The assessment and management of obstructive sleep apnoea–hypopnoea syndrome and obesity hypoventilation syndrome in obesity

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**ABSTRACT**

Obesity is associated with respiratory dysfunction. It is a key risk and contributory factor in the sleep related breathing disorders, obstructive sleep apnoea/hypopnoea syndrome (OSAHS) and obesity hypoventilation syndrome (OHS). Weight management is an integral part of the management of these disorders, in addition to continuous positive airways pressure (CPAP) and non-invasive ventilation (NIV). Untreated, these conditions are associated with a high disease burden and as treatment is effective, early recognition and referral is critical. Best practice in on-going care is multidisciplinary.

**KEYWORDS:** obstructive sleep apnoea/hypopnoea syndrome (OSAHS), obesity hypoventilation syndrome (OHS), continuous positive airway pressure (CPAP), non-invasive ventilation (NIV), weight management

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**Introduction**

The impact of obesity on lung function is multifactorial, related to mechanical and inflammatory aspects of obesity. The main sleep-related breathing disorders associated with obesity are obstructive sleep apnoea/hypopnoea syndrome (OSAHS) and obesity hypoventilation syndrome (OHS). Patients with OHS always have a body mass index (BMI) ≥ 30 mg/kg. Those with OSAHS have variable BMIs, although over 70% have been shown to have BMIs in the obese category. COPD–OSAHS overlap syndrome occurs in people who have both chronic obstructive pulmonary disease (COPD) and OSAHS (as these are two of the most prevalent pulmonary conditions, the combination is likely to be common). Although this syndrome can also be associated with obesity, this occurs in a smaller percentage of these patients compared with OSAHS and OHS; a recent large cross-sectional study found that only 7.8% of COPD patients also had a diagnosis of both OSAHS and obesity. For this article, we have therefore focused on OSAHS and OHS.

OSAHS is one of the most common sleep disorders. Estimated in 2014 to affect around 1.5 million adults in the UK, figures are now estimated to be likely double this. The prevalence of OHS is also likely to rise given the continued worldwide increase in obesity. It has been estimated that OHS affects 0.4% of the adult population, although this is likely to be an underestimate since OHS is known to be under-recognised. The prevalence of OHS increases as BMI rises. In retrospective studies among patients with OSAHS, the prevalence of OHS was observed to range from 8–12% in those with a BMI of 30 to 35 kg/m^2^, to 50% in those with a BMI ≥ 50 kg/m^2^.

Both conditions affect quality of life, and, untreated, the possible consequences are manifold. OSAHS is associated with increased risk of hypertension, stroke and heart disease. OSAHS is an independent risk factor for mortality and morbidity: severe OSAHS is associated with a 1.9 times increased risk in all-cause mortality and 2.65 times increased risk of cardiovascular mortality.

Untreated OHS is associated with significant morbidity including increased hospital admissions and need for respiratory or critical care support. Comorbidities such as heart failure, coronary artery disease and right heart failure are more common in patients with OHS. Studies have shown OHS patients could have poorer prognoses and a higher rate of hospitalisation and death when compared to OSAHS patients. Management, which centres on continuous positive airways pressure (CPAP) and non-invasive ventilation (NIV) and weight loss, can differ in OSAHS and OHS, so it is important to differentiate between them to ensure optimal management.

Although these sleep disorders are managed by respiratory sleep specialists, patients often initially present to primary care or other specialties across medicine, anaesthetics and surgery. Certain specialities should be mindful that there is a higher prevalence of sleep disorders in those with obesity, hypertension, type 2 diabetes, cardiac disorders and severe asthma. Treatment is effective, improves quality of life, reduces the disease burden associated with untreated disease, and is cost effective in both conditions. So early recognition is key to ensuring timely treatment, thereby reducing disease burden as well as sooner improvement in quality of life. However, awareness has been estimated to be low, with up to 85% of cases thought to be undiagnosed.

We have written this article to encourage increased awareness amongst non-specialists of the key symptoms and associated...
conditions of these obesity-related sleep disorders, using two case histories to illustrate how such patients may present to non-specialists. We have also outlined best practice in assessment and referral of those you suspect may have these conditions with an overview of their initial and ongoing management.

Case study 1: Obstructive sleep apnoea/hypopnoea syndrome (OSAHS)

OSAHS is a condition that occurs during sleep when the upper airway muscles relax, in which the upper airway repeatedly closes or narrows (obstructs). This causes apnoeas (stopping breathing) or hypopnoeas (under-breathing secondary to decreased airflow). During these episodes, efforts are made to breathe and the person briefly awakens to stop these episodes. This leads to disrupted sleep often resulting in excessive daytime sleepiness, often impacting quality of life.3

Presentation

A 57-year-old man who works in office administration attends the cardiology clinic. He has atrial fibrillation (AF) and had an ablation of AF a year ago. His AF has now recurred. He also has hypertension and has excellent adherence with three anti-hypertensive medications at maximum doses. His clinic, home and ambulatory blood pressure over the last 6 months has been 145/95 mmHg or more. He also mentions he feels tired all the time. At the time of his ablation, his BMI was 29 kg/m² and it is now 33 kg/m².

His history should lead you to suspect OSAHS. Additionally, untreated OSAHS is associated with hypertension. Although his hypertension may be associated with obesity, as it is inadequately controlled on triple anti-hypertensive medication, undiagnosed OSAHS may be a contributory factor in his hypertension and its response to treatment.17 OSAHS is also highly prevalent in AF, and those with untreated OSAHS are known to have a poor response to catheter ablation.18

The pathophysiological mechanisms by which OSAHS is associated with hypertension are multifactorial. They include peripheral vessel constriction, increased heart rate and arterial stiffness. These mechanisms are thought secondary to sympathetic nerve activation due to the periodic hypoxemia, frequent arousals and sleep deprivation occurring in OSAHS, as well as systemic inflammation and oxidative stress, and endothelial dysfunction.17

Assessment

As you suspect he may have OSAHS, you should take the following steps.

Ask the patient about key OSAHS symptoms, such as daytime sleepiness, snoring, witnessed apnoeas and waking headaches. His wife has noticed that while he is asleep he snores, at times stops breathing, and occasionally wakes up gasping for breath. He is concerned that he sometimes falls asleep during work meetings.

Check the patient’s score on the Epworth Sleepiness Scale (ESS) (Table 1) and consider checking a STOP-BANG (Table 2). His ESS of 20 is consistent with severe excessive daytime sleepiness and his STOP-BANG is 7 (indicating high risk of OSAHS). Note that some people with OSAHS do not have excessive sleepiness. For example, some patients present with tiredness and fatigue. Therefore, you should not use ESS alone to determine whether to refer someone.

As the patient has at least two of the key features of OSAHS and an increased ESS and STOP-BANG score, you should refer him to a sleep service. Depending on local processes, you may be able to request respiratory polygraphy or a nocturnal oximetry study yourself prior to referral.

Sleep studies: These are used to diagnose sleep disorders by recording multiple channels while the person is asleep. Box 1 describes the different types of sleep study and gives definitions of the terms used.

### Table 1. Epworth Sleepiness Scale

How likely are you to doze off or fall asleep in the following situations, in comparison to feeling just tired? This refers to your usual way of life in recent times.

<table>
<thead>
<tr>
<th>Scenarios:</th>
<th>Score:</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sitting and reading</td>
<td>0 = no chance of dozing</td>
</tr>
<tr>
<td>Watching TV</td>
<td>1 = mild chance of dozing</td>
</tr>
<tr>
<td>Sitting in a public place</td>
<td>2 = moderate chance of dozing</td>
</tr>
<tr>
<td>Passenger in a car for 1 hour without a break</td>
<td>3 = high chance of dozing</td>
</tr>
<tr>
<td>Lying down in the afternoon</td>
<td></td>
</tr>
<tr>
<td>Sitting and talking to someone</td>
<td></td>
</tr>
<tr>
<td>Sitting quietly after lunch, without having drunk alcohol</td>
<td></td>
</tr>
<tr>
<td>In a car/bus stopped at traffic for a couple of minutes</td>
<td></td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Epworth Sleepiness Scale (/24)</th>
<th>Excessive daytime sleepiness</th>
</tr>
</thead>
<tbody>
<tr>
<td>0–5</td>
<td>Lower normal daytime sleepiness</td>
</tr>
<tr>
<td>6–10</td>
<td>Higher normal daytime sleepiness</td>
</tr>
<tr>
<td>11–12</td>
<td>Mild excessive daytime sleepiness</td>
</tr>
<tr>
<td>13–15</td>
<td>Moderate excessive daytime sleepiness</td>
</tr>
<tr>
<td>16–24</td>
<td>Severe excessive daytime sleepiness</td>
</tr>
</tbody>
</table>

Obstructive sleep apnoea/hypopnoea syndrome and obesity hypoventilation syndrome

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**Table 2. STOP-BANG scoring**

<table>
<thead>
<tr>
<th>Question</th>
<th>Y/N</th>
</tr>
</thead>
<tbody>
<tr>
<td>Do you snore loudly (loud enough to be heard through closed doors or your bed-partner elbows you for snoring at night)?</td>
<td></td>
</tr>
<tr>
<td>Do you often feel tired, fatigued or sleepy during the daytime (such as falling asleep during driving or talking to someone)?</td>
<td></td>
</tr>
<tr>
<td>Has anyone observed you stop breathing or choking/gasping during your sleep?</td>
<td></td>
</tr>
<tr>
<td>Do you have or are being treated for high blood pressure?</td>
<td></td>
</tr>
<tr>
<td>Body mass index more than 35 kg/m²?</td>
<td></td>
</tr>
<tr>
<td>Age older than 50?</td>
<td></td>
</tr>
<tr>
<td>Neck size large? (Measured around Adams apple)</td>
<td></td>
</tr>
<tr>
<td>Is your shirt collar 16 inches/40 cm or larger?</td>
<td></td>
</tr>
<tr>
<td>Gender = male?</td>
<td></td>
</tr>
</tbody>
</table>

**Risk of OSAHS**

- Yes to 0–2 questions = Low risk
- Yes to 3–4 questions = Intermediate risk
- Yes to 5–8 questions = High risk

**Box 1. Sleep studies**

**Respiratory polygraphy**

Respiratory polygraphy is a multi-channel study that can be done at home or in hospital; it should initially be offered at home. Measurements are taken via a nasal flow sensor, thoracic and abdominal bands and oxygen saturation monitoring. It records apnoeas and hypopnoeas as well as oximetry, snoring and body position.¹⁰

**Polysomnography**

Polysomnography combines respiratory polygraphy with additional monitoring, including assessment of sleep quality and duration using monitoring of brain activity via electroencephalogram (EEG), eye movements via electrooculogram (EOG), and muscle tone via electromyogram (EMG). Polysomnography can be requested via sleep physicians if respiratory polygraphy results are negative but the person has significant symptoms (the AHI can be underestimated in respiratory polygraphy). Definitions used in sleep studies²⁰

**Apnoeas**: defined as ≥10 s of complete absence of airflow.

**Hypopnoeas**: defined as ≥30 % decrease in airflow from baseline, for ≥10 s, associated with desaturation of ≥3 % (with ≥4 % cut off also acceptable).

**The apnoea-hypopnea index (AHI)**: the number of apnoeas or hypopnoeas per hour of sleep; it is used for diagnosis and to classify severity. OSAHS is diagnosed when total AHI ≥5/hr with ≥5/h obstructive events (apnoeas/hypopnoeas with continued or increased inspiratory effort). The severity of OSAHS is stratified as follows:

- Normal: <5/hr
- Mild: 5–14.9/hr
- Moderate: 15–29.9/hr
- Severe: ≥30/hr

**The oxygen desaturation index (ODI)**: the number of desaturations (oxygen levels dropping by ≥3 % and/or 4 % from baseline) per hour of sleep. has been shown to reduce the severity of OSAHS by 50 % in moderately obese patients.⁸ With significant weight loss, a sleep study should be repeated. Advice on any sleep hygiene, smoking and alcohol issues should also be offered.

**Driving advice**

As he does not have excessive daytime sleepiness when driving, as per DVLA guidance, the patient does not need to stop driving or inform the DVLA (see Table 3).²¹,²² It is important that anyone with excessive sleepiness when driving must stop driving until their symptoms are controlled; there is an increased risk of road traffic accidents in OSAHS. Conversely, someone should not be told to stop driving simply because they have OSAHS if they do not have excessive sleepiness when driving, as this can lead to loss of livelihood and impact quality of life.

**CPAP**

The patient is started on auto-CPAP therapy with remote telemonitoring, as this is the recommended best practice for those

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**Oximetry studies**: If access to home respiratory polygraphy is limited, home oximetry for people with suspected OSAHS can be considered. However, oximetry cannot distinguish between obstructive and central sleep apnoea, and the severity of the sleep apnoea may be over or underestimated.

A referral letter should include assessment scores, and describe how sleepiness impacts the patient’s life, as well as including comorbidities, any occupational risk (such as being an HGV driver), and, if available, oxygen saturation and blood gas values.³

The patient is prioritised for review because of his suboptimally controlled arrhythmia and inadequately controlled hypertension. In addition to those with unstable cardiovascular disease, others who would be prioritised for assessment in the sleep service include those with a vocational driving job or a job for which vigilance is critical for safety, such as in security, and those undergoing assessment for major surgery.³

He undergoes a respiratory polygraphy sleep study (Fig 1). His snore percentage is 45%, his overall AHI is 31/h (all obstructive), his ODI is 28/h, his SpO₂ is <90% for 9% of the study, and his average oxygen saturations are 94%. He is diagnosed with symptomatic severe OSAHS.

**Management**

Management of OSHAS depends on symptoms and severity and should occur under a specialist sleep service. In this case, the patient is symptomatic and his untreated OSAHS may be associated with his cardiac disease.

**Lifestyle advice**

The patient is given advice on weight management. As he has struggled with weight loss, his GP is asked to refer him for further specialist support in this area. Weight loss of just 10–15%
Obstructive sleep apnoea/hypopnoea syndrome and obesity hypoventilation syndrome

reduce side effects such as nasal and mouth dryness that can limit adherence with therapy.

Follow up
After 2 weeks of CPAP therapy, he stops using it and contacts the sleep physiotherapy team. His usage has been limited by nasal and mouth dryness, and he has found the mask uncomfortable, often loosening the mask straps. On review, his remote monitoring shows a significant leak, so he is given a mask that he finds a more comfortable fit. A humidifier is also added. He starts to better tolerate the CPAP therapy. Ongoing multidisciplinary support is integral to supporting adherence with therapy.

3 months later, he is using his CPAP machine every night and his daytime sleepiness has significantly improved. The remote data shows complete correction of his OSAHS on CPAP therapy with an AHI on treatment of 3/h (31/h pre-CPAP). The data also confirms his excellent adherence of 7.5 hours on average per night and that there is no significant leak.

Other treatments
Other treatments for people with OSAHS include NIV, mandibular advancement, positional modifiers and surgery.  

Table 3. Driving advice

<table>
<thead>
<tr>
<th>Severity of OSAHS</th>
<th>Driving guidance</th>
</tr>
</thead>
<tbody>
<tr>
<td>Suspected OSAHS or mild OSA with ‘excessive sleepiness’ as defined by DVLA</td>
<td>Stop driving until symptoms controlled. Do not need to inform DVLA unless symptoms not under control in 3 months. Applies to both Group 1 (car and motorcycle) Group 2 (bus and lorry)</td>
</tr>
<tr>
<td>Moderate to severe OSAHS with ‘excessive sleepiness’ as defined by DVLA</td>
<td>Stop driving, inform DVLA. Can resume once condition controlled, symptoms improved and adherent to treatment. Medical confirmation required for licensing.</td>
</tr>
</tbody>
</table>

Group 1: 3 yearly review  
Group 2: annual review
Obesity hypoventilation syndrome (OHS) is thought to occur secondary to complex interactions between obesity-related changes in the respiratory system, alterations in respiratory drive, and breathing abnormalities during sleep. Box 3 shows the criteria for diagnosis of OHS.

Presentation
An acute medicine registrar reviews a 48-year-old man who is in the emergency department following a 3-day history of shortness of breath and a cough. His family has noticed that he has become more drowsy over the last 24 hours. He has a background of type 2 diabetes, a BMI of 52 kg/m², and is an ex-smoker (5 pack-year history). He mentions that he has had been feeling more sleepy than usual, and has been getting morning headaches over the last couple of months, although these tend to clear by midday. His bloods and chest X-ray are normal.

The registrar is concerned that the patient’s morning headaches and drowsiness indicate hypercapnia and checks an arterial blood gas on air. The results are as follows: pH 7.29, pCO₂ 8.0 kPa, pO₂ 7.3 kPa, SaO₂ 89 %, bicarbonate 38 mmol/L.

As the patient is an ex-smoker and is short of breath with a cough, the registrar wonders whether he might have an underlying diagnosis of COPD and manages him as such. After one hour of

Box 3. Diagnosis of OHS

A diagnosis of OHS can be made when, in the absence of other causes of hypventilation, a person has:

- Obesity (a BMI of 30 kg/m² or more) and
- Raised arterial or arterialised capillary carbon dioxide (CO₂) levels when awake, and
- Breathing abnormalities during sleep, either
  - hypoventilation (occurs in around 10% of patient with OHS)
  - OSAHS or a combination of OSAHS and hypventilation (occurs in around 90% of patient with OHS).

OHS can be seen as a diagnosis of exclusion, as other possible causes of hypventilation, such as COPD, kyphoscoliosis, neuropathic and myopathic conditions, and central causes such as cerebrovascular accidents need to be excluded. Factors that exacerbate hypventilation such as sedative medication or electrolyte imbalance also need to be reviewed.
Obstructive sleep apnoea/hypopnoea syndrome and obesity hypoventilation syndrome can be used if results are negative/inconclusive and symptoms are significant.

Measurement of venous bicarbonate. This can be used as a method of excluding OHS where there is a low suspicion of OHS. If bicarbonate levels are below 27 mmol/litre, OHS is unlikely, as serum bicarbonate <27 mmol/litre has a 97% negative predictive value for excluding a diagnosis of OHS.

Blood gas. This can be used to diagnose OHS and to assess extent of chronic ventilatory failure.

Chest X-ray, CT chest and/or spirometry. These can be used to exclude other causes of hypoventilation if suspected.

Echocardiogram. OHS patients are more likely to have pulmonary hypertension than OSAHS patients (50% vs 15%) and for it to be more severe than in those with OSAHS.

Following the patient’s admission with acute hypercapnic respiratory failure managed with acute NIV, the patient is reviewed in a respiratory follow up clinic 4 weeks following his recovery with the following results and a blood gas.

His spirometry is normal. The blood gas results are as follows: pH 7.36, pCO₂ 7.2 kPa, pO₂ 9.5 kPa, and bicarbonate 35 mmol/L, SaO₂ 91%.

In view of the persistent hypercapnia, he is referred to the sleep and ventilation service for consideration of home NIV. An ESS is 11/24 consistent with mild excessive daytime sleepiness although he does not have any excessive sleepiness when driving. His BMI remains 52 kg/m². OHS is suspected.

An outpatient respiratory polygraphy sleep study is requested. When referring suspected OHS patients to the sleep service, your referral letter should contain the same information as recommended for OSAHS patients, as well as any history of acute admissions requiring non-invasive ventilation.

This patient’s sleep study results (Fig 2) show AHI 7/h, ODI 9/h, SpO₂ <90% for 40% of the study and average oxygen saturations 86%. Three episodes of prolonged hypoxia are noted, suggestive of nocturnal hypoventilation.

Optimal medical treatment, the arterial blood gas has not improved, and NIV therapy for acute hypercapnic respiratory failure is started. The patient is successfully weaned off NIV, clinically improves, and is discharged a few days later with outpatient spirometry requested and a general respiratory follow up appointment arranged.

OHS is a specific form of chronic ventilatory failure. In patients who are undiagnosed, as respiratory pump failure is often insidious in its onset, acute hypercapnic respiratory failure—for example, if there is an intercurrent respiratory tract infection—may be unexpected. As people with OHS are more likely than those with OSAHS to have episodes of acute hypercapnic respiratory failure, they may be misdiagnosed as having COPD. Therefore, emergency and acute physicians need to be aware of the possibility of underlying OHS in patients with obesity who present to the emergency department with a first episode of acute ventilatory failure. Further investigations for suspected OHS can occur once the patient is stable.

Patients can also present with symptoms of excessive sleepiness, dyspnoea, morning headaches and right heart failure and may be polycythaemic. Lack of recognition of OHS also means a diagnosis of OHS could be overlooked in patients suspected of having (or diagnosed with) OSAHS.

Follow up and further assessment

When OHS is suspected, best practice guidance in assessment of OHS includes the following:

Checking assessment scores. The Epworth Sleepiness Scale should be used.

Sleep studies. Home respiratory polygraphy should be carried out if available. Consider adding transcutaneous CO₂ monitoring during sleep to help establish the severity of nocturnal hypoventilation—this will need hospital admission as home CO₂ monitoring is not widely available.

Polysomnography can be used if results are negative/inconclusive and symptoms are significant.

Blood gas. This can be used to diagnose OHS and to assess extent of chronic ventilatory failure.

Chest X-ray, CT chest and/or spirometry. These can be used to exclude other causes of hypoventilation if suspected.

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**Fig 2.** Area of sleep study showing a prolonged episode of hypoxia followed by a hypopnoea.
As the patient has a BMI ≥ 30 mg/kg², daytime hypercapnia, hypoventilation with mild OSAHS, and no other clear cause for his hypoventilation, he meets the diagnostic criteria for OHS. As there are different clinical phenotypes in OHS, diagnosing the presence of OSAHS and/or nocturnal hypoventilation in OHS is important in directing optimal management.³

Management
The key management steps in OHS are obesity management and ventilatory support. A multidisciplinary approach should be taken.

Ventilation support
Treatment of OHS with either CPAP or NIV, depending on the clinical phenotype, has been shown to improve gas exchange, as well as improve symptoms of sleepiness and dyspnoea, physical activity and health-related quality of life (HRQoL).³⁰ The ventilatory therapy is given and the response monitored and can be changed as necessary.

CPAP is the first-line treatment for people with OHS with severe OSAHS, while non-invasive ventilation should be considered for people with OHS and nocturnal hypoventilation who do not have OSAHS, or in whom OSAHS is not severe.³⁸,³¹ Of note, it has been shown that in newly diagnosed severe OHS, NIV and CPAP resulted in similar improvements in ventilatory failure, health-related quality of life and adherence. Baseline PaCO₂ predicted persistent ventilatory failure on treatment.³⁰ A markedly raised pCO₂ level suggests NIV rather than CPAP is likely be the treatment of choice.³ Long-term studies are needed to determine whether these treatments have different cost-effectiveness or impact on mortality. NIV can be set up as an inpatient, and in certain cases—as such as stable OHS with an auto titrating NIV device—as an outpatient. Outpatient set up requires active monitoring; however, it has been shown to be safe and effective.³²

In this case, as the patient has predominantly hypoventilation and has a recent presentation in acute hypercapnic respiratory failure, he is started on NIV. Telemonitoring is used to assess his adherence, residual AHI and any mask leak. An oximetry on his NIV therapy is checked to assess adequate correction of his hypoxia.

Once established on treatment, patients should ideally be monitored using telemonitoring.

Weight management
As the fundamental process underpinning the pathophysiology of OHS is obesity, management of obesity is key.³⁵ This may involve referral to an obesity/metabolic clinic for specialist support, including dietary and psychological support as well as consideration of further medical management of obesity, such as starting a GLP-1 agonist, for example semaglutide, or referral for bariatric surgery.

Follow up
In patients initiated on CPAP who have an inadequate response, such as persistence of symptoms, severe hypercapnia, and insufficient reduction of AHI and ODI, it is clinically prudent to change to NIV therapy.³ In patients started on NIV, if hypercapnia resolves, in some cases, NIV can be changed to CPAP.

If oximetry on treatment reveals that the person remains hypoxic during sleep despite control of their AHI and nocturnal hypercapnia on CPAP or NIV, entrained oxygen therapy will need to be given via the ventilator to correct this hypoxia. Following significant weight loss, a sleep study should be repeated.

COPD–OSAHS overlap syndrome
It is estimated that COPD–OSAHS overlap syndrome, describing the combination of COPD and OSAHS, has a prevalence of approximately 1%, although it is currently under recognised. With increased recognition, increased use of CPAP and NIV is expected. Treatment could reduce acute admissions and long-term complications.³

Future considerations
OHS classification
There is currently no widely used severity scale for OHS. With prevalence increasing, and with increasing focus on how to allocate healthcare resources, classifying OHS according to severity and potential outcomes could aid the targeting of treatments to those who would most benefit. Existing classification systems have not been tested in large-scale prospective studies.⁸,²⁵

Further work is also needed to demonstrate the extent to which serum bicarbonate can be used as both a screening tool for OHS and as a biomarker to monitor the effectiveness of treatment.⁸

Health economics
Investing in the identification and treatment of OSAHS and OHS could mitigate the long-term costs of untreated sleep disorders on the NHS budget. These costs are due to increased rates of strokes, cardiovascular events and road traffic accidents. The Obstructive sleep apnoea health economics report estimated that treating everyone with moderate to severe OSAHS could double the amount of cost savings to the NHS.⁵

Conclusion
The incidence of OSAHS and OHS is increasing with increasing obesity and life expectancy. Early recognition is key to improve patients’ symptoms and quality of life at an earlier stage, and to reduce the disease burden of untreated disease. Treatment is effective and cost effective.³⁵

Best practice in ongoing care is multidisciplinary and includes supportive interventions tailored to each person’s needs and preferences, with the aim of supporting and improving adherence with therapies and lifestyle modifications, in addition to understanding of their condition.

Key points
- There is a higher prevalence of sleep disorders in those with obesity.
- Early recognition is key. It is important to be aware of the symptoms related to OSAHS and OHS, the use of screening questionnaires, and to ask about sleepiness while driving and occupational risk.
- Some people with OSAHS do not have excessive sleepiness.
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> OHS is increasingly common, and the most common reason for home NIV.
> OHS is under-recognised, may be misdiagnosed as OSAHS or COPD, and when untreated is associated with poorer outcomes than OSAHS.
> Weight loss is fundamental in the treatment of sleep disordered breathing; weight loss of just 10–15% can reduce the severity of OSAHS by 50% in moderately obese patients.

References


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