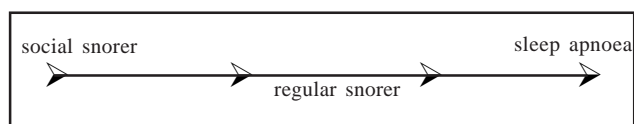
Obstructive Sleep apnoea is characterised by a series of signs and symptoms, but specifically by the occurrence of repetitive episodes of partial or complete collapse of the upper airway which prevents breathing. This is known as Apnoea. These apnoea episodes are usually accompanied by loud snoring, excessive daytime sleepiness and a reduction of blood oxygen saturation. Physiologically these episodes are generally associated with arousals, sleep fragmentation, intermittent hypoxemia, hypercapnia and nocturnal hypertension.

To add to the confusion there is also the condition Central Sleep Apnoea (CSA), which occurs due to a loss of sensitivity of the chemoreceptors and neural activity in the brain stem. This results in the brain 'forgetting' to initiate breathing during sleep. Some patients experience Mixed Sleep Apnoea which is a combination of both OSA and CSA. Central Sleep Apnoea is a very rare condition and cannot easily be recognised by signs and symptoms that present with OSA.

### Development of OSA

OSA is not a condition that develops spontaneously, rather it can best be described as a continuum of snoring (Fig 1).

Figure 1 snoring continuum



A 'social snorer' is typically a young male of around 25-30 years of age who snores occasionally, possibly following a large intake of alcohol. By the time the 'social snorer' has reached the age of 30-35 years without the snoring being addressed the likelihood of progressing to a 'regular snorer', whereby snoring is occurring every night, is high. If the snoring is still left unaddressed by the time the 'regular snorer' has reached the age of 40-45 years the snoring will most likely have developed into the more serious condition of OSA. Because of this slow development over many years it is often not recognised as the patient is unaware that their seemingly harmless snoring has deteriorated to such an extent that it has become a clinically serious condition.

### Mechanisms of OSA

OSA occurs due to a partial or complete obstruction somewhere in the upper airway (anywhere in the region of the nose, throat or mouth area) which prevents air from entering the lungs. This obstruction causes a narrowing of the airway and an increase in pharyngeal resistance with the generation of excessive negative pressure during inspiration. This narrowing requires an increase in pharyngeal dilator muscle activity to maintain airway patency and there is evidence that patients with OSA lack this muscle tone (McNicholas 2002 ).

### Physical properties which may contribute to the narrowing of the airway include:

#### Neck obesity

(anatomically short, fat neck)

#### Fat deposition in the oropharynx

(due to increased body weight/obesity)

#### Mandibular-pharyngeal abnormalities

(incorrect jaw shape)

#### Loss of muscle tone in the pharyngeal dilator muscles during sleep

(due to vibratory trauma from long term snoring or simply from an excess of alcohol intake or sedatives)

#### Size of surrounding soft tissue structures

(tongue, soft palate, lateral pharyngeal walls, tonsillar hypertrophy)

## Assessment of OSA

There are various terms used to describe the events that occur during sleep and it is generally agreed that a definitive diagnosis of OSA depends on certain criteria whose presence can only be elicited by polysomnography performed in a specialized sleep laboratory (Neven et al 1998). However due to the large numbers of patients presenting for assessment it is not possible to perform full polysomnography on every patient and it is for this reason that home-based sleep studies have become more frequent. Douglas (2003) in his paper discusses the benefits and limitations of home diagnosis.

A full polysomnography would include tests to determine apnoea/hypopnoea index (AHI), desaturation index (DI), sleep staging, various indices of sleep disordered breathing and arousal, body position, respiratory disturbance index for each stage and posture. A 'home study' kit will offer varying information depending on the equipment used, but data will be limited compared to full polysomnography. The only common variable to all diagnostic systems is oxygen saturation (SaO<sub>2</sub>) (3).

### What information do we need to make a clinical diagnosis?

Apnoea/Hypopnoea Index (AHI) - the number of respiratory disturbances per hour of sleep. Hypopnoeas are usually considered as a reduction in airflow, or respiratory movement of 50% with desaturation.

An AHI of at least 5, each lasting >10 seconds

A fall in SaO<sub>2</sub> >4%.

Degree of Excessive Daytime Sleepiness (EDS) usually defined using the Epworth Sleepiness Scale (ESS) questionnaire. EDS is the most important symptom of OSA and a score >10 would be highly significant.

The severity of OSA is defined by the AHI ranging from >5 (mild) to >20 (severe).

When a patient presents for assessment there are several signs and symptoms that are often immediately recognisable. Some of these symptoms are better described by the bed partner than the patient:

Obesity (a collar size >16½" will predispose to OSA.)

Excessive alcohol intake.

Heavy smoking.

Extremely loud heavy snoring often interrupted by pauses and gasps.

Excessive daytime sleepiness, e.g., falling asleep at work, whilst driving, during a conversation or when watching TV (this should not be confused with excessive tiredness with which we all suffer from time to time).

Morning headaches.

Irritability and short temper,

Forgetfulness.

Changes in mood or behavior.

Anxiety or depression.

Decreased increase in sex.

Every one of us has at some time experienced one or all of these symptoms. Does this mean we have all developed OSA? Of course such a notion would be absurd. However, it does demonstrate that we need to look carefully at the individual patient's symptom severity rather than make a diagnosis on whether their symptoms are within the clinical definition.

### Prevalence of OSA

OSA is estimated to affect around 4% of men (Kryger 2002, Levy et al 2002, McArdle et al 2001, Pack et al 2001, Schwab1999) and 2% of women (Kryger 2002, Levy et al 2002, McArdle et al 2001, Pack et al 2001, Schwab1999).

However, some studies have suggested that figures are much higher. Sjostrom et al (2002) estimate 24% of men and 9% of women in the middle aged population suffer from OSA, whilst Neven et al (1998) estimate that at least 45% of men aged 35 and over suffer from clinically significant OSA. Due to lack of awareness among the general population and physicians it has been suggested that an estimated 80 to 90% of OSA sufferers have not received a clinical diagnosis (Hossain & Shapiro 2002). These

widely ranging estimates are probably due to differences in definitions, in the design of the studies and the investigations performed, in the age, sex and other characteristics of the populations surveyed (Neven 1998).

Having discussed differences in definition and investigation outcomes and the notion that OSA can be classed anywhere from 'mild' to 'severe', how then do clinicians make a decision on treatment. The recommended treatment for OSA is CPAP (Continuous Positive Airway Pressure)(Kryger 2002, Levy et al 2002, McNicholas 2003), but should patients be prescribed this treatment regardless of the severity of their symptoms? If their symptoms fall within the clinical criteria should *all* patients be prescribed CPAP? Conversely, if patients falls short of clinically significant SA but are clearly suffering from other symptoms such as EDS, why should they not be treated with CPAP to alleviate these symptoms? These dilemmas are epitomised by the following two scenarios which are common reasons for patients contacting the office of British Snoring & Sleep Apnoea Association (BSSAA)

One patient had recently undergone a sleep study and the results confirmed clinically diagnosed sleep apnoea. The patient was offered CPAP treatment which he declined. His reasoning for refusing CPAP was that he only sought a consultation in order to obtain a resolution to his snoring and as far as he was concerned he experienced none of the symptoms of OSA. In fact this patient was quite categoric in that his only symptom was excessively loud snoring so why should he need CPAP? But the results of an overnight polysomnography clearly demonstrated, according to the recognised diagnostic criteria, that he had OSA and therefore he should be treated accordingly!

Another patient following several overnight sleep studies did not demonstrate clinically recognised OSA. He was clearly suffering from all the symptoms, in particular EDS to the point where he regularly had to force himself to stay awake at work, and when driving, always had to pull off the road at some point for a short 'nap'. But according to the clinical criteria the patient

did not have OSA and was not offered CPAP treatment.

According to Kryger (2002) "Every physiological function has a normal range which can be defined statistically if we can measure the function. Being outside the normal statistic range of measurement does not mean that a disease is present or there is anything to treat... Do we really know the normal range of breathing abnormalities during sleep or even how best to measure them? The number of abnormal events is not important but whether the abnormal events cause abnormal function that is deleterious".

Kryger (2002) continues "If episodes of OSA did not affect the function of the patient, (e.g. sleepiness, cognitive impairment and so forth), would we treat it? The answer is no. A patient with a small number of apnoeic episodes may have only mild sleepiness; mild sleepiness can be catastrophic to the lawyer who can no longer concentrate and remember the fine details of a case".

If we use this analysis for the two previous scenarios the treatment regime appears irrational. Evaluating a patient requires skill and diagnostic criteria surely is only a guide. If the quality of life of patients is impaired because of their symptoms should CPAP not be offered? It is our experience at BSSAA that patients whose quality of life is most impaired are more likely to comply with long-term use of CPAP as opposed to those patients whose significant diagnostic criteria and absent symptoms cause them little or no dysfunction.

### **Treatment of OSA**

Continuous Positive Airway Pressure (CPAP) is the recommended treatment for sleep apnoea. Since Sullivan et al introduced CPAP over 20 years ago, it has remained a well proven treatment in reversing the symptoms of OSA. CPAP therapy acts as a 'pneumatic splint' blowing room air into the airway, normally via a nasal mask, at a positive pressure in order to keep the airway open and thus preventing collapse.

However, according to Malhotra & White (2002) CPAP adherence is difficult with the best compliance being in patients with severe OSA and substantial sleepiness.

Patients discontinue the use of their CPAP for several reasons. Physical problems include: mask leak, dry throat/mouth, pressure sores and eye infections. However, according to Engleman & Wild (2003) "Biomedical investigations of determinants of CPAP adherence have shown largely weak relationships between symptomatic/polysomnographic disease severity and subsequent CPAP use". They believe that "cognitively derived perceptions of health status, health beliefs and attitudes may underlie CPAP adherence behavior". Based on this evidence it would be advantageous for providers to offer a long-term support service in order to increase compliance. Engleman & Wild (2003) in their paper offer a series of practical solutions to ensure greater compliance for their patients.

### **CPAP is not for all patients**

In mild OSA there are alternative treatments. Oral appliances such Mandibular Advancement Splints (MAS) have become popular not least because they are far less obtrusive than CPAP.

However before other treatments are considered, individuals with clearly reversible causes should be advised to make some life-style changes which would contribute considerably to alleviating their symptoms. Malhotra & White (2002) suggest "conservative measures should be emphasized, including maintenance of nasal patency, avoidance of depressants including alcohol, and the goal of 7-8 hours sleep per night. In addition, individuals with documented positional apnoea should be encouraged not to sleep on their backs".

For some patients CPAP does not always resolve EDS and additional treatments may be of benefit for regular users of CPAP therapy. In a recent study the use of Modafinil (Provigil) significantly reduced symptoms of EDS and is now 'recognised as a useful adjunct treatment for the management of residual daytime

sleepiness in patients with OSA/Hypopnoea syndrome who are regular users of nasal continuous positive airway pressure therapy.(Pack et al 2001) Kingshott et al (2001) are clear to point out that this is not a treatment of first-line therapy in SA as it does not treat the underlying upper airway collapse or the resulting arousals and blood pressure changes, and from their study the benefits and limitations should be carefully considered before administration.

### **Conclusion**

In recent years our understanding of OSA has increased considerably in both progression of the disease and treatment options. Diagnostic criteria offers a general guide as to symptom severity but let us not make decisions on treatment from 20 pages of data collection alone. Let us make life simple for both clinicians and patients and ask the patient 'how do you feel?'

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