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The obstructive sleep apnoea/hypopnoea syndrome – An overview

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A B S T R A C T

In the last 30 years, there has been an explosion in facilities for the diagnosis and treatment of the obstructive sleep apnoea/hypopnoea syndrome (OSAHS) as well as a rapid advancement in the understanding of its consequences. In the general population, the prevalence of OSAHS is approximately 3–7% in adult men and 2–5% in adult women. OSAHS has been recognised as an independent risk factor for disorders such as hypertension, cardiovascular disease and sleepiness-related accidents. Currently, it is considered to be a systemic disease. This review provides a general overview of OSAHS: its epidemiology, pathophysiology, clinical features, diagnosis and treatment, as well as its consequences for public health.

Educational aims

- To outline the pathophysiology, epidemiology and clinical presentation of the obstructive sleep apnoea/hypopnoea syndrome.
- To outline the investigation of OSAHS and the potential difficulties in its diagnosis.
- To outline the management of OSAHS and associated potential problems.

1. Introduction

The obstructive sleep apnoea/hypopnoea syndrome (OSAHS) affects approximately 2–4% of the middle-aged population and is defined on the basis of symptoms of daytime sleepiness and objective measures of disordered breathing during sleep. OSAHS is characterised by obstruction of the upper airway during sleep, resulting in repetitive breathing pauses accompanied by oxygen desaturation and arousal from sleep. This results in diurnal sleepiness and can lead to cognitive impairment and cardiovascular morbidity. The clinical presentation and diagnostic criteria of abnormal breathing in sleep are different for adult and paediatric cases.[1,2] This review will focus on OSAHS in adults.

2. Pathophysiology of the obstructive sleep apnoea/hypopnoea syndrome (OSAHS)

OSAHS is characterised by episodes of pharyngeal obstruction which are termed apnoeas if complete and hypopnoeas if partial. The term apnoea is defined, as a cessation of airflow for at least 10 s. Apnoeas/hypopnoeas are often, but not always, associated with an electroencephalographic arousal at their termination and with a drop in oxygen saturation.[2] Controversy remains regarding a standardised definition of hypopnoea, but the definition adopted by the American Academy of Sleep Medicine (AASM) is a 50% reduction in thoraco-abdominal movement for at least 10 s from the preceding stable baseline when asleep.[2]

Apnoeas and hypopnoeas may occur at sleep onset or during rapid eye movement (REM) sleep in healthy individuals. Such events are not repetitive; airflow cessation is less than 10 s and is not usually accompanied by arousals, hypoxemia or an increased in arterial $PaCO_2$.

The pharynx is the site of UA obstruction during sleep in patients with OSAHS. During inspiration, the size of the pharyngeal lumen depends on the balance between narrowing forces resulting from intrapharyngeal suction pressure and dilating forces generated principally by pharyngeal muscles. Patients with OSA commonly have anatomical abnormalities of the UA when awake.[3] The sites of UA narrowing can be broadly classified into three regions: the retropalatal region, the retroglossal region and the hypopharyngeal region. UA occlusion is the result of an interaction between multiple anatomic and physiological abnormalities which involve a small, highly compliant pharynx, central breathing instability leading to reduced ventilatory motor output to UA dilators and collapsing transmural pressure. The increase in air velocity resulting from inspiratory narrowing during sleep is thought to cause decreased intraluminal pressure according to the Bernoulli principle causing greater collapse of the UA.[3] Extraluminal pressures are also thought to influence UA patency including passive gravitational forces generated by craniofacial

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structure or adipose tissue surrounding the UA.[4] In addition, the loss of caudal traction on UA structures during sleep, as lung volume is reduced due to the displacement of the diaphragm and thorax toward the head, further increases UA resistance.[3] Surface mucosal factors are also thought to influence airway patency especially in subjects with a lot of mucosal inflammation from repeated trauma.[3]

3. Epidemiology of OSAHS

Large-scale epidemiological studies have attempted to address questions on the incidence and prevalence of OSAHS.[5] Differences in sampling methods, techniques used for monitoring sleep and breathing and variability in definitions can alter disease prevalence. The prevalence of OSAHS is approximately 3–7% for adult men and 2–5% for adult women in the general population.[5] The prevalence is higher in different population subsets, including overweight or obese people, those of a minority race, and older individuals. Minimally symptomatic or asymptomatic sleep apnoea is estimated to occur in one of five adults and is rarely recognised.[6]

4. Risk factors for OSAHS

4.1. Age

The prevalence of OSAHS increases with age [5–7] and reaches a plateau after the age of 60 years. Mechanisms proposed for the age-related increase in prevalence include increased deposition of fat in the parapharyngeal area, lengthening of the soft palate and changes in structures surrounding the pharynx.[8]

4.2. Obesity

The association between obesity and OSA has been noted in many studies.[4–6,9] Morbid obesity defined as a body mass index (BMI) of >30 kg/m^2, is present in 60–90% of patients with OSAHS. Central obesity characterised by a high waist:hip ratio or increased neck circumference is better correlated with OSAHS even in those with a normal BMI.[4] Fat distribution around the neck results in increased extraluminal pressure and may affect the geometry of the airway making collapse more likely.[4] Data suggest that weight gain has a greater effect on OSA than an equivalent weight loss.[10] There is evidence of a significant correlation between metabolic syndrome (central obesity, insulin resistance, impaired glucose tolerance, dyslipidaemia, and hypertension) and OSAHS.[11] Sleep deprivation and poor sleep quality as consequences of OSAHS can lead to changes in appetite.[4,9] Obesity *per se* is an inflammatory state and its metabolic consequences may affect ventilatory control.[4,9]

4.3. Men, menopause and pregnancy

OSAHS is more common in men.[5,6,12] The male predisposition for the disorder has been attributed to differences in anatomical and functional properties of the UA, differences in craniofacial morphology and fat deposition and different ventilatory responses to arousals from sleep.[12] Women with sleep apnoea often do not report loud snoring, gasping and witnessed apnoeas, but may more frequently report symptoms of fatigue and lack of energy. Health care providers should have a lower index of suspicion for considering OSAHS in women[5] and there may be a non-response of the bed partner to the symptoms of obstructive breathing during sleep. Women tend to have a lower AHI in non-rapid eye movement (non-REM) sleep but have a similar AHI in REM sleep to men.[12]

Hormonal influences also have an important role in the pathogenesis of OSAHS. The disease prevalence is higher in post- versus pre-menopausal women and hormone replacement therapy has been associated with a lower prevalence.[13] Exogenous androgen therapy in men and women can aggravate OSAHS severity[14] and women with polycystic ovary syndrome[15] have a high prevalence of OSAHS.

Pregnancy is associated with a higher prevalence of sleep apnoea, particularly during the third trimester. Gestational weight gain, decrease in pharyngeal luminal size and alterations in pulmonary physiology are likely to predispose to sleep disordered breathing.[12] However, some physiologic changes that accompany pregnancy, like higher progesterone levels and a decrease in sleep time in the supine position may protect against OSA. OSA during pregnancy may lead to intrauterine growth retardation, pre-eclampsia, lower Apgar scores and birth weight.[16]

4.4. Race

OSAHS prevalence is as high or higher in African-Americans and in Hispanics compared with Caucasians.[5,6,17] For a given age, sex, and BMI, Asians have greater disease severity than whites, despite their lesser degrees of obesity, probably due to differences in craniofacial features, such as a more crowded UA, relative retrognathia and a shorter cranial base.[17]

4.5. Craniofacial abnormalities

OSAHS is associated with craniofacial abnormalities such as retrognathia, tonsillar hypertrophy, enlarged tongue or soft palate, inferiorly positioned hyoid bone, maxillary and mandibular retropositions. Jaw abnormalities are important in thin OSAHS patients.[18] First-degree relatives of OSAS patients are more likely to be at risk compared with the relatives of people without the disorder. Genetic determinants of cephalometric parameters, obesity and regional fat distribution are also relevant. It is likely that OSAHS is a polygenic disorder with multifactorial genetic influence.[19] Certain congenital conditions such as Marfan's, Down's and the Pierre–Robin syndromes predispose to the development of OSAHS.[20]

4.6. Lifestyle factors

Smoking is associated with a higher prevalence of snoring and OSAHS and passive smoking has been linked with habitual snoring.[21] Alcohol can increase UA collapsibility by reducing the genioglossus muscle activity, prolonging apnoea duration and exacerbating OSAHS.[22]

4.7. Other conditions

Acquired conditions such as acromegaly and hypothyroidism are also associated with OSAHS.[20,23] Sedative use, sleep deprivation and supine posture can all exacerbate sleep apnoea. Reduced nasal patency, due to congestion or anatomical defects as well as respiratory allergies can also contribute to OSAHS.[18]

5. Clinical features of OSAHS

OSAHS symptoms can be divided into those manifesting during sleep and those present during wakefulness (Table 1).

Patients with OSAHS most commonly complain of excessive daytime sleepiness (EDS). This may range from subtle to severe. However, EDS is not present in all patients with OSAHS. A recent multicenter cohort study concluded that apnoea and sleep

Table 1
Symptoms of the obstructive sleep apnoea/hypopnoea syndrome.

During sleep	When awake
Nonrestorative sleep	Daytime sleepiness
Witnessed apnoeas by bed partner	Lack of concentration
Awakening with choking	Cognitive deficits
Nocturnal restlessness	Changes in mood
Vivid, strange, or threatening dreams	Morning headaches
Gastroesophageal reflux	Dry mouth
Insomnia with frequent awakenings	Impotence or decreased libido
Nocturia	
Drooling	
Diaphoresis	

disruption were not the primary determinants of EDS, although patients with EDS were characterised by longer sleep duration, increased slow wave sleep and sleep fragmentation.[24] So the approach to a sleepy patient presenting with symptoms of sleep apnoea should not exclude consideration and assessment of metabolic status, the presence of depression or other possible causes of EDS (Table 2).[25]

Nocturnal symptoms of OSAHS are sometimes apparent to the patient but generally, are reported by a bed partner. The most common include snoring, snorting, choking attacks terminating a snore and witnessed apnoeas. Although an absence of snoring does not exclude the diagnosis of OSAHS, virtually all patients snore. Apnoeic episodes are reported by about 75% of bed partners.[26] Bed partners will generally report a sudden cessation of snoring followed by a loud snort and a resumption of snoring. Some patients will awake after such a sequence; however, most are unaware of their disordered breathing.[27] Those who are aware of frequent events leading to awakening can try to delay and prevent themselves from falling asleep, thus presenting with a 'paradoxical' insomnia. A number of clinical features are associated with OSAHS (Table 3), but the predictive value of any single one is limited in confirming the diagnosis.[26]

History and clinical examination alone (including blood pressure and BMI) can predict the presence of OSAHS in only 50% of patients attending a sleep disorders clinic.[26] Definitive diagnosis requires overnight investigation of breathing patterns (Table 4).

6. Morbidity and mortality associated with OSAHS

Untreated OSAHS can contribute to the development or progression of other disorders. Patients with OSAHS have higher mortality, use more medical resources and have greater medical disability.[28,29]

6.1. Systemic hypertension

OSAHS is a cause of systemic hypertension.[30] The mechanisms contributing to this include tonic elevation of sympathetic neural activity by the augmentation of peripheral chemoreflex sensitivity (intermittent hypoxia at night), direct effects on sites of central sympathetic regulation and the disturbance of nocturnal renin and aldosterone levels due to severe sleep fragmentation.[31]

Table 2
Differential diagnosis of excessive daytime sleepiness.

Obstructive sleep apnoea/hypopnoea (OSAHS)
Upper airway resistance syndrome (UARS)
Narcolepsy – Central nervous system hypersomnolence
Periodic leg movements during sleep
Insufficient sleep syndrome
Alcohol, medication and drug use
Schedule disorders/shift work
Chronic pain and discomfort
Delayed sleep phase syndrome
Severe lung disease
Neurologic – Neuromuscular disease

Table 3
Clinical features of OSAHS.

Obesity (particularly central, BMI > 28 kg/m)
Large neck circumference (>40 cm)
Narrow mandible, narrow maxilla
Retrognathia
Dental malocclusion, overjet
Nasal problems (i.e. deviated nasal septum)
High and narrow hard palate
Elongated and low-lying uvula
Enlarged tonsils and adenoids
Macroglossia

6.2. Cardiac disease, dysrhythmias

OSAHS is associated with the development of ischemic heart disease.[32] Hypoxia, hypercapnia and pressure changes accompanying obstructive apnoeic events (reduction in intra-thoracic pressure) may stimulate the release of vasoactive substances. The reduction in intra-thoracic pressure increases the left ventricular transmural pressure and left ventricular afterload. Venous return is enhanced, resulting in right ventricular distension and leftward shift of the inter-ventricular septum. Cardiac function is compromised by a combination of diminished left ventricular pre-load and augmented left ventricular afterload, which together reduces the stroke volume. Chronic intermittent hypoxia can induce oxidative stress and an inflammatory state and thus contribute to the development of atherosclerosis in OSAHS patients.[32] OSAHS also leads to abnormalities of coagulation (hypercoagulability) and excessive platelet activation.[32]

Nocturnal disturbances in cardiac rhythm (heart block, atrial fibrillation, ventricular ectopy) have been reported in patients with OSAHS,[32] but the most common arrhythmias are severe sinus bradycardia and atrioventricular block, as a response to apnoea and hypoxia.[32] OSAHS may also be involved in the pathogenesis of nocturnal sudden death.[32]

6.3. Cerebrovascular disease

Patients with OSAHS appear to be at increased risk of having a stroke.[33] The negative intra-thoracic pressure generated during an apnoea decreases cerebral blood flow predisposing to ischemic changes in some patients. Other risk factors for stroke, such as systemic hypertension, heart disease, impaired vascular endothelial function and proinflammatory states may be exacerbated by OSAHS. Some studies show no increase in OSAHS in those with TIA,[34] whilst others show a high prevalence of OSAHS in those with stroke.[33] There are insufficient data to support that treatment of

Table 4
Differential diagnosis of OSAHS.

Primary snoring
Sleep-choking disorder
Nocturnal asthma
Gastroesophageal reflux disease
Insomnia
Panic attacks
Nocturnal seizures
Underlying pulmonary disease
Sleep-related laryngospasm

OSAHS will reduce stroke risk or that treating OSAHS post-stroke leads to improvement in quality of life.[35]

6.4. Pulmonary hypertension – Cor pulmonale

OSAHS may contribute to the development of pulmonary hypertension (PH). The apnoea-induced hypoxia appears to be the most important determinant of PH. Development of right ventricular hypertrophy is related to the severity of OSA, but development of right heart failure requires additional alterations in daytime respiratory function and is associated with lung disease or morbid obesity.[36]

6.5. Co-existent lung disease

The combination of chronic obstructive pulmonary disease (COPD) and OSAHS has been termed as "overlap syndrome".[37] Quality of sleep in COPD is influenced by the presence of OSAHS but not by the severity of airway obstruction. Patients with overlap syndrome, are more sleepy, have lower total sleep time and higher AHI compared with those who have COPD alone and are at greater risk of developing PH, right heart failure and hypercapnia even though their obstructive defect may not be severe.[37] Nocturnal asthma may also be worsened by sleep apnoea.[38]

6.6. Neuropsychological impairment-quality of life

OSAHS leads to neuropsychological impairment that includes deficits in attention, concentration, vigilance, manual dexterity, visuomotor skills, memory, verbal fluency and executive function.[27] Sleepiness, depression, fatigue and obesity have an important impact on the person's quality of life.[39] Patients report problems with relationships and sex, as well as concerns about poor memory and a fear of dying.[39]

6.7. Accidents

One of the most important complications and the one which has the greatest impact from the public health perspective is driving accidents.[40] Vigilance testing and driving simulators in studies assessing driving performance in OSAHS patients reveal that performance is markedly reduced and the impairment is not just limited to periods when patients actually fall asleep but also when they are awake due to reduced vigilance. Motor vehicle crashes correlate with the degree of sleepiness and not the AHI.[40] There is also evidence that OSAHS patients have an increased risk of work place accidents.[41]

6.8. OSAHS as a systemic disease

OSAHS patients have increased levels of certain biomarkers relating to metabolic, cardiovascular and other systemic consequences.[11,42] The levels of some biomarkers decrease with continuous positive airway pressure (CPAP) treatment and this observation indicates that OSAHS is more than a local abnormality and should be considered a systemic disease.[11,42]

7. Diagnosis of OSAHS

A definitive diagnosis of OSAHS requires objective recording and measurement of sleep and breathing during the night in addition to a measure of daytime sleepiness (objective or subjective) and other symptoms.

7.1. Recording and measurement of sleep and breathing during the night

The method most widely used, and which is considered by some to be the 'gold standard' for diagnosis of OSAHS, is overnight polysomnography (PSG).[43] Most PSG studies monitor the following: nasal and/or oral airflow; thoraco-abdominal movement; snoring; electroencephalogram (EEG); electro-oculogram (EOG); electromyogram (EMG); and oxygen saturation. Also recording of any abnormal movements using video may help identify changes in airflow or desaturations. Signal collection and interpretation are usually computerised, but manual scoring of the trace should still be performed using guidelines for interpretation of the EEG[2,44] for scoring of respiratory and other events. Night to night variability makes it possible for a single study to underestimate the severity of OSAHS. A negative PSG should be viewed with scepticism if the clinical suspicion of OSAHS is high.[45]

Split-night studies[46] in which the first half of the study night is used for diagnosis and the second half to monitor treatment response using continuous positive airway pressure are also used. Split-night studies are considered accurate and cost effective when criteria for conducting them are met.[46]

A more recent introduction to the assessment of sleep apnoea has been cardiorespiratory monitoring alone. This involves the measurement of airflow, respiratory effort, oxygen saturation and heart rate, but not EEG. The great advantages of these systems are price and portability and the ability of patients to monitor themselves at home.[47] Overnight oximetry is sometimes used as a screening test for identifying patients who are at risk of having significant OSAHS but should never be seen as a substitute for in-lab PSG or home cardiorespiratory monitoring. There are severe limitations of this technique, especially the inability to detect apnoeas or hypopnoeas not associated with oxygen desaturation. Furthermore, nocturnal oxygen desaturation may be related to sleep hypoventilation without UA obstruction e.g. COPD, severe kyphoscoliosis, muscular dystrophy.[43,47]

7.2. Assessing daytime sleepiness

Sleepiness is difficult to define.[48] It can be classified using behavioural measures, performance tests, self-evaluation by rating scales or direct electrophysiological measures. The most widely used and best-validated scale for assessing daytime sleepiness is the Epworth Sleepiness Scale (ESS).[49] An ESS of >11 out of 24 (maximum score) is generally indicative of abnormal levels of daytime sleepiness, irrespective of age. The ESS aims at measuring the general level of daytime sleepiness as a stable individual characteristic and has satisfactory test–retest reliability.[49]

8. Treatment of OSAHS

Until 1981, when Sullivan et al.[50] introduced CPAP as a treatment for OSAHS, tracheostomy[51] and weight loss[4] were the only established beneficial remedies. A number of modalities have been employed in the treatment of OSAHS, each with inherent limitations.[52,53]

8.1. Conservative measures

Lifestyle modification is probably the simplest, cheapest and least effective method for the long-term treatment of OSAHS. This includes advice about weight loss, exercise, and sleep hygiene, avoidance of smoking and alcohol and sedative medications. Weight loss can lead to clinically significant improvement.[4] Sleep position has also been looked at in terms of

modifying the degree of OSA.[52] Exercise is recommended as an adjunct to weight loss and to alter sleep structure. Sleep hygiene advice includes measures to improve the sleep environment, avoid stimulants before bedtime, avoid daytime naps, sleep deprivation etc.[52] Avoiding excessive alcohol in the evenings has been shown to be important,[22] whilst smokers have higher risk of snoring and OSA.[21] There are very few randomised controlled trials looking at the effects of these lifestyle modifications in the context of OSAHS but, as they are non-invasive, they continue to be recommended.

8.2. Continuous positive airways pressure (CPAP)

CPAP treatment is considered the treatment of choice in patients with moderate-to-severe OSAHS.[53,54] Patients with mild OSAHS or those with limited symptoms but comorbid conditions (e.g. ischemic heart disease) or who perform mission critical work (e.g. airline pilot, bus driver) must also be offered treatment.[54]

CPAP acts as a pneumatic splint, delivering a predetermined constant pressure during both inspiration and exhalation, preventing collapse of the pharyngeal airway by elevating the pressure in the oropharyngeal airway and reversing the transmural pressure gradient across the pharyngeal airway. The optimal pressure (cmH_2O) is titrated to reduce the AHI to less than five ideally. There are various mask sizes and types available. CPAP results in significant improvements in objective and subjective sleepiness, measures of oxygenation, mean arterial blood pressure and pulmonary haemodynamics as well as in health status and the mental health.[52–54] CPAP treatment reduces biomarkers that reflect proatherogenic inflammation, improves cardiovascular mortality and morbidity, reduces prevalent dysrhythmias and improves left ventricular function.[32] CPAP treatment effectiveness is limited by variable adherence to therapy. One can determine compliance using a patient questionnaire, by electrically timing how long the device is on each night or by electronically recording how long the patient is actually breathing through it each night.[54,55] When adherence is defined as greater than 4 h per night, 46–83% of patients with obstructive sleep apnoea have been reported to be nonadherent to treatment.[55] Specific predictors of CPAP adherence have not been consistently isolated. Symptomatic severity as measured by ESS, patient characteristics, such as claustrophobia, other psychological and social variables, increased nasal resistance, partner interaction and the method of CPAP initiation influence adherence.[55]

CPAP is not without problems and common side-effects can include: nasal congestion, nasal dryness, epistaxis, skin abrasions, partner intolerance and inconvenience.[54,55] Ways to improve adherence to therapy are humidification, behavioural interventions, patient-centered mask and device selection, patient education, early intervention and support of side-effects.[55]

8.3. Oral appliances (OA)

Today, the most commonly used oral appliances are the mandibular advancement devices (MADs) which reposition the mandible and the tongue in a forward position during sleep.[56] Excessive salivation, tooth discomfort and dental defects are some of the reported side-effects. Although MADs are effective at treating OSAHS, their benefit appears to be less than that of CPAP.[53] MADs should be used in the treatment of simple snoring or mild sleep apnoea, being reserved for the treatment of moderate or severe sleep apnoea only in instances where CPAP therapy had failed or has been declined by the patient.[53,56]

8.4. Surgery

Surgery for sleep apnoea is a complex and controversial field and encompasses procedures carried out by surgeons attached to the subspecialties of otolaryngology, maxillofacial surgery and specialised surgery of the gastrointestinal system (bariatric surgery).[57,58] The aim of surgical treatment is to bypass or remove the site of UA obstruction. Some surgeons hold the tracheostomy to be the 'gold standard' for treating OSAHS, as it conveniently bypasses the upper respiratory tract where the problem resides; but this solution comes with its fair share of comorbidities. Severe nasal mucosal congestion may impede nasal CPAP therapy or OA therapy and specific abnormalities such as a grossly deviated nasal septum or nasal polyposis should be treated in selected patients.[57]

8.5. Pharmacotherapy

There is currently little evidence for any of the drugs trialled in the management of OSAHS to be effective.[59] The effectiveness of modafinil, a central nervous system stimulant, for the treatment of residual sleepiness has been demonstrated, but there are conflicting data regarding the impact of modafinil therapy on CPAP use.[60]

9. Concluding remarks

OSAHS is an important condition within our community with the potential of being a significant health burden. OSAHS has been recognised as an independent risk factor for disorders such as hypertension, cardiovascular disease, and abnormalities in glucose metabolism, depression and sleepiness-related accidents. It can be easily diagnosed and readily, although not always easily, treated.

Conflict of interest statement

The authors have no conflict of interest.

CME section

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Educational questions

Answer the following question:

1. The prevalence of OSAHS in the general population for adult men is approximately:
   a. 3–7%
   b. 1–2%
   c. 2–5%
   d. 10–15%

2. OSAHS prevalence is higher:
   a. in pre- versus post-menopausal women
   b. in women on hormone replacement therapy
   c. in exogenous androgen therapy in men
   d. in people with BMI < 25 kg/m^2

3. Which one of the following is true when making a diagnosis of OSAHS?
   a. A negative PSG means OSAHS can be safely excluded
   b. Overnight oximetry can be used as a substitute for in-lab PSG or home cardiorespiratory monitoring for all patients
   c. Split-night studies are considered accurate and cost effective when criteria for conducting them are met
   d. An ESS in the range of <11 out of 24 is indicative of abnormal levels of daytime sleepiness.

4. Which of the following statements is true regarding the treatment of OSAHS?
   a. The most effective long-term treatment for patients with mild OSAHS is exercise and sleep hygiene
   b. MADs should be used routinely for treating severe sleep apnoea
   c. CPAP treatment increases biomarkers that reflect proatherogenic inflammation
   d. CPAP treatment effectiveness is limited by variable adherence to therapy.

5. Which of the following is true of OSAHS?
   a. Mild OSAHS alone can lead to the development of right heart failure
   b. OSAHS can contribute to the development of atherosclerosis
   c. History and clinical examination alone can predict the presence of OSAHS in 90% of patients attending a sleep disorders clinic
   d. All patients with OSAHS complain of excessive daytime sleepiness

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