Scottish Intercollegiate Guidelines Network

73 Management of Obstructive Sleep Apnoea/Hypopnoea Syndrome in Adults

A national clinical guideline

This guideline is endorsed by the British Thoracic Society

June 2003

KEY TO EVIDENCE STATEMENTS AND GRADES OF RECOMMENDATIONS

LEVELS OF EVIDENCE

1^{++}	High quality meta-analyses, systematic reviews of randomised controlled trials (RCTs), or RCTs with a very low risk of bias
1^{+}	Well conducted meta-analyses, systematic reviews of RCTs, or RCTs with a low risk of bias
1^{-}	Meta-analyses, systematic reviews of RCTs, or RCTs with a high risk of bias
2^{++}	High quality systematic reviews of case control or cohort studies High quality case control or cohort studies with a very low risk of confounding or bias and a high probability that the relationship is causal
2^{+}	Well conducted case control or cohort studies with a low risk of confounding or bias and a moderate probability that the relationship is causal
2^{-}	Case control or cohort studies with a high risk of confounding or bias and a significant risk that the relationship is not causal
3	Non-analytic studies, e.g. case reports, case series
4	Expert opinion

GRADES OF RECOMMENDATION

Note: The grade of recommendation relates to the strength of the evidence on which the recommendation is based. It does not reflect the clinical importance of the recommendation.

A	At least one meta-analysis, systematic review of RCTs, or RCT rated as 1^{++} and directly applicable to the target population*; or* A body of evidence consisting principally of studies rated as 1^{+}, directly applicable to the target population, and demonstrating overall consistency of results
B	A body of evidence including studies rated as 2^{++}, directly applicable to the target population, and demonstrating overall consistency of results*; or* Extrapolated evidence from studies rated as 1^{++} or 1^{+}
C	A body of evidence including studies rated as 2^{+}, directly applicable to the target population and demonstrating overall consistency of results*; or* Extrapolated evidence from studies rated as 2^{++}
D	Evidence level 3 or 4*; or* Extrapolated evidence from studies rated as 2^{+}

GOOD PRACTICE POINTS

☑	Recommended best practice based on the clinical experience of the guideline development group

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Scottish Intercollegiate Guidelines Network
Royal College of Physicians
9 Queen Street
Edinburgh EH2 1JQ

www.sign.ac.uk

1 Introduction

1.1 THE NEED FOR THE GUIDELINE

Obstructive sleep apnoea/hypopnoea syndrome (OSAHS) can be defined as the coexistence of excessive daytime sleepiness with irregular breathing at night. The abbreviations OSAS and OSA are used widely and synonymously with OSAHS, however this guideline will use only the latter. OSAHS is a significant public health problem and there is a large and increasing demand for sleep service facilities due to the high prevalence and growing public awareness of sleep disorders, including OSAHS. A conservative estimate of the prevalence of OSAHS in middle-aged men (30-65 years) is in the range 0.3–4%, with most studies giving a prevalence of 1-2% which is a similar prevalence to Type 1 diabetes and approximately double that of severe asthma.[1-4] The prevalence of OSAHS in middle-aged women has been less well studied but is probably about half that in males, at around 0.5–1%.[2]

A significant variation exists across the UK, both in the availability of diagnostic tests and the provision for treatment of sleep-disordered breathing.

The consequences of untreated sleep apnoea on daily function are multiple and include increased daytime sleepiness, impairment of cognitive function, mood and personality changes.[5] Sleep apnoea is also associated with a reduction in quality of life[6] and there can be adverse effects on others including impaired relationships between spouses and partners.[7] Symptoms of sleepiness and impaired concentration resulting from untreated sleep apnoea are thought to have serious consequences during activities where reduced alertness is dangerous, such as driving, leading to an increased risk of road traffic accidents.[8,9] There is objective evidence for a 1.3 to 12-fold increase in accident rates among patients with OSAHS.[2,8,10] Sleepiness at the wheel is estimated to cause about 20% of road accidents on major highways, although it is unclear how many of these are due to OSAHS. These accidents usually occur at high speed, without avoidance reactions and are associated with serious injuries and a high mortality rate.[9,11,12]

The estimated cost to society of a fatal road traffic accident is approximately £1,250,000, making it highly desirable to produce a national guideline which may help to reduce the medical, social and financial costs of excessive sleepiness.[13]

1.2 REMIT OF THE GUIDELINE

This guideline presents evidence based recommendations for the diagnosis and management of obstructive sleep apnoea/hypopnoea syndrome in males and females over 16 years. It is not intended to exhaustively cover all causes of excessive daytime sleepiness in adults nor does it deal with central sleep apnoea nor specifically with snoring. The guideline aims to produce recommendations which can be used to aid patients, general practitioners (GPs), secondary care physicians and surgeons to recognise the symptoms of this common disorder, to prioritise referral requests, to understand how sufferers may be investigated and which treatment modalities are currently available.

1.3 STATEMENT OF INTENT

This guideline is not intended to be construed or to serve as a standard of medical care. Standards of care are determined on the basis of all clinical data available for an individual case and are subject to change as scientific knowledge and technology advance and patterns of care evolve. These parameters of practice should be considered guidelines only. Adherence to them will not ensure a successful outcome in every case, nor should they be construed as including all proper methods of care or excluding other acceptable methods of care aimed at the same results. The ultimate judgement regarding a particular clinical procedure or treatment plan must be made by the doctor, following discussion of the options with the patient, in light of the diagnostic and treatment choices available. However, it is advised that significant departures from the national guideline or any local guidelines derived from it should be fully documented in the patient's case notes at the time the relevant decision is taken.

1.4 REVIEW AND UPDATING

This guideline was issued in 2003 and will be considered for review in 2006, or sooner if new evidence becomes available. Any updates to the guideline will be available on the SIGN website: **www.sign.ac.uk**

2 Definitions and clinical background

2.1 CLINICAL FEATURES

There are many causes of excessive sleepiness but the commonest treatable medical cause is the obstructive sleep apnoea/hypopnoea syndrome (OSAHS). This is a clinical condition, with recognisable symptoms, that occurs because the upper airway collapses intermittently and repeatedly during sleep. This collapse can be complete, with total obstruction of the airway lumen and no respiratory airflow (apnoea), or partial, with reduction in the cross-sectional area of the upper airway lumen causing hypoventilation (hypopnoea). An apnoea is arbitrarily defined in adults as a ten second breathing pause and an hypopnoea as a ten second event where there is continued breathing but ventilation is reduced by at least 50% from the previous baseline during sleep. In some centres, hypopnoeas are defined using additional criteria including associated oxygen desaturation (dips) or electroencephalogram (EEG) arousal.

As the sufferer falls asleep the muscle tone in the upper pharyngeal airway decreases leading to upper airway narrowing. This, in turn, produces an increase in inspiratory effort in an attempt to overcome this airway narrowing which then leads to a transient arousal from deep sleep to wakefulness or a lighter sleep phase which allows restoration of normal airway muscular tone and calibre. The patient then falls more deeply asleep again and the whole cycle repeats itself. This can occur many hundreds of times throughout the night leading to fragmentation of normal sleep architecture and a reduction in the quality of sleep with the generation of restless, disturbed and unsatisfying sleep. This in turn produces the symptoms of excessive daytime sleepiness, poor concentration and a reduction in alertness.

Factors predisposing to apnoeas and hypopnoeas include:

- increasing age
- male gender
- obesity
- sedative drugs
- smoking and alcohol consumption.[4,14]

There may also be a familial component to OSAHS possibly linked to facial or pharyngeal morphology or function but this area requires further research in order to clarify the role played by genetics.[15,16]

2.1.1 OSAHS AND HYPERTENSION

There is an independent association between OSAHS and hypertension. Studies have shown that patients with OSAHS have significantly higher blood pressure (BP) than matched controls.[17-20] Confounding factors such as obesity, age, gender and alcohol consumption make interpretation of many of these studies difficult.[21] Epidemiological studies have shown that the presence of OSAHS is an independent predictor of raised blood pressure even when all known confounding variables have been allowed for.[22-26] Treatment with continuous positive airway pressure (CPAP) therapy reduces BP by up to 3.3 mm Hg over 24 hours. The decrease was greatest in those with most marked nocturnal hypoxaemia (>twenty 4% desaturations/hour) in whom the mean 24 hour fall in diastolic BP was 5 mm Hg.[27] Reduction in BP by this magnitude may decrease cardiac risk by 20% and stroke risk by 40% over a five to ten year period.[28,29]

Claims of a direct association between OSAHS and myocardial infarction and stroke are as yet unproven.

2.2 DEFINITIONS

OSAHS represents one end of a spectrum with normal quiet regular breathing at one end, moving through worsening levels of snoring, to increased upper airways resistance, and to hypopnoeas and apnoeas at the other end. The frequency of apnoeas and hypopnoeas hourly is used to assess the severity of the OSAHS and is called the apnoea/hypopnoea index (AHI) or the respiratory disturbance index (RDI). It is unclear if it is the best measure of this disorder but it is the one most commonly used. Other measures including oximetry, computerised EEG analysis, autonomic arousal detection or body movement analysis, may be equally as good at characterising the severity of sleep apnoea.[30] As these indices are defined in different ways in different centres, comparisons may be difficult. In an attempt to overcome these difficulties recommendations aiming to standardise definitions of apnoeas/hypopnoeas and related indices have recently been published.[31]

OSAHS may be subdivided into varying degrees of breathing abnormality, for example, depending on the AHI:

- mild: AHI 5-14/hr
- moderate: AHI 15-30/hr
- severe: AHI >30/hr

Any cut off in AHI attempting to stratify the severity of OSAHS is arbitrary. Severity can vary from night to night and symptoms from day to day in any individual. Stratification is used to assign patients to an approximate level of severity when considering treatment strategies. Stratification also depends on the severity of the symptoms. In general, the more severe the breathing abnormality, the more symptomatic the patient becomes, but there may be cases where the severity of the symptoms does not correlate with the degree of breathing abnormality. AHI may rise with age in the population and require modification of any stratification system.[32] Further research is required to confirm this.

Table 1: Features of Obstructive Sleep Apnoea

- excessive daytime sleepiness
- impaired concentration
- snoring
- unrefreshing sleep
- choking episodes during sleep
- witnessed apnoeas
- restless sleep
- irritability / personality change
- nocturia
- decreased libido

The dominant symptoms of OSAHS are excessive sleepiness, impaired concentration and snoring.

Clinically significant OSAHS is likely to be present when AHI ≥ 15 events/hour slept, in association with unexplained daytime sleepiness or a minimum of two of the other features of the condition *(see table 1).* There is some evidence of benefit from the treatment of symptomatic individuals with AHI of 5-14, however further studies are required to confirm this *(see section 4.3.1)*.[33,34]

It should be remembered that not every symptom is present in every case. The sufferer may also fail to recognise or indeed underplay some of these symptoms and it is often very useful to seek information from the partner regarding witnessed apnoeas, snoring, nocturnal restlessness and irritability or personality change.

Patients who present with any of the symptoms in table 1 and with specific associated symptoms may be suffering from significant underlying pathologies that merit urgent ear, nose and throat (ENT) assessment. These associated symptoms include: unilateral nasal bleeding, change in voice character, severe nasal obstruction, unexplained hoarseness, dysphagia or unusually rapid onset of symptoms in the absence of marked weight gain.

☑ Patients suspected of serious underlying pathologies should be referred for urgent ear, nose and throat (ENT) assessment.

2.3 EXCLUDING OTHER CAUSES OF DAYTIME SLEEPINESS

OSAHS is one of the commonest medical causes of excessive daytime sleepiness, but clinicians should be aware that other conditions can produce similar symptoms, some of which are listed in table 2.

Table 2: *Potential causes of excessive daytime sleepiness in adults*

- fragmented sleep (quality of sleep)
- sleep deprivation (quantity of sleep)
- shift work
- depression
- narcolepsy
- hypothyroidism
- restless leg syndrome /periodic limb movement disorder
- drugs
  - sedatives
  - stimulants (caffeine, theophyllines, amphetamines)
  - β-blockers
  - selective serotonin reuptake inhibitors (SSRIs)
- idiopathic hypersomnolence
- excess alcohol
- neurological conditions
  - dystrophica myotonica
  - previous encephalitis
  - previous head injury
  - parkinsonism

3 Diagnosis

3.1 SUBJECTIVE ASSESSMENT OF SLEEPINESS

Many patients may initially present with non-specific symptoms such as irritability, personality change, work or family problems or poor concentration. This may result from poor sleep quality and a high index of suspicion of OSAHS is necessary to allow the diagnosis to be made. The following questions should be asked whenever a diagnosis of OSAHS is under consideration:

- Is this patient falling asleep regularly against their will?
- Is this patient often sleepy whilst driving?
- Is this patient experiencing difficulties at work because of excessive sleepiness?
- Is surgery for snoring being contemplated?

Various equations have been developed in an attempt to try to predict the likelihood of a patient having OSAHS from the history and examination findings but none have been successful or helpful in clinical practice.[35,36]

Patients with significant sleep apnoea may not realise that they have a problem as many of the features may be reported by a spouse or partner. Subjective assessment of sleepiness (by both patient and partner) is important as it is unlikely that patients will accept treatment unless they can perceive benefit with a reduction in subjective sleepiness or improvement in work performance. This benefit is related to the severity of pre-existing impairment.[37] 2+

The Epworth Sleepiness Scale (ESS; *see appendix 1*) is a validated method of assessing the likelihood of falling asleep in a variety of situations. The maximum score is 24.[38] The score can be used to clinically subdivide the patients into either the normal range (ESS <11), mild subjective daytime sleepiness (ESS = 11–14), moderate subjective daytime sleepiness (ESS = 15 –18) or severe subjective daytime sleepiness (ESS>18).[39] The Scale should be completed independently by both the patient and their partner as the patient may underestimate the severity of their sleepiness due to its insidious onset, or in order to hide concerns over driving ability. Although the correlation between ESS and OSAHS severity is relatively weak, the ESS is the best available tool to guide the clinician as to the patient's perception of his/her sleepiness.[40-42] 2+ 3

ESS can also be used to predict the likelihood of long term compliance with nasal CPAP *(see section 4.3.1)*.[37] 2+

C **All patients who have suspected sleep apnoea and their partners should complete an Epworth questionnaire to subjectively assess the degree of pretreatment sleepiness.**

3.2 REFERRAL

A subjective measure of daytime sleepiness (ESS >10) or sleepiness in dangerous situations, even with a normal ESS, in combination with symptoms associated with OSAHS (*see table 1*) should prompt referral to a sleep service. The main category of patient requiring urgent assessment to establish the presence of underlying sleep apnoea is one who has excessive daytime sleepiness, despite a normal time in bed at night, which may interfere with his/her driving ability or occupation.

Patients with severe obstructive sleep apnoea may decompensate with cor pulmonale or hypercapnic respiratory failure especially if there is coexisting chronic obstructive pulmonary disease (COPD).

☑ The combination of severe OSAHS and COPD is potentially dangerous. In such cases clinicians should consider urgent referral to a sleep centre.

☑ Patients with symptoms suggestive of OSAHS, who are sleepy whilst driving or working with machinery, or are employed in hazardous occupations should be considered for urgent referral to a sleep centre, as should those with ventilatory failure.

☑ OSAHS should be excluded in patients before they are considered for surgery for snoring.

Patients and their partners should routinely complete questionnaires about their sleep habits and key symptoms before the initial consultation with a specialist to aid overall assessment. A sample sleep habit questionnaire is available on the SIGN website – www.sign.ac.uk

Hypertension is associated with sleep apnoea and successful treatment of the apnoea may lead to a mean fall in diastolic blood pressure of 5 mm Hg in those with over twenty 4% desaturations/hr.[27,29] 1+

This may translate to a reduction in risk of death from both coronary artery disease and cerebrovascular disease. Hypertensive patients may not require urgent referral, as obstructive sleep apnoea may cause cardiovascular morbidity to develop over many years and not acutely.

3.3 OBJECTIVE ASSESSMENT OF SLEEPINESS

The Multiple Sleep Latency Test (MSLT) measures the time to fall asleep (using EEG criteria) in a darkened room on at least four separate occasions across the day following an instruction to fall asleep.[43-45] This period of time is known as sleep latency. Each challenge is terminated at 20 minutes or upon sleep onset, and an average time of seven minutes or less is regarded as evidence of pathological sleepiness. An alternative to the MSLT is the maintenance of wakefulness test (MWT) where the subject is instructed to stay awake, rather than to fall asleep.[46,47] This test is terminated at 40 minutes and a result of < 20 minutes is regarded as abnormal. A variant of the MWT (the OSLER test), using a behavioural assessment of sleep onset rather than one based on EEG, gives similar results but requires less technical input.[48] Although these tests measure sleepiness and its resolution with treatment, there is still a relatively poor correlation with other indices of OSAHS severity. In addition, it is unclear whether these objective tests predict functional impairment in real life, such as driving. At present they should therefore not generally be relied on to make individual clinical decisions as to fitness to drive, for example. These objective tests are not available outside large sleep centres.

3.4 PHYSICAL EXAMINATION

Examination by itself cannot allow an accurate diagnosis of OSAHS but it does help to exclude other causes for the patient's symptoms.

- weight and height should be documented at the first clinic visit. Continue to document weight at all subsequent visits. Approximately 50% of patients with OSAHS are obese (BMI >30 kg/m^2)
- measure neck circumference. Patients with OSAHS often have a neck circumference >17″ (43 cm)
- visually inspect for abnormally small mandible size
- assess nasal patency visually
- assess upper airway for obvious obstruction using indirect laryngoscopy if available
- inspect the tongue for macroglossia, assess dentition and presence or absence of teeth
- assess pharyngeal appearance (tonsillar size, uvular appearance, lumen size)
- measure BP (with an appropriately sized cuff)
- perform routine respiratory, cardiovascular and neurological examination to detect any coexisting disease (eg cor pulmonale, chest wall deformity, myopathies) as clinically indicated
- measure forced expiratory volume (FEV_1) and forced vital capacity (FVC) to detect any significant spirometric abnormalities
- the possibility of hypothyroidism, acromegaly and Marfan's syndrome as underlying causes for OSAHS should always be considered and thyroid function tests are often indicated

3.5 DIAGNOSTIC TOOLS

3.5.1 SLEEP STUDIES

The main purposes of a sleep study are to confirm the clinical suspicion of OSAHS and to assess its severity in order to guide the therapeutic choices to offer patients. To do this, sleep studies measure some aspect of ventilation and assess its possible compromise by upper airway obstruction, and any consequences on sleep quality. There are many ways to assess these aspects of OSAHS and the choice of sleep study equipment depends on many factors.

Studies of patterns of sleep, breathing and/or movements during sleep can be performed with decreasing degrees of complexity, varying from full polysomnography (PSG) eg 12-30 channels of various electrophysiological, breathing and movement signals, to one channel of information, eg oximetry. These may be recorded during part or all of a night's sleep. Limited sleep studies give less information than a full PSG and may only give indirect information about breathing patterns and often none about sleep duration or quality. Studies using oximetry alone are even more limited in the information they provide.

Simple or complex sleep studies can be performed either in hospital or at home depending on local and personal circumstances but the relative merits of these are unclear at present.

3.5.2 POLYSOMNOGRAPHY

Polysomnography records sleep and breathing patterns simultaneously. It is conventionally performed in a sleep centre with the aid of a technician, but portable home based versions are available.

PSG is carried out overnight at a sleep centre and is a relatively intrusive and costly study whose interpretation can be complex. A standard PSG typically consists of EEG, segmental (+/-) tibialis electromyogram, electro-oculogram, respiratory airflow (usually measured by oronasal flow monitors), thoraco-abdominal movement and oxygen saturation tracings (oximetry). Electrocardiogram (ECG) and body position are also frequently monitored, as is snoring.

Polysomnography usually requires about 30-60 minutes set up time before sleep and about 30 minutes detachment time in the morning. Staff must be available for at least ten hours overnight to perform and monitor this test. The study can then take up to four hours to analyse. The cost of PSG depends on the staffing levels employed, the number and complexity of studies performed and the cost of the equipment and premises.[49]

Although PSG is accepted in North America as the gold standard test for the diagnosis of sleep apnoea it has never been independently validated. Observational studies indicate that PSG may be useful in the diagnosis of sleep apnoea although there is night-to-night variation in PSG reproducibility. Different centres also use different thresholds in the diagnosis of sleep apnoea.[50-52] | 2^{++} 4

The clinical value of performing PSGs on all patients with daytime sleepiness has been questioned. In a prospective study of 200 patients with possible OSAHS, overnight PSG records were analysed to determine which signals contributed to diagnosis. Respiratory variables (thoraco-abdominal movement and oximetry) and the leg movement sensors were found to be helpful but neurophysiological signals did not contribute significantly to the diagnosis. [53] | 3

3.5.3 LIMITED SLEEP STUDIES

Theoretically, limited sleep studies may use any reduced combination of the full range of variables present with a full PSG. In practice, they usually incorporate some measurement of respiratory signals often with an indirect measure of arousal. Common combinations are airflow, thoraco-abdominal movement, oximetry and heart rate measurement with some adding snoring and indirect evidence of episodes of airflow obstruction. Many new machines are emerging and this field is changing rapidly.[49,50,54]

Whether PSG should be the gold standard with which to compare these simpler techniques is unclear. One recent trial has measured the improvement in symptoms following CPAP and correlated these with sleep study signals to identify the best predictors of this improvement.

In this study PSG was not the best predictor.[30] In this, and other studies, PSG derivatives (such as AHI, or EEG arousals) were no better at predicting improvement than the simpler indices (such as the number of oxygen desaturation dips or numbers of body movements).[30,55]

Three studies of hospital-based partial channel PSGs involving 213 patients with AHI >10 revealed a sensitivity range from 82-94% and a specificity ranging from 82-100% compared to full channel PSGs.[56-58]

Limited sleep studies can often be performed at home by the patient him/herself after adequate instruction from sleep technicians. A written instruction sheet should be provided. Studies performed in this way can save both the costs of the accommodation in the sleep centre and the attendant staff costs.[49] The cost of a typical home based limited study may be only approximately 20% of the cost of a hospital based PSG. If a technician has to go to the patient's home to attach the equipment this may be as expensive as a sleep centre PSG once travel time and costs are included. A disadvantage of this approach is the possible decrease in diagnostic certainty as limited studies do not allow assessment of sleep presence, quality or duration. Some respiratory events occurring during wakefulness will be scored and patients who fail to sleep will have negative results (although this is rare in OSAHS as these patients usually fall asleep very easily whatever the circumstances). There is also an inherent inability for home studies to diagnose conditions other than OSAHS with the technology currently available. 2+

There may be significant problems with equipment failure rates, night-to-night reproducibility, cost, compliance and reliability when used in the unattended home environment. Sensitivity ranges from 32-100% and specificity from 33-100% compared to full PSG in a sleep centre.[59] These figures are highly dependent on the equipment and definitions of events used. 1++

It has been suggested that patients diagnosed with OSAHS in limited sleep studies have poorer CPAP use thereafter[60] but this was not found in another study when the CPAP education process was carried out carefully.[49]

Provided these concerns are appreciated, limited studies may be useful, cost-effective and convenient for patients and can significantly speed up the investigation pathway. Hospital-based PSGs are the overnight investigation of choice for a minority of patients who cannot be investigated adequately at home or whose home study result does not fit with the clinical suspicion of the investigating doctor.

3.5.4 OXIMETRY

Oximetry alone is often used as the first screening tool for OSAHS due to the universal availability of cheap recording pulse oximeters. They are spectrophometric devices that detect and calculate the differential absorption of light by oxygenated and deoxygenated haemoglobin in blood to produce a measurement called the SpO_2. This is an assessment of the oxygen saturation of the arterial blood arriving at the fingertip or earlobe with each pulse beat.

Oximeters, however, have significant limitations which must be fully appreciated before they can be used alone to diagnose clinical problems or to influence patient management. They have an accuracy of $\pm$ 3% between individuals and all become less reliable if tissue perfusion is poor or if coloured nail varnish is used. They can give false negative results if used on young, thin patients who generally fail to desaturate during short apnoeic or hypopnoeic episodes as they maintain their lung volumes when lying flat and their baseline oxygen saturation tends to be on the plateau part of the oxygen dissociation curve as opposed to obese individuals who desaturate easily in a similar situation.

Oximeters are easy to use but they differ in probe design, sensitivity, sampling frequency, artefact rejection computation and averaging time. There can also be differences present in the techniques used to analyse the oximetry signals produced. Commonly employed methods include counting the number of oxygen desaturations (dips) per hour greater than an agreed value (often a 4% SpO_2 dip rate of more than 10 per hour) or alternatively the time spent during the study at less than an agreed SpO_2 level (often 90%). Thus it can be difficult to compare results from different centres using different machines or modes of analysis.

Oximeters also register oscillations in SpO_2 when the baseline SpO_2 is low, such as in COPD which may be confused with dips due to OSAHS. This is because at SpO_2 values below 93% the slope of the haemoglobin saturation curve is steep and normal physiological variations in ventilation and PaO_2 produce large changes in SpO_2. In the Cheyne-Stokes breathing of heart failure, oscillations in SpO_2 can be indistinguishable from those due to OSAHS.

Oximeters also measure heart rate and brief increases are an indirect marker of transient arousal from sleep. With each arousal the heart rate rises by about 6-10 beats per minute. Reviewing oximeter tracings with the accompanying pulse rate can provide information about sleep fragmentation.

Studies using oximetry alone to diagnose OSAHS have reached widely diverging conclusions. Some found oximetry useful[61-63] while others did not.[53,64] These differences may be explained by differences in the oximeters themselves, the analysis algorithm, and the diagnostic criteria used. Studies which required desaturation to occur before a hypopnoea could be diagnosed, found better correlations between AHI and desaturation frequency.[31] When compared with full PSG, oximetry alone showed a mean sensitivity of 87% (SE 4%, CI 36-100%) and mean specificity of 65% (SE 7%, CI 23-99%), which suggests that oximetry may be useful in selected patients with significant symptoms (AHI range 24-47).[59] A trained observer can diagnose OSAHS from positive oximetry traces, but false negatives occur in up to a third of patients with OSAHS.[53] The limitations of oximetry need to be clearly understood, namely, oximetry can positively diagnose OSAHS but cannot be used to exclude OSAHS. 2++ 4

☑ A normal oximetry tracing does not exclude OSAHS.

☑ The characteristic oximetric tracing in OSAHS is a sawtooth pattern, however care should be taken interpreting this pattern as it may also appear in patients with COPD or congestive heart failure.

☑ Automated analysis is available for many sleep systems but the validity of this should be checked by an experienced operator.

3.5.5 FLOW VOLUME LOOPS

Inspiratory flow volume loops have not proved to be useful in diagnosing OSAHS.[65,66]

3.5.6 RADIOLOGICAL IMAGING

Studying upper airway size or shape by computed tomography (CT), magnetic resonance imaging (MRI), or cephalometric radiology does not accurately differentiate patients with OSAHS from normal subjects and cannot be recommended in the routine assessment of patients with possible OSAHS.[67-70]

3.5.7 QUESTIONNAIRES

Three studies were identified comparing questionnaire sampling in OSAHS patients to full PSG. These reported a mean sensitivity and specificity of only 42% and 68% respectively. Questionnaires are useful in the initial assessment of the potential OSAHS patient but cannot, by themselves, make the diagnosis.[38,70,71]

3.5.8 NASENDOSCOPY UNDER SEDATION

Direct visualisation of the site of airway obstruction during sedation has been widely used by ENT surgeons to predict the occurrence and site of airway obstruction during sleep. There are no good prospective studies showing that these measures reflect what actually happens during spontaneous sleep and nasendoscopy cannot be recommended.

3.5.9 TRAINING

The appropriate training of personnel involved in the assessment of patients and their sleep studies is paramount. Although there is no direct evidence about who should perform such assessments, the research evidence upon which treatment effectiveness is based uses well-trained individuals in well-equipped centres.

Training in sleep studies is essential in the specialist registrar (SpR) programs for respiratory medicine, anaesthesia, and ENT surgery. Those intending to run a specific sleep service require a minimum of 12 months, and all respiratory trainees should spend three months, within a specialist referral centre.[72] A specialist centre would need to treat at least 100 new patients with CPAP a year to allow an SpR to see at least 25 cases during a three month attachment.

Training for the support staff, such as specialist technicians and nurses is equally important. At present, training is best acquired through an apprenticeship, although there are an increasing number of specialist courses designed for support staff.

3.6 SUMMARY OF DIAGNOSTIC STRATEGIES

Numerous diagnostic strategies incorporating the tests discussed in section 3.5 have been reported, although the published evidence for the individual tests is limited.

A combination of a positive limited sleep study in the context of clinical suspicion of sleep apnoea (excessive daytime sleepiness, Epworth subjective sleepiness score of more than 10, witnessed apnoeic episodes and loud snoring) allows a diagnosis of significant sleep apnoea with banding into mild, moderate or severe subdivisions. Such a limited sleep study should include one or more of the following: oximetry; thoraco-abdominal respiratory movement and airflow; recordings of snoring, heart rate or general video.[59] 2++

B **Limited sleep studies to assess respiratory events are an adequate first-line method of diagnostic assessment for OSAHS.**

☑ Individual sleep centres should examine the balance of benefits associated with using a specific sleep study against their resources, their geographical catchment area, the equipment available and the diagnostic algorithm used.

☑ Full PSG with EEG-based sleep staging is not necessary to diagnose sleep apnoea in most patients. It should be available in regional sleep centres for patients who have typical symptoms of excessive daytime somnolence but no objective evidence of obstructive sleep apnoea on limited testing.

☑ Oximetry studies cannot exclude OSAHS. Studies using oximetry alone may have a role in the initial assessment of OSAHS, however their significant limitations must be fully appreciated before using them to make diagnostic and therapeutic decisions.

☑ The specific technology used to make the diagnosis is less important than the level of experience and training available to interpret the results.

Other types of assessments using anthropometric measurements, ENT and dental assessments, radiological measurements and questionnaires do not offer sufficient sensitivity or specificity to be of value.

4 Treatment of OSAHS

4.1 INTRODUCTION

Deciding which of the various treatment options is most appropriate for the management of OSAHS depends on both the severity of the condition and the characteristics of an individual patient. The recommended treatment for moderate or severe OSHAS, continuous positive airway pressure (CPAP) is not always easy for patients to accept and the eventual decision to use it rests firmly with the patient.

Treatment options can be broadly divided into:

- behavioural interventions
- non-surgical options
- surgical options.

4.1.1 WHOM TO TREAT?

Current evidence from randomised controlled trials (RCTs) indicates that improvements with treatment can be found in symptomatic patients with AHI ≥15 or a 4% oxygen saturation dip rate at the level of >10/hour.[73-75] There is some evidence of benefit from the treatment of symptomatic individuals with AHI of 5-14, however further studies are required to confirm this *(see section 4.3.1)*.[33,34] These benefits are in daytime sleepiness, simulated driving performance, quality of life, blood pressure and mood. Treatment should thus be focussed on these benefits and not given in the hope of diminishing vascular risk in patients without daytime sleepiness or in asymptomatic individuals, for whom RCTs show no benefit.[76]

4.1.2 COMMUNICATION WITH PATIENTS

Following diagnosis, treatment options with their respective advantages and disadvantages should be discussed with the patient and their partner, fully involving them in the decision making process. This should be done using a multidisciplinary approach with written and video material where available. As patients have to commit to lifelong treatment they and their partners need to fully understand the implications of treatment. This is likely to aid compliance and treatment outcomes.[77]

4.2 BEHAVIOURAL INTERVENTIONS

Overweight patients should be advised to lose weight as weight reduction improves OSAHS symptoms and other excess weight related disorders. Unfortunately sustained weight reduction is rarely achieved whatever resources are applied.[78-80] In a limited proportion of cases, weight reduction of 10-15% has been associated with improvement in desaturation index and other markers of OSAHS. There is a poor correlation between the amount of weight lost and clinical response.[79,80] The role of gastro-intestinal surgery in the morbidly obese patient is under debate but may be considered in appropriate patients.[80] OSAHS can recur even after surgically induced weight loss.[81]

1++
2+
3

Patients who smoke should be advised to stop for general health reasons. While there is epidemiological evidence linking smoking to OSAHS,[4] there is no evidence that stopping smoking improves apnoeic symptoms, indeed any weight gain after quitting smoking may worsen OSAHS.

Alcohol should not be taken in the evenings and sedatives and sleeping tablets avoided as all of these decrease airway dilator function and worsen OSAHS.

C **Weight loss should be encouraged in all patients with obesity contributing to their OSAHS. Attempts at weight loss should not delay the initiation of further treatment. Weight loss should also be encouraged as an adjunct to CPAP or intra-oral devices as it may allow discontinuation of therapy.**

☑
- Patients who smoke should be advised to stop
- Alcohol and sedatives or sleeping tablets should be avoided
- Non-sleepy snorers should be discouraged from sleeping on their backs.

These measures may suffice in simple snorers or in those with very mild OSAHS and few symptoms but most patients with OSAHS need additional treatment.

A Cochrane review of lifestyle modifications for OSAHS identified no RCT evidence supporting their use and concluded that any decision to institute such interventions should not delay the institution of therapies of proven effectiveness, such as CPAP.[82] 2+

4.3 NON-SURGICAL INTERVENTIONS

4.3.1 CONTINUOUS POSITIVE AIRWAY PRESSURE

Continuous positive airway pressure (CPAP) functions as a pneumatic splint to maintain upper airway patency throughout all phases of sleep breathing. It operates by means of a flow generator which delivers pressure through air tubing to a nasal or face mask worn overnight. Most patients require lifelong treatment and therefore long term access to a CPAP machine.

Efficacy

CPAP has been established as the treatment of OSAHS with the firmest evidence base.[73,83] Randomised controlled trials show that CPAP improves subjective and objective sleepiness[84-87] 1++ cognitive function, vigilance, mood[73,88] and quality of life measures.[74,84,89] Objective improvements 1+ are found in symptomatic patients with AHI >15 or > ten 4% desaturations/hr.[74]

Meta-analysis[73] of several RCTs showed evidence of differential outcome with OSAHS severity, with significant improvements in subjective Epworth sleepiness score in two studies of patients with severe OSAHS[89,90] (mean –6 points, 95% CI –8 to –3, Effect Size 1.5 SDs) and non-significant changes in four studies of patients with mild OSAHS (-1, CI -1 to + 0.3).[34,85-87] Objective mean sleep latency, from the multiple sleep latency test *(see section 3.3),* showed similar significant improvements in sleep latency in a single trial of patients with severe OSAHS[90] (+ 2 mins, CI 1 to 3, ES 0.5 SDs) but not amongst trials of patients with mild OSAHS (0 mins, CI -1 to + 1).[85,87] A sham-controlled RCT of CPAP in asymptomatic individuals with raised AHI (but not diagnosed OSAHS) found no benefit for sleepiness from active CPAP.[76] In an RCT on patients with moderate to severe OSAHS using maintenance of wakefulness as the outcome measure, the change following treatment was from 23.4 to 30.4 mins (Effect Size 0.7 SDs).[74]

Randomised controlled trials of treatment for patients with polysomnographically mild OSAHS do not support reduced objective sleepiness after CPAP.[73,76] Within the population sampled in these studies are conflicting individual findings. Sampling biases vary across these RCTs with excessive sleepiness constituting an inclusion criterion in one study[86] and an exclusion criterion in another.[87] A single study of patients with mild OSAHS but considerable daytime sleepiness did find significant improvements in Epworth score with CPAP, in contrast to the others in less sleepy samples.[73,89]

Autotitrating CPAP machines adjust the CPAP pressure delivered overnight to prevent apnoeic episodes.[91,92] These devices can be used to establish the pressure required for later home use. They can be useful in a troubleshooting role to investigate problems with CPAP use, or when there may be pressure requirement changes following, for example, weight change. Theoretically, these machines should be more comfortable for long term use as the pressure will match changes in required pressure and in general be lower. This may produce less mask leakage and flow through the nose leading to fewer nasal side effects.[93,94] They are more expensive than conventional CPAP machines and, as yet, there is no convincing evidence that they produce better outcomes than conventional fixed pressure machines.

A **CPAP is the first choice therapy for patients with moderate or severe OSAHS that is sufficiently symptomatic to require intervention.**

Side effects and compliance

Major side effects of CPAP use (eg significant epistaxis, paranasal sinusitis) are rare, but minor side effects (rhinitis, nasal bridge sores, discomfort, claustrophobia, abdominal bloating, noise) are common. Intensive efforts can achieve CPAP uptake of up to 95%[37] and an average nightly use of three to five hours.[95,96] Nasal symptoms are usually due to mouth leaks causing high flows of cool air through the nose. Attempts should be made to reduce these using chin straps or full face masks. In a few patients nasal corticosteroids can be useful. A heated humidifier may help to improve comfort and compliance.[93]

The requirements of an adequate CPAP service have not been clearly established, and a variety of locally tailored systems may be satisfactory.[97] An essential element appears to be a dedicated CPAP nurse or technician responsible for mask fit, CPAP initiation and patient support. Few studies of CPAP have specified type or sequence of mask fitting, pressure set-up (titration) or pattern of humidification use, although all these elements are considered relevant to effectiveness and tolerability.[93] Published series are largely based on traditional single night CPAP titration studies. There is some evidence that split night studies with a diagnostic study for the first part of the night and a CPAP titration study for the remainder can be effective in many patients.[98]

The early pattern of use during the first few days and weeks (hours used per night) predicts long term use.[99,100] A threshold of less than two hours of CPAP use per night appears to distinguish those who are prepared to comply with therapy in the long term from those who withdraw. The likelihood of stopping treatment is far greater in those who at three months use CPAP for less than two hours per night.[37] There is some correlation between severity of symptoms and CPAP use, with those patients having more commanding symptoms more likely to tolerate the treatment Also, the lowest effective CPAP pressure contributes to better compliance.[96] Higher CPAP pressures are associated with a greater reduction in daytime sleepiness.[37] 2^{+} 2^{++}

C **Persistent low CPAP use (less than two hours per night) over six months, following efforts to improve patient comfort, should lead to a review of treatment.**

☑ CPAP therapy should not be abandoned without:

- the attention of a trained CPAP nurse / technician
- a titration study / use of autotitrating CPAP to troubleshoot problems
- the use of heated humidification.

4.3.2 ECONOMIC ANALYSIS OF CPAP USAGE IN OSAHS

Patients with undiagnosed OSAHS are heavy users of the healthcare system. Expenditure on undiagnosed patients is approximately twice that of age and gender matched controls. This difference extends back over 10 years prior to the diagnosis of OSAHS being made.[101]

Treatment with CPAP reduces these costs with evidence of decreased hospitalisation due to cardiovascular and pulmonary disease.[102] Hospitalisation and other costs associated with road traffic accidents are also reduced in those using CPAP therapy.[103-105] Overall mean hospitalisation days per year decreased with CPAP use.[106]

There is a need for more studies on the cost effectiveness of CPAP and other treatments for OSAHS. One study carried out in 1994 has estimated that CPAP resulted in an average gain of 5.4 Quality Adjusted Life Years (QALYs) at a cost of Canadian $3400 to $9800 / QALY gained, equivalent to £1500-£4400.[107] A British study suggests a cost of £3200 / QALY gained over a five year period.[108] These do not include any benefits that would be expected to accrue from a decrease in BP or road traffic accident decrease with CPAP treatment. These studies indicate a cost-effectiveness ratio in line with other routinely funded procedures within the NHS, but more studies are needed to confirm this. (*See Annex 2*)

4.3.3 BI-LEVEL POSITIVE AIRWAY PRESSURE

These machines allow independent adjustment of inspiratory and expiratory pressures rather than having a fixed pressure as with CPAP. A well-designed randomised study found no advantage for bi-level support over CPAP in straightforward OSAHS.[109] This mode of non-invasive ventilation may be more appropriate for patients with ventilatory failure.[110] 1+ 4

B **Bi-level ventilation should not be used routinely in OSAHS but should be reserved for patients with ventilatory failure.**

4.3.4 INTRA-ORAL DEVICES

Intra-oral devices (IODs) are a range of appliances designed to alter upper airway patency. Several techniques have been employed, but mandibular advancement has gained most acceptance. Most mechanisms have a similar action in producing anterior displacement of the mandible, thus increasing upper airway diameter in some appliances. The degree of mandibular protrusion can be specified and subsequently adjusted.

Effectiveness

Recent randomised controlled trials have evaluated the effectiveness of intra-oral devices (IODs) against no treatment[111,112] or placebo IOD[113-117] with all trials incorporating crossover designs. IODs have also been assessed against CPAP[83,118-122] and against uvulopalatopharyngoplasty.[123]

The patients recruited into these trials comprised snorers without desaturation[114,124] patients with mild to moderate, symptomatic OSAHS[112,113,115,116] or mixed snorers and apnoeic patients.[111] These trials have examined outcomes including symptom ratings,[113,114,116] daytime sleepiness[113,114,116] and polysomnography[111,113,115,116] but many trials have excluded participants who were intolerant of IODs.[111,113,115]

The effect sizes of changes in apnoea/hypopnoea index with IOD compared to no treatment or a placebo device in patients with mild to moderate OSAHS have varied from small (0.3 SD)[112] to very large (1.6 SD),[115] as have differences in snoring frequency (0.3 SD[111] to 1.1 SD[115]). In these studies mean AHI following therapy using real IODs ranged from 12[116] to 23[113] per hour, suggesting that IODs failed to normalise sleep disordered breathing variables in these subjects. 1- 1+

Symptom and sleepiness ratings from this group of trials also favour IOD over no IOD or a placebo IOD. In snorers lacking significant desaturation a real IOD produced large effect sizes (>1 SD) in partners' ratings of loudness and frequency of snoring over a placebo device but smaller benefits for patient ratings of Epworth sleepiness (0.2 SD) and waking unrefreshed (0.5 SD).[114] In patients with mild to moderate OSAHS, the same IOD produced better mean scores on these ratings than the placebo IOD, but these remained non-significant (Epworth difference of 1 point (0.2 SD), snoring loudness 0.7 SD).[113] In another placebo-controlled trial of IOD in OSAHS patients, sleep latency on the MSLT was significantly improved by one minute on real IOD (effect size 0.3 SD) and Epworth score significantly decreased by two points (0.4 SD).[116] 1+

Randomised controlled trials comparing IODs to CPAP treatment are all crossover studies conducted in patients with symptomatic OSAHS and have assessed outcomes of polysomnography,[118-121] sleepiness[119-121] and health status and cognitive function.[120,121] Most have experienced some subject attrition due to refusal to crossover and some have analysed only patients tolerant of one or both treatments.[117,118,120]

For polysomnographic outcomes, the mean values for AHI, oxygen saturation and sleep fragmentation favour CPAP in all studies. Not all studies have reported statistical tests of IOD versus CPAP.[118,119] Of those that have, two demonstrated a significantly greater reduction in AHI whilst using CPAP than with IODs.[120,121] The intertreatment effect sizes for AHI range from moderate (0.4 SDs)[120,121] to large (0.9 SDs).[118] Mean AHI following CPAP therapy ranged from 3 - 8 per hour, and following IOD therapy ranged from 8 - 15 per hour.[118-121] The highest AHI values for both treatments were from an intention-to-treat study in which treatment was used as long as tolerable during polysomnography.[120] 1++ 1+

Baseline Epworth scores before treatment averaged 11,14 and 13 in the three studies evaluating this.[119-121] Post-treatment Epworth sleepiness ratings on IOD and CPAP were not statistically different in two studies[119,121] and significantly better by 5 points (0.8 SDs) with CPAP in the third which had most severe baseline subjective sleepiness.[120] Objective sleep latency from a maintenance of wakefulness test and four objective cognitive performance scores from this third study were not different between treatments, but all three SF-36 health status summary scores (effect sizes 0.2 - 0.5 SDs) were significantly better on CPAP. Satisfaction with treatment was significantly higher for IOD in two studies[118,119] and higher for CPAP in another.[120] For long term treatment, IOD was preferred by 17 of 25 patients,[118] 13 of 20,[119] 19 of 48[120] and 17 of 21.[121] Thus 66 of 114 patients in these trials preferred IOD treatment, despite worse sleep-disordered breathing on this treatment. 1++ 1+

A meta-analysis of patients' treatment preference (CPAP and IODs) in three crossover studies in mild to moderate OSAHS showed a significant patient preference for IODs (OR 9.5, 95% CI 4 to 21), despite lesser nocturnal efficacy for breathing pauses (-7 per hr, 95% CI -10 to –5).[83] This was not confirmed in a later study.[55] Patients' preference for IODs is important, but it is not known if this means that they feel symptomatically better when using IODs or whether they find the concept of an unobtrusive intra-oral device preferable to using an obtrusive CPAP device. 1+

One group has compared the effectiveness of IOD against UPPP in a parallel-group, longitudinal follow-up study, with the latest report at four years post-randomisation.[123] In this, 72 of 95 patients with mild to moderate OSAHS have returned for polysomnography, which showed large effect sizes (>1.0 SDs) significantly favouring IOD over UPPP for improvements in AHI and desaturation index, but no significant difference in snoring duration between treatments. 1+

It is unclear whether CPAP or IODs have the greater cost effectiveness and the answer may vary with OSAHS severity. The basic cost of many IODs is less than the cost of a CPAP machine. Some adjustable IODs are more expensive than CPAP, especially when the cost of multiple dental visits to adjust the IOD are included. (*See Annex 2*) The role of IODs as a first line management strategy for subjects with mild OSAHS has been demonstrated. However, their effectiveness in managing more severe OSAHS is limited by the present lack of identifiable prognostic indicators for their success.

A **Intra-oral devices are an appropriate therapy for snorers and for patients with mild OSAHS with normal daytime alertness.**

B **Intra-oral devices are an appropriate alternative therapy for patients who are unable to tolerate CPAP.**

Side effects and compliance

Hypersalivation and teeth/gum discomfort are common early side effects but usually decline if patients are able to persevere with IOD use.[125] Recurrent dislodgement of the device during sleep and temporomandibular pain are longer term problems. Side effects abate with appliance withdrawal. Early perseveration of use inside the first three months predicts long term success. These devices often require adjustment and may need to be replaced.[126] 3

The reasons for IOD failure, even when worn correctly, are complex and mainly relate to different combinations of skeletal, soft tissue and functional factors that produce upper airway obstruction in each patient. Failure to control the OSAHS will lead to poor symptom control and therefore follow up of these patients is important, particularly as regards a return to driving.[119,127] 1++ 3

D **The use of intra oral devices should be monitored following initiation of therapy to allow device adjustment and assessment of OSAHS control and symptoms.**

4.4 PHARMACOLOGICAL TREATMENTS

The evidence base to support pharmacological treatment as an effective therapeutic option is small. The main systematic review of pharmacotherapy concluded that no medication demonstrated a consistent response.[128,129]

Drugs which suppress rapid eye movement (eg protryptyline, acetozolamide and progesterone) do not show clinical benefit in treating OSAHS in controlled trials. Trials of theophylline show an inconsistent effect with a tendency to disrupt sleep in patients with OSAHS, but improve Cheyne- Stokes respiration. Hypnotics, such as benzodiazepines, may worsen OSAHS.[128] 1++ 1+

There is some evidence to suggest that the addition of alerting drugs, such as modafanil, may have a small beneficial effect on sleepiness in some patients who remain sleepy despite good CPAP compliance. However, they may decrease CPAP use and longer term studies of their value and risks are needed.[130] There is no evidence to suggest that they could be used as an alternative to CPAP and they are not a substitute for careful attention to improving CPAP comfort and efficacy.

A **Pharmacological therapy should not be used as first line therapy for OSAHS.**

5 Surgical interventions

5.1 INTRODUCTION

Many different surgical approaches have been used in the treatment of OSAHS, all with the intention of increasing pharyngeal calibre and reducing pharyngeal resistance during sleep.

5.1.1 METHODOLOGICAL DIFFICULTIES

There are no RCTs comparing surgical treatments for OSAHS. Unfortunately, because of large placebo and regression to the mean effects in OSAHS, uncontrolled trials are rarely convincing. Regression to the mean occurs when there is fluctuation in the objective and subjective severity of a condition with time. On the whole, patients will present, and be more likely to be selected for treatment, when they are at their worst. Therefore, on average, they will be better the next time they are studied, regardless of any intervention. Attempts to control for this can be made by having two or three pre-intervention studies, but even this is not as good as randomised, placebo-controlled trials.

An alternative experimental design, which may partially control for regression to the mean, is to show different response rates to surgery depending on some preoperatively determined criteria. For example, the result of a preoperative anatomical assessment of patients with OSAHS could be used to subdivide a group of patients, who are otherwise similar. If the surgical outcome was significantly better in one group compared to the other, then this latter group is a form of control. Such studies have produced differing results, but unfortunately such preoperative subdivisions tend to produce groups unmatched for some other measures, such as OSAHS severity or body weight.

5.2 UVULOPALATOPHARYNGOPLASTY (UPPP)

5.2.1 EFFECTIVENESS

There have been two systematic reviews that concluded there was no RCT evidence supporting the use of UPPP in OSAHS. Uncontrolled case series suggest, at best, a 50% improvement in 50% of patients.[131,132] The effects on objective measures of OSAHS were poor and largely unpredictable, although statistically significant overall. A meta-analysis of laser-assisted uvulopalatopharyngoplasty (LAUP) also concluded that LAUP and related procedures should not be used for any severity of OSAHS.[133] 2++

B **Use of UPPP or LAUP for the treatment of OSAHS is not recommended.**

Tonsillectomy is usually carried out in conjunction with conventional UPPP, but may in its own right improve OSAHS. Case series support this conclusion but no RCT data exist.[134,135] 3

☑ The presence of large tonsils in a patient with diagnosed OSAHS should prompt referral to an ENT surgeon for consideration of tonsillectomy.

Three studies with some potential for controlling for placebo, or regression to the mean, effects were identified.[136-138] One study identified 90 patients having UPPP of whom 44 had complete preoperative assessment of pharyngeal collapsibility during an awake Mueller manoeuvre (generation of subatmospheric intra-pharyngeal pressure) and 31 patients were monitored postoperatively.[136] The postoperative improvement in AHI was better in the group with collapse limited to the palatal area, compared to those with more extensive collapse. The division of the patients in this way produced groups with differing OSAHS severity and, by chance, a very different time to the follow up assessment. It is impossible to confidently ascribe benefits to the surgery itself. 2+

Another study compared patients randomly assigned to receive UPPP or conservative management, which should have removed regression to the mean, but not placebo, effects.[137] There was no significant difference in the main objective measures of OSAHS severity, although there was improvement in the subjective assessment of symptoms. Sub-analysis of oximetry indices of severity showed more improvement in the surgically treated group (number with a normal SpO_2 dip rate at one year, 14% versus 45%). Three of 14 patients in the control group were operated on despite their randomisation to conservative management.

The third study randomised 95 patients with symptomatic OSAHS to either IODs or UPPP.[138] The follow up analysis was not on an intention to treat basis and the dental result may be biased in favour of a bigger effect. Overall dental devices were more effective than UPPP. The effect sizes at 12 months were small for both dental and UPPP treatments, (0.14 and 0.1 respectively based on oxygen desaturation index, and 0.33 compared with 0.24 based on AHI).

5.2.2 SIDE EFFECTS

Two studies have described, and estimated the immediate and long term prevalence of, the morbidity and mortality following UPPP.[139,140] This issue is of interest to anaesthetists as deaths have been reported in the perioperative period. The deaths have been assumed to be due to worsening of upper airway obstruction and depression of ventilatory drive. One study reported that most problems occur in patients with comorbidity, such as a BMI > 35.[141] It appears prudent to provide nasal CPAP postoperatively, or even a temporary tracheostomy. Severe postoperative pain occurs and changes in voice and nasal regurgitation of food following UPPP are also possible.[142]

5.2.3 SNORING

UPPP and related procedures are also used extensively in the treatment of snoring.[143-146] Significant OSAHS is present in over 30% of snorers presenting to a specialist clinic, even when not overtly sleepy.[146] In order not to inadvertently operate on patients with OSAHS, sleep study assessment to exclude this diagnosis is an advisable part of any preoperative evaluation. UPPP is associated with the standard risks of surgical morbidity and is not an effective treatment for OSAHS. In addition, UPPP has an adverse effect on the patient's subsequent ability to use nasal CPAP.[144] 3

☑ OSAHS should be excluded in patients before they are considered for surgery for snoring.

D **Patients being offered palatal surgery should be informed of the risk of difficulty with CPAP use if they later develop OSAHS.**

5.3 TRACHEOSTOMY

Tracheostomy was the first surgical treatment for OSAHS and bypasses the obstruction completely. There have been no controlled trials to assess longer term outcomes, particularly self-assessed health benefits, which are important given the potential complications of a tracheostomy.

☑ Tracheostomy should only be considered when all else fails in carefully selected individuals.

5.4 OTHER SURGICAL TECHNIQUES

5.4.1 MANDIBULAR ADVANCEMENT

One controlled trial has shown that permanent mandibular and maxillary advancement considerably reduces OSAHS severity and improves symptoms in patients followed up for two years.[147] There are no RCTs and only limited long term follow up data available, and the treatment remains experimental.

5.4.2 SUPRAHYOID TENSING

A randomised study of a surgical procedure to tense the suprahyoid muscles (hyoid suspension) was halted due to worsening of sleep study indices, despite apparent symptomatic improvement.[148]

5.4.3 BARIATRIC (WEIGHT REDUCING) SURGERY

Weight is known to influence the severity of OSAHS and weight loss is likely to be an effective treatment for OSAHS in some patients. Bariatric surgery to provoke significant weight loss has been used to treat OSAHS, assessed in case series.[149] In the absence of a controlled trial, the relative benefits and disadvantages cannot be assessed. This area urgently needs evaluation.

5.4.4 NASAL SURGERY

Nasal surgery may play a role in improving compliance with nasal CPAP by reducing nasal resistance and allowing a reduction in the pressure required.[150,151] There is no evidence that it produces an improvement in OSAHS symptoms. 2+ 4

☑ Alternative surgical approaches to OSAHS are experimental and should not be used outside the context of an RCT.

5.5 ANAESTHESIA

Obstructive sleep apnoea presents specific problems for the anaesthetist and regional anaesthetic techniques should be used where possible. Even following minor surgery, the patient suffering from severe OSAHS is likely to require care in a high dependency or intensive care unit (HDU or ICU) postoperatively. Premedication sedative drugs are not advised and postoperative opiate analgesia should be titrated carefully to avoid increasing the frequency of apnoeic episodes and precipitating oxygen desaturation. Obesity adds to the anaesthetic risk as do other associated conditions such as hypertension and cor pulmonale.[152] 4

There is an increase in intubation difficulty even in mild to moderate OSAHS and anaesthetists should be aware of this possibility in all OSAHS patients.[153]

☑ The effect of anaesthesia during surgery may increase the severity of the apnoea postoperatively. When a patient is being treated by CPAP preoperatively this should be continued immediately following surgery.

☑ All patients with OSAHS should be monitored with oximetry postoperatively and further management decided on an individual basis.

6 Effects of treatment on driving and quality of life

6.1 INTRODUCTION

The consequences of OSAHS vary from annoying to life threatening and include excessive daytime sleepiness, (eg falling asleep at work or when on the telephone or whilst driving), depression, irritability, marital disharmony, sexual dysfunction and learning and memory difficulties.

6.2 DRIVER AND VEHICLE LICENSING AGENCY (DVLA) RECOMMENDATIONS

Untreated sleep apnoea often causes sleepiness which is dangerous whilst driving and can lead to an increased likelihood of having an accident. Patients should be informed that they must not drive if they feel sleepy, even if the diagnosis of OSAHS is only suspected, and that falling asleep at the wheel is a criminal offence and can potentially lead to a prison sentence. When a person is diagnosed as suffering from sleep apnoea they must be told verbally and in writing that they should inform the Driver and Vehicle Licensing Agency (DVLA) of the diagnosis. This information must also be given to the GP. There should be no problem about keeping a licence provided that patients comply with an effective treatment regimen.

After diagnosis the patient should also inform their insurance company.

The DVLA recommends:[154]

Group 1 Licences (normal car licence)

Driving must cease if continuing to cause excessive awake time sleepiness. Driving will be permitted when satisfactory control of symptoms achieved.

Group 2 Licences (HGV, PSV)

Driving must cease if continuing to cause excessive awake time sleepiness. Driving will be permitted when satisfactory control of symptoms achieved and confirmed *by specialist opinion*.

6.3 THE EFFECT OF CPAP ON DRIVING

The relationship between OSAHS, excessive daytime sleepiness and road traffic accidents has been shown in driving simulator tests and in accident surveys.[75,155] Epidemiological studies have suggested a particularly high prevalence of OSAHS in truck drivers.[156,157]

Sleepiness is estimated to cause 20% of accidents on motorways, and is associated with both increased rates and severity of accidents.[11] A systematic review has documented a substantial base of case-control and cohort studies suggesting that driving performance is impaired, and accident rates increase with sleepiness and OSAHS.[88] 2++ 3

Meta-analyses of daytime function identify no RCTs of treatment which affect real-world accident rates.[73,83] Future placebo-controlled RCTs of this outcome are not anticipated, not least because of ethical considerations. Laboratory studies of objective driving simulator performance offer surrogate evidence for this area.

A recent sham-controlled RCT in patients with severe OSAHS has demonstrated cross-validating improvements in both sleepiness and driving simulator performance following CPAP.[75] Active CPAP treatment produced significant improvement in steering accuracy, maintenance of performance over time and reaction times.[158] 1+ 2+

Attention-biased cognitive performance tests (SteerClear, Trailmaking B, PASAT, Digit symbol) may represent a further surrogate measure relevant to driving. While none have the face validity of a driving simulator, scores are thought to measure performance skills common to real-world driving, including response speed and accuracy, vigilance and visuomotor coordination. In one systematic review, results from the extended vigilance task SteerClear showed no significant improvement following treatment with CPAP in patients with mild OSAHS, although a significant improvement in a single study in patients with severe OSAHS (-6 errors, CI -13 to –1).[73] The visuomotor trailmaking B time test did not distinguish individual improvement in the three studies of CPAP treatment for patients with mild OSAHS or the single study for patients with severe OSAHS, but a significant improvement (-4 secs, CI -8 to –1) in a pooled analysis of all four studies. Digit symbol substitution (short-term coding speed) was unchanged by CPAP in mild, severe or pooled RCT patients. Rapid mental arithmetic (PASAT) was improved by CPAP within two mild trials, one severe trial and a pooled analysis (+ 2 sums, CI 1 to 3).[73] 1+

A **CPAP should be considered for the improvement of driving ability in patients with severe OSAHS as it reduces daytime sleepiness.**

☑ CPAP treatment should be prioritised to sleepy drivers and occupational drivers with OSAHS given the public health consequences of untreated OSAHS, sleepiness and accidents.

6.4 EFFECTS OF CPAP ON QUALITY OF LIFE

Other consequences of sleepiness may impact on patients' functional level across a range of work, home and social environments. These have been assessed using a variety of instruments.[83,73]

6.4.1 OSAHS-SPECIFIC SYMPTOM SCALES

One systematic review of RCTs of treatment with CPAP reports significant improvements in symptom scores in polysomnographically mild and severe samples.[73] The varying symptom scales had broadly overlapping, OSAHS-specific content, covering common nocturnal and daytime symptoms. While significant score improvements were found across all three trials of mild OSAHS and both of the trials of severe OSAHS, p-values were lower in the more severe samples ($p<0.001$). 1++

6.4.2 GENERIC WELL-BEING SCALES

Assorted generic scales (NHP, SF-36 UMACL) containing energy or vitality subscales (with enhanced disease-specific sensitivity) were used to estimate aspects of subjective function and health status in several RCTs of mild and severe OSAHS.[85,86,89] Energy/vitality scores were highly significantly improved in the pooled analysis of all studies ($p < 0.0001$). After clustering by polysomnographic severity, studies indicated that patients with severe OSAHS showed individually significant effects from CPAP.[89,104] However, two RCTs in patients with mild OSAHS showed no individually significant improvement in energy/vitality scores.[34,86] Quality of life scales with no dimension measuring sleepiness or fatigue (eg the EuroQol) fail to demonstrate any response to CPAP.[74] 1+

Another area of harm associated with OSAHS, and with chronic disease generally, is impairment to psychological well-being. In meta-analysis, combined Hospital Anxiety and Depression Scores (HADS) and Beck Depression Inventory scores were highly significantly improved overall, across four RCTs of mild and severe OSAHS following treatment.[73] There was heterogeneity, with one of three trials of mild OSAHS showing no significant improvement in depression rating following CPAP therapy. Meta-analysis of HADS anxiety scores (3 trials) showed no benefit from CPAP either individually or by pooling the RCTs. The least disease-specific outcome included in the meta-analysis was the general health subscore from the SF-36, reported in two RCTs of mild OSAHS and one RCT of severe OSAHS. Although the pooled meta-analysis found an overall improvement in SF-36 well-being scores following CPAP therapy, individual significance was observed only in the single trial involving patients with severe OSAHS. 1++

An additional outcome relevant to quality of life was treatment preference in placebo-controlled RCTs of CPAP. The Cochrane analysis reported pooled significant preference for CPAP over an oral placebo for two studies of mild and mixed-severity samples (OR 0.4, 0.2 to 0.8).[83] A later meta-analysis of treatment preference sampled an overlapping set of three RCTs, in patients with mild OSAHS. These pooled studies showed no significant preference for CPAP (95%CI 40-65%).[73] 1+ 1++

6.5 THE EFFECT OF INTRA-ORAL DEVICES ON SLEEPINESS, DRIVING AND QUALITY OF LIFE

Uncontrolled improvements in subjective sleepiness scores (baseline vs. IOD) are frequently reported across studies. Ratings relevant to driving (sleepiness while driving) and functional status (work performance, interference with daily tasks) also improve following treatment with an IOD.[117-119] 1+

6.6 THE EFFECT OF SURGERY ON SLEEPINESS, DRIVING AND QUALITY OF LIFE

The most widely assessed surgical procedure (UPPP) did not appear to produce reliable, long term clinical benefits, and daytime outcomes were scarce and unvalidated. Other surgical techniques may show greater promise, but good quality evidence of benefit is lacking.[132]

Further and more robust evidence for specific surgical techniques is required, and clinical recommendations for surgical treatment for OSAHS cannot be made until this is achieved.

☑ Pharyngeal surgery for OSAHS has no proven benefit and should only be undertaken as part of a randomised controlled trial.

7 Information for discussion with patients and carers

The following sample information sheet can be used in discussion with patients to highlight issues of particular importance. It is intended as a guide only and should not be used to plan treatment or to replace the important consultations that should be held between patients and healthcare professionals.

7.1 NOTES FOR DISCUSSION WITH PATIENTS AND CARERS

What is Sleep Apnoea/Hypopnoea Syndrome?

People who suffer from obstructive sleep apnoea/hypopnoea syndrome (OSAHS) breathe shallowly or stop breathing for short periods while sleeping. This can happen many times during the night. It results in poor sleep leading to excessive sleepiness during the day. Because these events occur during sleep, a person suffering from OSAHS is often the last one to know what is happening.

In deep sleep, the muscles of the throat relax. Normally this doesn't cause any problems with breathing. In OSAHS, complete relaxation of the throat muscles causes blockage of the upper airway at the back of the tongue. Normal breathing then slows or stops completely. Such an episode is called an **apnoea**. During an apnoea, people with OSAHS make constant efforts to breathe against their blocked airway until the blood oxygen level begins to fall. The brain then needs to arouse the person from deep relaxed sleep so that the muscle tone returns, the upper airway then opens and breathing begins again. Unfortunately, when a person with OSAHS falls back into deep sleep, the muscles relax once more and the cycle repeats itself again and again overnight.

In OSAHS, the apnoeas can last for several seconds and in severe cases the cycle of apnoeas and broken sleep is repeated hundreds of times per night. Most sufferers are unaware of their disrupted sleep but awaken unrefreshed, feeling sleepy and in need of further refreshing sleep.

Who gets OSAHS?

Whilst OSAHS is more common in overweight middle-aged males who snore, it can also affect females, although female hormones and a difference in throat structures may protect women until the menopause. Narrowing of the back of the throat and the upper airway can also contribute to the risk of getting OSAHS, even in people who are not overweight or middle-aged. In such people a small jaw, enlarged tongue, big tonsils and big soft palate help to block the upper airway in deep sleep, making OSAHS more likely to occur. Several of these problems can be present in any person at the same time.

The use of alcohol, sleeping tablets and tranquillisers prior to sleep relaxes the upper airway muscles and make OSAHS worse. Alcohol can also reduce the brain's response to an apnoea which in turn leads to longer and more severe apnoeas in people who would otherwise have only mild OSAHS and who would otherwise only snore.

What are the symptoms of OSAHS?

Most people with OSAHS snore loudly and breathing during sleep may be laboured and noisy. Sleeping partners may report multiple apnoeas which often end in deep gasping and loud snorting. Sufferers may report waking for short periods after struggling for breath. Symptoms are often worse when lying on the back in deepest sleep.

Although a person with OSAHS may not be aware of the many arousals from deep sleep, they suffer from poor quality sleep in spite of long periods of time spent in bed. Such people wake feeling that they haven't had a full refreshing night's sleep. They report difficulty maintaining concentration during the day, have a poor memory, and suffer from excessive daytime sleepiness.

At first an OSAHS sufferer may be sleepy only when seated and relaxed, eg watching TV, but eventually sleepiness becomes so severe that car accidents and accidents in the workplace occur. Other symptoms of OSAHS include morning headache, nocturia, depression, short temper, grumpiness, personality change, and impotence in males, leading to loss of interest in sex.

What are the consequences of untreated OSAHS?

The most serious potential consequences of untreated OSAHS are road traffic accidents and accidents at work because of sleepiness. Untreated OSAHS is associated with a sixfold increase in risk of such accidents. Patients may also experience difficulties with concentration due to tiredness, increased irritability and depression. There is evidence that patients with OSAHS have an increased risk of high blood pressure and may have a slightly increased risk of angina, heart attacks and strokes. Because OSAHS significantly increases the risk of road traffic accidents patients must not drive if experiencing excessive daytime sleepiness. Patients must inform the DVLA in Swansea following a diagnosis of the condition. In most cases, the DVLA are happy to allow car drivers to continue driving once they are established on a successful therapy.

How is OSAHS assessed?

When a person is suspected to have OSAHS, their doctor will ask questions about waking and sleeping habits and will make a physical examination. Reports from the sleeping partner or household member about any apnoeas are extremely helpful.

Referral to a sleep disorders centre for an overnight sleep study will probably be required to confirm the diagnosis of OSAHS and to allow its severity to be measured.

During a sleep study, sleep quality and breathing are measured overnight by a computer while the person sleeps. Procedures in different hospitals vary but small coin-sized electrodes may be taped to special points on the scalp, face, chest and legs. Chest and stomach wall movements are also measured and a special sensor placed on the upper lip measures airflow. The oxygen level in the blood is assessed by a device placed on the finger or the ear-lobe. None of these procedures are uncomfortable or painful.

How is OSAHS treated?

The simplest treatment is to lose weight. This is best done by cutting down on all foods, especially fatty foods, sweet things and alcohol. Alcohol within six hours of bedtime should be avoided as it contributes to OSAHS symptoms. If these measures are not enough, the best form of treatment is continuous positive airway pressure (CPAP) therapy in which a gentle flow of air is applied through the nose at night keeping the pressure in the throat above atmospheric pressure and stopping the throat narrowing to prevent breathing pauses and snoring.

Other forms of treatment include gumshield-like devices (mandibular repositioning devices) which attempt to keep the airway clear by moving the jaw forward. Surgery to remove excess tissue from the throat is another option, but it is not recommended. Both of these alternatives are less effective than CPAP and not appropriate for all patients.

7.2 RESOURCES FOR PATIENTS

The Scottish Association for Sleep Apnoea
18 Albert Avenue, Grangemouth, FK3 9AT
Tel: 01324 471 879. Fax: 01324 471 879
E-mail: smtprice@bigfoot.com

SATA (The Sleep Apnoea Trust),
7 Bailey Close, High Wycombe, HP13 6QA
Tel: 01494 527772
www.sleep-apnoea-trust.org, Sleepnet: www.sleepnet.com

American Sleep Apnoea Association:
www.sleepapnea.org/

The Sleep Medicine Home Page:
www.users.cloud9.net/~thorpy/

8 Development of the guideline

8.1 INTRODUCTION

SIGN is a collaborative network of clinicians and other health care professionals, funded by NHS Quality Improvement Scotland. SIGN guidelines are developed by multidisciplinary groups of practicing clinicians using a standard methodology, based on a systematic review of the evidence. Further details about SIGN and the guideline development methodology are contained in "SIGN 50; A Guideline developer's handbook" available at **www.sign.ac.uk**

8.2 THE GUIDELINE DEVELOPMENT GROUP

Dr Tom Mackay *(Chairman)*	*Consultant Respiratory Physician, Sleep Centre, Edinburgh Royal Infirmary*
Dr Steve Banham	*Consultant Respiratory Physician,Glasgow Royal Infirmary*
Miss Alison Beattie	*Senior Dietitian, Department of Nutrition and Dietetics, Perth Royal Infirmary*
Dr Roger Carter	*Principal Scientist, Glasgow Royal Infirmary*
Dr Alistair Dorward	*Consultant Physician, Royal Alexandra Hospital, Paisley*
Dr Heather Engelman	*Senior Research Fellow, Sleep Centre, Edinburgh Royal Infirmary*
Professor Colin Espie	*Clinical Psychologist, Glasgow University*
Mrs Jean Gall	*Chair, Scottish Association for Sleep Apnoea*
Mr Robin Harbour	*Quality and Information Director, SIGN*
Sister Carol Hoy	*Senior Specialist Sleep Nurse, Sleep Centre, Edinburgh Royal Infirmary*
Dr Peter Hutchison	*General Practitioner, Dumfries*
Dr William Kinnear	*Consultant Physician, University Hospital, Nottingham*
Mr Jim McDonald	*Consultant Orthodontist, Edinburgh*
Dr Moray Nairn	*Programme Manager, SIGN*
Dr Janet Pollock	*Consultant Anaesthetist, Southern General Hospital, Glasgow*
Dr Michal Scullion	*General Practitioner, Alexandria*
Dr Robin Smith	*Consultant Respiratory Physician, Ninewells Hospital, Dundee*
Professor John Stradling	*Consultant Respiratory Physician, Churchill Hospital, Oxford*
Mr Paul White	*Consultant Otolaryngologist, Ninewells Hospital, Dundee*

The membership of the guideline development group was confirmed following consultation with the member organisations of SIGN. Declarations of interests were made by all members of the guideline development group. Further details are available from the SIGN Executive. Guideline development and literature review expertise, support, and facilitation were provided by the SIGN Executive

Professor Stradling and Dr Kinnear formally represented the British Thoracic Society in the development of this guideline and incorporated feedback from the BTS into the discussions of the group.

8.3 SYSTEMATIC LITERATURE REVIEW

The evidence base for this guideline was synthesised in accordance with SIGN methodology. A systematic review of the literature was carried out using an explicit search strategy devised by a SIGN Information Officer in collaboration with members of the guideline development group.

Internet searches were carried out on the Web sites of the Canadian Practice Guidelines Infobase, the New Zealand Guidelines Programme, the UK Health Technology Assessment Programme, the US National Guidelines Clearinghouse, and the US Agency for Healthcare Research and Quality. Searches were also carried out using Google and OMNI search engines, and all suitable links followed up.

Database searches were carried out on the Cochrane Library, Embase, Medline, and Psychological Abstracts. With the exception of the Cochrane Library, all searches were restricted to the period 1991 – 2000. The Medline version of the main search strategies is available on the SIGN Web site, in the section covering supporting material for published guidelines.

The main searches were supplemented by material identified by individual members of the development group.

8.4 CONSULTATION AND PEER REVIEW

8.4.1 NATIONAL OPEN MEETING

A national open meeting is the main consultative phase of SIGN guideline development, at which the guideline development group present their draft recommendations for the first time. The national open meeting for this guideline was held on 3 April 2001 and was attended by 77 representatives of all the key specialties relevant to the guideline. The draft guideline was also available on the SIGN web site for a limited period at this stage to allow those unable to attend the meeting to contribute to the development of the guideline.

8.4.2 SPECIALIST REVIEWERS INVITED TO COMMENT ON THIS DRAFT

The guideline was also reviewed in draft form by the following independent expert referees, who were asked to comment primarily on the comprehensiveness and accuracy of interpretation of the evidence base supporting the recommendations in the guideline. SIGN is very grateful to all of these experts for their contribution to the guideline.

Mr Grant Bates	*ENT Surgeon, Radcliffe Infirmary, Oxford*
Dr Jim Beattie	*Director of Guideline Development, RCGP*
Dr Alan Begg	*General Practitioner, Montrose*
Professor Peter Calverley	*Professor of Respiratory Medicine, University Hospital, Liverpool*
Dr Jim Catterall	*Consultant Respiratory Physician, Bristol Royal Infirmary*
Mr Charles Croft	*Consultant ENT Surgeon, Royal Ear Nose and Throat Hospital, London*
Mr John Crowther	*Consultant ENT Surgeon, Victoria Infirmary, Glasgow*
Professor John Gibson	*Consultant Respiratory Physician, Newcastle upon Tyne*
Mr Frank Govan	*Chairman, Sleep Apnoea Trust*
Dr Geoff Hulks	*Consultant Physician, Raigmore Hospital, Inverness*
Dr Ama Johal	*Consultant Orthodontist, Department of Oral Growth and Development, Dental School Division, London*
Dr Max Kalsi	*Specialist Registrar in Public Health Medicine, Chesterfield Primary Care Trust*
Mr Ken MacKenzie	*Consultant Head and Neck Surgeon, Glasgow Royal Infirmary*
Mr William McKerrow	*Consultant ENT Surgeon,Raigmore Hospital, Inverness*
Dr Allan Merry	*General Practitioner, Ardrossan*
Ms Debby Nicoll	*Specialist Nurse, Sleep Unit, Churchill Hospital, Oxford*
Dr Paul Rafferty	*Consultant Physician, Dumfries & Galloway Royal Infirmary*
Dr John Wright	*Consultant in Public Health, Bradford*

8.4.3 SIGN EDITORIAL GROUP

As a final quality control check, the guideline is reviewed by an Editorial Group comprising the relevant specialty representatives on SIGN Council to ensure that the specialist reviewers' comments have been addressed adequately and that any risk of bias in the guideline development process as a whole has been minimised. The Editorial Group for this guideline was as follows.

Dr David Alexander	*BMA Scottish General Practice Committee*
Mr Douglas Harper	*Royal College of Surgeons of Edinburgh*
Professor Gordon Lowe	*Chairman of SIGN*
Dr Lesley Macdonald	*Faculty of Public Health Medicine*
Professor Nigel Pitts	*National Dental Advisory Committee*
Dr Safia Qureshi	*SIGN Programme Director*
Dr Sara Twaddle	*Director of SIGN*

9 Implementation and audit

9.1 LOCAL IMPLEMENTATION

Implementation of national clinical guidelines is the responsibility of each NHS Trust and is an essential part of clinical governance. It is acknowledged that every Trust cannot implement every guideline immediately on publication, but mechanisms should be in place to ensure that the care provided is reviewed against the guideline recommendations and the reasons for any differences assessed and, where appropriate, addressed. These discussions should involve both clinical staff and management. Local arrangements may then be made to implement the national guideline in individual hospitals, units and practices, and to monitor compliance. This may be done by a variety of means including patient-specific reminders, continuing education and training, and clinical audit.

9.2 RESOURCE IMPLICATIONS

Recommendations likely to be associated with additional resource use for NHSScotland are highlighted in Annex 2. There are likely to be corresponding reductions in costs associated with cardiovascular and pulmonary diseases in patients with OSAHS and in road traffic accidents, associated with treating these conditions.

9.3 KEY POINTS FOR AUDIT

- Availability of specific diagnostic tests and treatment options for OSAHS
- Proportion of patients with a positive diagnosis of OSAHS receiving treatment according to locally agreed protocols
- Proportion of patients with OSAHS using CPAP for more than two hours each night
- Proportion of patients being treated with CPAP who are using a heated humidifier system to aid compliance
- Proportion of patients with OSAHS failing to tolerate IODs and CPAP and the underlying reasons for failure
- Determination of frequency, nature and outcome of the surgery that is being performed for the treatment of OSAHS and snoring
- Proportion of postoperative patients suffering from previously undiagnosed OSAHS

9.4 RECOMMENDATIONS FOR RESEARCH

- Identification of the severity threshold for long term benefits from treatment for OSAHS
- Large scale trials comparing the benefits of treatments proven to be effective in OSAHS
- Evaluation of the cost effectiveness of treating OSAHS and screening in high risk groups (eg professional drivers and pilots)
- Clarification of the causes of excessive sleepiness whilst driving
- Improving the available subjective and objective tests to accurately assess whether patients are safe to drive
- Clarification of the mechanism of the association between OSAHS and hypertension
- Assessing whether OSAHS predisposes to myocardial infarction and stroke
- Assessment of the role of nurse led clinics in the diagnosis and subsequent management of patients with OSAHS
- Assessment of efficacy of algorithms for the diagnosis and treatment of OSAHS
- Comparison of hospital vs home titration of CPAP pressures in the management of OSAHS
- Comparison of treatment outcomes of conventional vs “smart” autotitrating CPAP in the management of OSAHS

Annex 1

THE EPWORTH SLEEPINESS SCALE

How likely are you to doze off or fall asleep in the following situations in contrast to just feeling tired? This refers to your usual way of life in recent times. Even if you have not done some of these things, try to work out how they would have affected you.

Use the following scale to choose the most appropriate number for each situation.

0 = would never doze

1 = slight chance of dozing

2 = moderate chance of dozing

3 = high chance of dozing

Situation	Chance of Dozing
Sitting and reading	_____
Watching TV	_____
Sitting inactive in a public place (eg 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 a lunch without alcohol	_____
In a car, while stopped for a few minutes in traffic	_____
TOTAL (max. 24)	

Annex 2 Resource Implications of recommendations

Guideline section		Recommendation / Good practice point	Likely resource implication
3.1	C	**All patients who have suspected sleep apnoea and their partners should complete an Epworth questionnaire to subjectively assess the degree of pretreatment sleepiness.**	There is a need for awareness raising about the Epworth Sleepiness Scale and its use, particularly in primary care.
3.2	☑	Patients with symptoms suggestive of OSAHS, who are sleepy whilst driving or working with machinery, or are employed in hazardous occupations should be considered for urgent referral to a sleep centre, as should those with ventilatory failure.	Prevalence of OSAHS in adults in Scotland aged 30-65 can be estimated in the range from 18,000 to 25,000. Experience from the Sleep Centre at Edinburgh Royal Infirmary suggests a referral rate of approximately 600/780,000 population – this equates to 4,000 referrals per annum in Scotland. During 2001/2 there were 1,529 referrals to the Edinburgh Sleep Centre. Approximately 800-1000 patients were referred to the other centres in total. These figures suggest an increase in referrals per annum in the region of 1,500 to 1,700 across Scotland. It is unlikely that such a significant increase in referrals could be achieved within existing facilities
3.6	B	**Limited sleep studies to assess respiratory events are an adequate first line method for diagnostic assessment for OSAHS.**	There are 6 centres in Scotland with dedicated sleep beds, with a total of 13 available beds. A further 3 centres provide facilities for investigations (British Sleep Society survey 2002). Approximately 50% referrals result in hospital based studies. This would equate to a requirement for approximately 2,000 hospital based studies each year. It is likely therefore that full implementation of this recommendation would require expansion in workload of the existing centres or new centres being set up
4.3.1	A	**CPAP is the first choice therapy for patients with moderate or severe OSAHS that is sufficiently symptomatic to require intervention.**	CPAP use requires an initial purchase of the appliance, annual maintenance and replacement at the end of the machine's lifespan (expected to be 10 years). In addition, there are costs associated with clinical review and an annual mask replacement. Experience from the Edinburgh Royal Infirmary Sleep Centre suggests that between 30% and 50% of referrals lead to CPAP use. This would equate to between 1,200 and 2,000 new patients on CPAP per annum. An increase in referral to sleep centres is likely to lead to an increase in the numbers of CPAP appliances being used in Scotland.
4.3.4	A	**Intra-oral devices are an appropriate therapy for snorers and for patients with mild OSAHS with normal daytime alertness.**	Intra-oral devices are custom made for individuals and last around two years. In addition to the cost of the device there will be costs associated with annual review of patients.
4.3.4	B	**Intra-oral devices are an appropriate alternative therapy for patients who are unable to tolerate CPAP.**	An increase in referral to sleep centres is likely to lead to an increase in the numbers of intra-oral devices used in Scotland.

Abbreviations

AHI	Apnoea/hypopnoea index
BMI	Body Mass Index
BP	Blood pressure
BTS	British Thoracic Society
COPD	Chronic obstructive pulmonary disease
CPAP	Continuous positive airway pressure
CT	Computed tomography
DVLA	Driver and Vehicle Licencing Agency
ECG	Electrocardiogram
EEG	Electroencephalogram
ENT	Ear, nose and throat
ESS	Epworth Sleepiness Scale
FEV_1	Forced expiratory volume
FVC	Forced vital capacity
GP	General Practitioner
HADS	Hospital Anxiety and Depression Score
HDU	High dependency unit
ICU	Intensive care unit
IOD	Intra-oral device
LAUP	Laser-assisted uvulopalatopharyngoplasty
MRI	Magnetic resonance imaging
MSLT	Multiple Sleep Latency Test
OSAHS	Obstructive sleep apnoea/hypopnoea syndrome
PSG	Polysomnography
QALY	Quality Adjusted Life Year
RCT	Randomised controlled trial
RDI	Respiratory disturbance index
SF-36	36 item short form health survey
SpO_2	Oxygen saturation of arterial blood by pulse oximetry
SIGN	Scottish Intercollegiate Guidelines Network
SpR	Specialist registrar
SSRI	Selective serotonin reuptake inhibitor
UPPP	Uvulopalatopharyngoplasty

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OBSTRUCTIVE SLEEP APNOEA

DEFINITIONS AND CLINICAL BACKGROUND

- Apnoea - a ten second breathing pause
- Hypopnoea - a ten second event where there is continued breathing but ventilation is reduced by at least 50% from the previous baseline during sleep
- Obstructive sleep apnoea/hypopnoea syndrome (OSAHS) - the coexistence of excessive daytime sleepiness with irregular breathing at night
- Apnoea/hypopnoea index (AHI) - the frequency of apnoeas and hypopnoeas hourly (used to assess the severity of OSAHS)

As the sufferer falls asleep the muscle tone in the upper pharyngeal airway decreases leading to upper airway narrowing. This in turn produces an increase in inspiratory effort in an attempt to overcome this airway narrowing which then leads to a transient arousal from deep sleep to wakefulness or a lighter sleep phase which allows restoration of normal airway muscular tone and calibre. The patient then falls more deeply asleep again and the whole cycle repeats itself. This can occur many hundreds of times throughout the night leading to fragmentation of normal sleep architecture and a reduction in the quality of sleep with the generation of restless, disturbed and unsatisfying sleep.

CLINICAL FEATURES

- excessive daytime sleepiness
- impaired concentration
- snoring
- unrefreshing sleep
- choking episodes during sleep
- witnessed apnoeas
- restless sleep
- irritability / personality change
- nocturia
- decreased libido

} *Dominant features*

SEVERITY OF OSAHS

OSAHS may be subdivided into varying degrees of breathing abnormality, for example, depending on the AHI:

Mild	AHI 5-14/hr
Moderate	AHI 15-30/hr
Severe	AHI >30/hr

ABBREVIATIONS

AHI	Apnoea/hypopnoea index
COPD	Chronic Obstructive Pulmonary Disease
CPAP	Continuous Positive Airway Pressure
OSAHS	Obstructive Sleep Apnoea / Hypopnoea Syndrome
PSG	Polysomnography

DIAGNOSIS

C **All patients who have suspected sleep apnoea and their partners should complete an Epworth questionnaire to subjectively assess the degree of pretreatment sleepiness.**

☑ The combination of severe OSAHS and COPD is potentially dangerous and in such cases clinicians should consider urgent referral to a sleep centre.

☑ Patients with symptoms suggestive of OSAHS, who are sleepy whilst driving or working with machinery, or are employed in hazardous occupations should be considered for urgent referral to a sleep centre, as should those with ventilatory failure.

☑ OSAHS should be excluded in patients before they are considered for surgery for snoring.

PHYSICAL EXAMINATION

Examination by itself cannot allow an accurate diagnosis of OSAHS but it does help to exclude other causes for the patient's symptoms. The following should be included in a physical examination:

- weight and height
- neck circumference
- mandible size
- nasal patency
- upper airway obstruction
- oral cavity (for macroglossia and dentition status)
- pharyngeal appearance
- blood pressure
- routine respiratory, cardiovascular and neurological measures

DIAGNOSTIC TOOLS

Various diagnostic tools are used in the assessment of OSAHS. Of these polysomnography, limited sleep studies and oximetry have been shown to be of value; flow volume loops, radiological imaging, questionnaires and nasendoscopy have not.

B **Limited sleep studies to assess respiratory events are an adequate first-line method of diagnostic assessment for OSAHS.**

☑ Full PSG with EEG-based sleep staging is not necessary to diagnose sleep apnoea in most patients. It should be available in regional sleep centres for patients who have typical symptoms of excessive daytime somnolence but no objective evidence of obstructive sleep apnoea on limited testing.

☑ Oximetry studies cannot exclude OSAHS. Studies using oximetry alone may have a role in the initial assessment of OSAHS, however their significant limitations must be fully appreciated before using them to make diagnostic and therapeutic decisions.

TREATMENT

Current evidence from randomised controlled trials indicates that improvements with treatment can be found in symptomatic patients with AHI ≥15 or a 4% oxygen saturation dip rate at the level of > 10/hour.

BEHAVIOURAL INTERVENTIONS

C **Weight loss should be encouraged in all patients with obesity contributing to their OSAHS. Attempts at weight loss should not delay the initiation of further treatment. Weight loss should also be encouraged as an adjunct to CPAP or intra-oral devices as it may allow discontinuation of therapy.**

☑
- Patients who smoke should be advised to stop
- Alcohol and sedatives or sleeping tablets should be avoided
- Non-sleepy snorers should be discouraged from sleeping on their backs.

NON-SURGICAL INTERVENTIONS

Continuous Positive Airway Pressure (CPAP)

A **CPAP is the first choice therapy for patients with moderate or severe OSAHS that is sufficiently symptomatic to require intervention.**

C **Persistent low CPAP use (less than two hours per night) over six months, following efforts to improve patient comfort, should lead to a review of treatment.**

☑ CPAP therapy should not be abandoned without:
- the attention of a trained CPAP nurse / technician
- a titration study / use of autotitrating CPAP to troubleshoot problems
- the use of heated humidification.

B **Bi-level ventilation should not be used routinely in OSAHS but should be reserved for patients with ventilatory failure.**

Intra-oral devices

A **Intra-oral devices are an appropriate therapy for snorers and for patients with mild OSAHS with normal daytime alertness.**

B **Intra-oral devices are an appropriate alternative therapy for patients who are unable to tolerate CPAP.**

D **The use of intra-oral devices should be monitored following initiation of therapy to allow device adjustment and assessment of OSAHS control and symptoms.**

Pharmacological therapy

A **Pharmacological therapy should not be used as first line therapy for OSAHS.**

SURGICAL INTERVENTIONS AND ANAESTHESIA

B **Use of UPPP or LAUP for the treatment of OSAHS is not recommended.**

☑ The presence of large tonsils in a patient with diagnosed OSAHS should prompt referral to an ENT surgeon for consideration of tonsillectomy.

☑ The effect of anaesthesia during surgery may increase the severity of the apnoea postoperatively. When a patient is being treated by CPAP preoperatively this should be continued immediately following surgery.

☑ All patients with OSAHS should be monitored with oximetry postoperatively and further management decided on an individual basis.