# Obstructive Sleep Apnoea & Obesity Hypoventilation Syndrome

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Based on NICE NG202 • BTS guidance/position statements • ERS evidence

# 1. Learning objectives

Recognise, investigate and manage OSAHS and OHS safely in respiratory practice.

# Learning objectives

- Define OSAHS and OHS and distinguish airway obstruction from hypoventilation.
- Explain the physiology of upper-airway collapse, obesity-related respiratory mechanics and chronic hypercapnia.
- Interpret AHI/ODI, oximetry, respiratory polygraphy and $CO_2$ measurements.
- Select CPAP versus NIV using the OSA/OHS phenotype and clinical context.
- Manage acute-on-chronic hypercapnic respiratory failure and avoid common pitfalls.
- Discuss alternatives to PAP, weight-loss strategies, adherence and expected health benefits.

# 2. Obstructive sleep apnoea

A common sleep-related upper-airway disorder with important respiratory, cardiovascular and safety consequences.

# What is OSAHS?

- Recurrent narrowing or closure of the upper airway during sleep → hypopnoea and/or apnoea.
- Obstructive events occur despite ongoing respiratory effort.
- Events cause intermittent hypoxaemia, hypercapnia, arousals and sleep fragmentation.
- Clinical consequences include sleepiness, impaired vigilance/cognition, poor quality of life and cardiovascular risk.
- Risk is increased with obesity, male sex, increasing age and craniofacial/upper-airway factors.

# Physiology: why does the airway collapse?

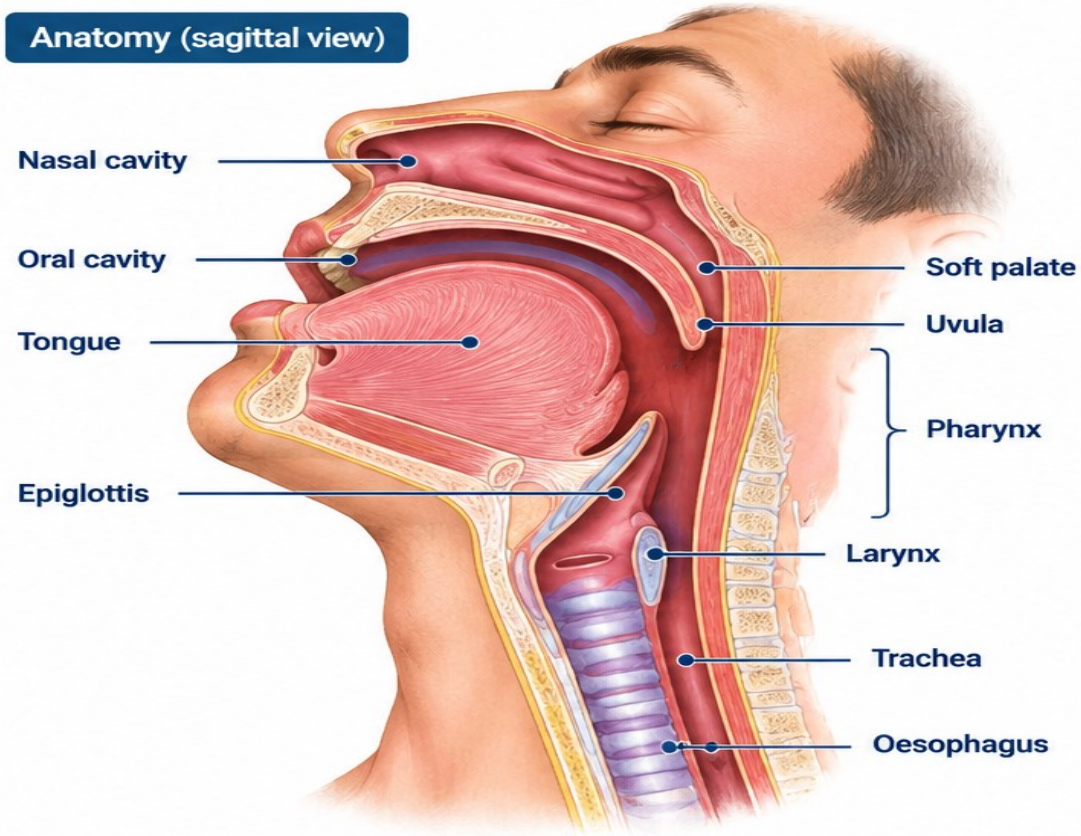
- Sleep reduces upper-airway dilator muscle activity and protective arousal responses.
- Pharyngeal collapsibility depends on anatomy, neuromuscular compensation and ventilatory control stability.
- Obesity increases peripharyngeal soft tissue and reduces functional residual capacity (FRC).
- Reduced lung volume decreases caudal traction on the upper airway → greater collapsibility.
- Obstructive event → negative intrathoracic pressure + intermittent hypoxaemia/hypercapnia → arousal → airway reopening.
- Repeated cycles produce sympathetic activation and fragmented sleep.

# 1. Anatomy of obstructive sleep apnoea

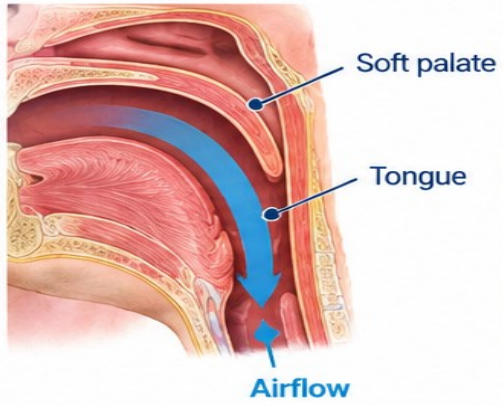
## Obstructive Sleep Apnoea (OSA) – Anatomy

During sleep, the upper airway can become narrowed or blocked due to relaxation of the muscles and soft tissues, leading to airflow limitation or complete obstruction.

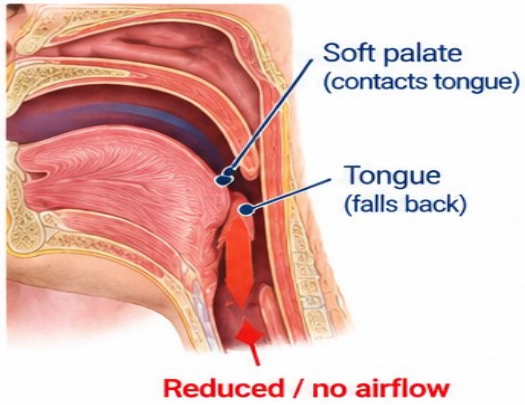
**Anatomy (sagittal view)**




**1. Awake – open airway**




**2. Sleeping – obstructed airway**




### Key points

- The upper airway is kept open by muscle tone and the position of the tongue, soft palate and surrounding structures.
- During sleep, reduced muscle tone can lead to airway collapse, especially at the level of the soft palate and tongue.
- This causes intermittent airflow limitation or complete obstruction (apnoea), leading to oxygen desaturation and sleep fragmentation.

Illustration for educational purposes – based on NICE NG202, BTS and ERS guidelines. Anatomy alone does not diagnose OSA.

Educational illustration generated for this presentation. NICE NG202: diagnosis requires appropriate sleep testing.

# Clinical presentation & when to suspect OSAHS

- Ask about: loud snoring, witnessed apnoeas, choking/gasping, unrefreshing sleep, morning headache, nocturia, fragmented sleep.
- Daytime: excessive sleepiness, fatigue, poor concentration, memory/cognitive dysfunction.
- NICE: assess for OSAHS when ≥2 characteristic symptoms/features are present.
- Think beyond sleepiness: not all patients with OSAHS are sleepy.
- High-risk groups: obesity, resistant hypertension, T2DM, AF, stroke/TIA, heart failure, PCOS, hypothyroidism, acromegaly.

# Assessment: ESS, STOP-Bang and clinical risk

- Epworth Sleepiness Scale (ESS): useful measure of subjective sleepiness; do not use alone to decide referral.
- STOP-Bang can help estimate OSA risk, particularly in higher-risk populations.
- Record BMI, neck circumference, blood pressure, cardiovascular disease and occupational/driving risk.
- Look for alternative explanations of sleepiness: insufficient sleep, drugs/alcohol, depression, circadian disorder, anaemia, hypothyroidism and other sleep disorders.
- Examine upper airway and craniofacial anatomy when relevant.

# Diagnosis: sleep testing

- Home respiratory polygraphy is recommended by NICE for suspected OSAHS in appropriate adults.
- Typical channels: airflow, respiratory effort, oxygen saturation, heart rate ± snoring/body position.
- Polysomnography adds EEG/EOG/EMG → measures sleep time and sleep architecture; useful when diagnosis is uncertain or another sleep disorder is suspected.
- Oximetry alone may support triage but can miss arousal-related events and cannot reliably phenotype OSA.
- Interpret results alongside symptoms, comorbidities and technical quality.

# AHI/ODI Index

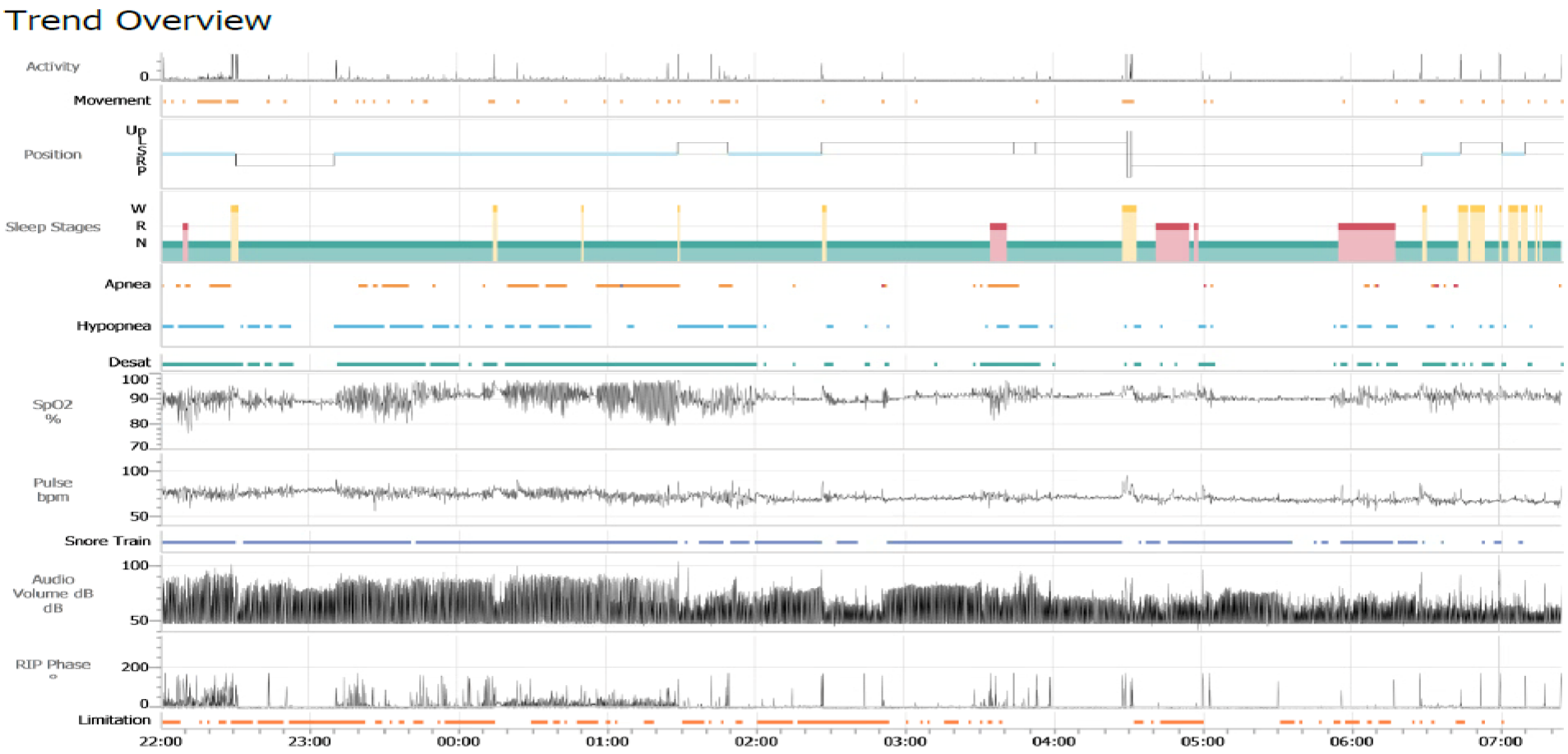
- **1. AHI – Apnoea–Hypopnoea Index**
- The **AHI** is the number of **apnoeas + hypopnoeas per hour of sleep**.
- **Apnoea:** near-complete/complete cessation of airflow for **≥10 seconds**.
- **Hypopnoea:** significant reduction in airflow, usually associated with oxygen desaturation and/or an arousal.
- **AHI = total apnoeas + hypopnoeas ÷ hours of sleep**

- **2. ODI – Oxygen Desaturation Index**
- The **ODI** is the number of **oxygen desaturations per hour**, usually expressed as events/hour.
- For example, **ODI-4% = 12/hour** means the patient's oxygen saturation fell by **≥4% from baseline on average 12 times per hour**.
- Common definitions:
- **ODI-3%:** ≥3% fall in $SpO_2$
- **ODI-4%:** ≥4% fall in $SpO_2$

# OSAHS severity: AHI and ODI

- Mild: AHI ≥5 to <15 events/hour.
- Moderate: AHI ≥15 to <30 events/hour.
- Severe: AHI ≥30 events/hour.
- AHI = apnoeas + hypopnoeas per hour of sleep (or recording time depending on test methodology).
- ODI = number of oxygen desaturations per hour; useful when sleep time is not available.
- Severity is not determined by AHI alone: symptoms, hypoxaemia, comorbidity and safety risk matter.

# Sleep study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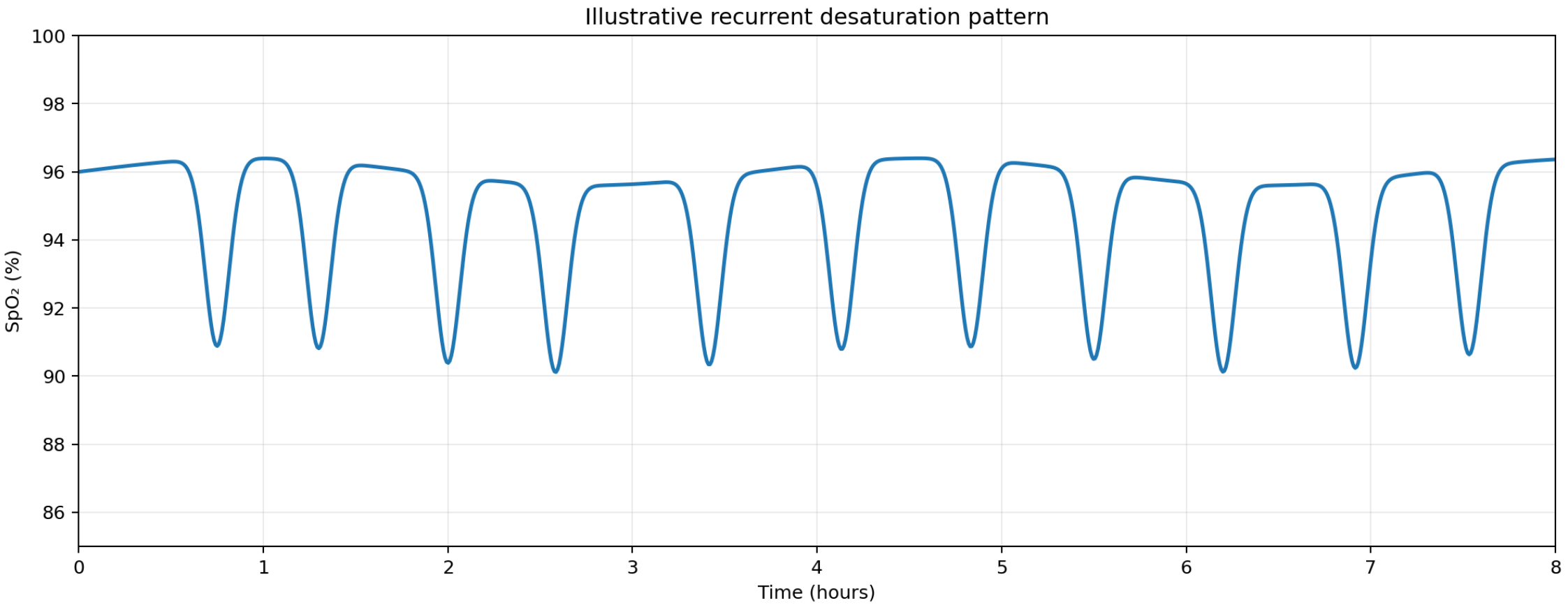
# 3. Example of overnight pulse oximetry




Look for repetitive desaturations, baseline $SpO_2$, artefact and clustering. Oximetry does not measure airflow and cannot by itself distinguish OSA from other causes of nocturnal hypoxaemia.

# Treatment goals in OSAHS

- Reduce symptoms—especially excessive daytime sleepiness—and improve quality of life.
- Control respiratory events and oxygen desaturation.
- Improve sleep continuity and daytime function.
- Reduce risks associated with untreated disease, including hypertension and road-traffic/occupational accidents.
- Address modifiable contributors: weight, alcohol, smoking, sleep hygiene and sedatives.
- Use shared decision-making and focus on long-term adherence.

# CPAP: first-line treatment

- Moderate/severe symptomatic OSAHS: offer fixed-level CPAP plus lifestyle advice.
- Auto-CPAP can be considered in selected patients—e.g. variable pressure requirement or intolerance of fixed CPAP.
- Before declaring failure: check mask fit/leak, pressure, nasal obstruction, humidification, usage pattern and patient understanding.
- Telemonitoring should be offered with CPAP for up to 12 months; consider longer use where helpful.
- Early review: NICE recommends initial follow-up within 1 month, then until symptoms and AHI/ODI are optimised.

# Mild OSAHS: individualise treatment

- Mild OSAHS without troublesome daytime symptoms: treatment is often unnecessary; lifestyle/sleep-habit measures may be sufficient.
- Mild symptomatic disease affecting daytime activities: fixed CPAP may be offered alongside lifestyle advice when indicated.
- If CPAP is declined or not tolerated: consider a customised/semi-customised mandibular advancement splint in suitable adults with good dental health.
- Positional therapy can be considered for mild/moderate positional OSAHS when other options are unsuitable.
- Severe OSAHS is unlikely to respond adequately to positional therapy alone.

# Alternatives to CPAP in OSAHS

- Mandibular advancement splint (MAD): particularly useful when CPAP is declined or poorly tolerated; dental assessment is important.
- Positional therapy: selected positional OSAHS, mainly mild/moderate.
- Weight management: should be discussed with all patients; meaningful weight loss can reduce OSA severity.
- Upper-airway surgery: specialist assessment in selected severe OSAHS after failure/intolerance of CPAP and MAD.
- Treat nasal disease and other contributors; heated humidification can improve upper-airway side effects.
- Emerging pharmacological/weight-loss therapies may reduce OSA burden through weight reduction, but do not replace established PAP indications.

# CPAP troubleshooting: practical approach

- Poor adherence → ask what prevents use rather than simply increasing pressure.
- Mask leak: refit/change interface; assess mouth leak and consider humidification or alternative interface.
- Nasal blockage/rhinitis: saline, topical therapy when appropriate, humidification; investigate persistent obstruction.
- Claustrophobia/anxiety: desensitisation, gradual exposure and supervised acclimatisation.
- Residual AHI: assess leak, positional dependence, central events, pressure adequacy and sleep duration.
- Persistent sleepiness despite good control → reassess adherence, sleep duration, medicines, mood and alternative diagnoses.

# 3. Obesity hypoventilation syndrome

Think OHS whenever obesity and unexplained daytime hypercapnia coexist.

# Definition of OHS

- Obesity: BMI ≥30 kg/m².
- Awake daytime hypercapnia: $PaCO_2$ ≥6.0 kPa (45 mmHg) at sea level.
- Sleep-disordered breathing/hypoventilation is usually present.
- Diagnosis requires exclusion of alternative causes of hypoventilation: COPD/other lung disease, neuromuscular weakness, chest-wall disease, severe hypothyroidism, drugs/opioids and congenital/central disorders.
- OHS is chronic ventilatory failure—not simply severe OSA.

# OHS physiology: the 'perfect storm'

- Obesity increases chest-wall and abdominal mass → reduced compliance and FRC.
- Reduced FRC promotes airway closure and V/Q mismatch, especially supine.
- Work of breathing rises; respiratory muscle efficiency may fall.
- Leptin resistance and impaired ventilatory responsiveness may reduce ventilatory drive.
- Sleep-related hypoventilation and recurrent obstruction add nocturnal $CO_2$ retention.
- Over time: renal bicarbonate retention buffers chronic respiratory acidosis → persistent daytime hypercapnia.
- Pulmonary hypertension, right-heart strain and metabolic/cardiovascular disease are common comorbidities.

# When should I suspect OHS?

- BMI ≥30 kg/m² plus OSA symptoms or features of nocturnal hypoventilation.
- Red flags: waking headache, resting hypoxaemia (e.g. $SpO_2$ <94% on air), peripheral oedema, polycythaemia, unexplained dyspnoea.
- Think OHS in recurrent admissions with acute-on-chronic hypercapnic respiratory failure.
- Severe hypercapnia, hypoxaemia, unstable cardiovascular disease or vocational driving → prioritise rapid sleep/ventilation assessment.
- Do not assume all hypercapnia in obesity is 'just COPD' or 'just OSA'.

# Diagnosing OHS: a practical algorithm

1. BMI ≥30 kg/m² + clinical suspicion.
2. If pre-test probability is low: serum venous bicarbonate can be used as a screening test.
3. Bicarbonate <27 mmol/L makes OHS unlikely in low-risk patients.
4. Confirm with awake arterial or arterialised capillary blood gas: $PaCO_2$ ≥6.0 kPa.
5. Exclude alternative causes of chronic hypercapnia.
6. Perform respiratory polygraphy; add transcutaneous $CO_2$ monitoring when available to define nocturnal hypoventilation.
7. Do not use oximetry alone to diagnose OSAHS in suspected OHS.

# OHS: CPAP or NIV?

- Stable OHS + severe OSAHS → CPAP first-line.
- Stable OHS + no OSAHS or non-severe OSAHS with nocturnal hypoventilation → consider NIV first-line.
- CPAP failure → switch/consider NIV if symptoms persist, hypercapnia persists, AHI/ODI remains inadequately controlled or CPAP is poorly tolerated.
- Do not label CPAP failure until adherence and technical issues have been objectively assessed.
- Both CPAP and NIV improve symptoms and gas exchange; NIV provides ventilatory assistance and is particularly useful when hypoventilation predominates.

# NIV in OHS: what are we trying to achieve?

- EPAP: splints the upper airway open and treats obstructive events.
- Pressure support (IPAP − EPAP): increases alveolar ventilation and reduces $CO_2$.
- Use a backup rate when clinically indicated by the NIV strategy/device and patient phenotype.
- Monitor symptoms, overnight oxygenation and $CO_2$, daytime ABG and device downloads.
- OHS often requires relatively high pressures; volume-assured modes may be useful in selected patients, particularly when high pressure requirements are difficult to manage.
- Optimise mask/interface, leak and synchrony before escalating complexity.

# Acute-on-chronic hypercapnic respiratory failure in OHS

- Recognise: obesity + acute deterioration + respiratory acidosis/hypercapnia, often with hypoxaemia.
- Treat precipitant: infection, fluid overload, sedatives/opioids, cardiac failure, PE or other acute pathology.
- Controlled oxygen—avoid excessive oxygen in hypercapnic respiratory failure.
- Start NIV promptly when acute ventilatory failure is present; do not delay for sleep-study investigations.
- Commonly high IPAP/EPAP requirements; monitor closely for NIV response, leak, aspiration risk and fluid overload.
- After stabilisation: reassess whether CPAP is appropriate (especially if severe OSA predominates) or whether ongoing home NIV is needed.

# Weight management: the disease-modifying intervention

Weight reduction should be part of OHS treatment for every patient.

Target should be individualised and delivered through structured obesity-management pathways.

Weight loss improves respiratory mechanics, OSA severity and gas exchange; substantial sustained loss may reverse OHS in some patients.

Consider anti-obesity pharmacotherapy where appropriate under current obesity-management guidance.

Bariatric/metabolic surgery can produce major and durable weight loss and may substantially improve OSA/OHS, but perioperative respiratory risk requires specialist planning.

PAP should not be withheld while weight-loss treatment is being pursued.

# Health benefits: what can treatment change?

CPAP/NIV: rapid improvement in nocturnal obstruction/hypoventilation and daytime symptoms when adherence is good.

Sleepiness, vigilance and quality of life generally improve substantially with effective CPAP.

Blood pressure: CPAP produces modest average reductions, with greater effects in some hypertensive/high-risk phenotypes.

OHS: PAP improves daytime $PaCO_2$, oxygenation and sleep-disordered breathing; pulmonary hypertension may improve.

Weight loss: improves OSA severity, metabolic health and respiratory mechanics; larger sustained losses provide the greatest chance of OHS remission.

Do not promise cardiovascular mortality benefit from CPAP alone—the evidence for hard cardiovascular outcomes is more nuanced than the symptomatic benefit.

# Follow-up: what should I check?

Symptoms: ESS, sleep quality, morning headaches, daytime function and driving safety.

Objective control: residual AHI/ODI, leak, pressure profile, usage hours and overnight oxygenation/$CO_2$ when relevant.

OHS: repeat daytime ABG or $CO_2$ assessment after PAP optimisation; track bicarbonate where useful.

Adherence: nights used, hours/night, barriers and equipment problems.

Weight/BMI, smoking, alcohol, sedatives/opioids and cardiovascular comorbidities.

Once stable on CPAP, NICE suggests considering annual follow-up; complex OHS/NIV patients usually need individualised respiratory follow-up.

# Driving & occupational safety

Ask specifically about sleepiness while driving, near-misses and accidents.

Occupational risk matters: commercial/vocational drivers and safety-critical workers need rapid assessment.

Advise patients not to drive if they are excessively sleepy or unsafe to drive.

Follow current DVLA guidance and document advice/notification requirements.

Effective PAP treatment should reduce sleepiness, but driving advice depends on clinical control and the individual's circumstances.

# 4. Putting it together

A phenotype-based approach for clinic and acute respiratory practice.

# OSAHS vs OHS: high-yield comparison

OSAHS: upper-airway obstruction during sleep; daytime $PaCO_2$ usually normal.

OHS: obesity + awake daytime hypercapnia + sleep-disordered breathing, after excluding other causes.

Severe OSA is common in OHS, but OHS can occur with absent/non-severe OSA.

OSAHS → CPAP is the mainstay.

OHS + severe OSA → CPAP first-line when stable.

OHS without severe OSA or with predominant hypoventilation → NIV is generally preferred.

OHS with acute ventilatory failure → NIV.

# 6. OSA vs OHS: why $CO_2$ matters

## OSAHS

- Upper-airway obstruction during sleep
- Daytime $PaCO_2$ usually normal
- CPAP is the main treatment for moderate/severe OSAHS

## OHS

- BMI ≥30 kg/m² + awake $PaCO_2$ ≥6.0 kPa
- Exclude alternative causes of hypoventilation
- Severe OSA → CPAP; predominant hypoventilation → NIV

NICE NG202; ERS evidence on OHS.

# Case 1 — severe OSAHS

52-year-old man, BMI 36 kg/m², loud snoring and witnessed apnoeas; ESS 16.

Respiratory polygraphy: AHI 48/h, ODI 42/h. Daytime $SpO_2$ 96%, $PaCO_2$ 5.2 kPa.

Diagnosis: severe OSAHS without daytime hypercapnia.

Management: lifestyle/weight intervention + fixed CPAP; discuss telemonitoring and early troubleshooting.

Ask about driving and occupational risk.

At follow-up: review adherence, leak, residual AHI/ODI and sleepiness.

# Case 2 — OHS phenotype

58-year-old woman, BMI 49 kg/m², morning headaches, oedema and daytime $SpO_2$ 91%.

ABG: pH 7.37, $PaCO_2$ 7.2 kPa, $HCO_3^-$ 32 mmol/L.

Polygraphy: AHI 12/h with sustained nocturnal desaturation and $CO_2$ rise.

Diagnosis: OHS with non-severe OSA and predominant hypoventilation.

Management: NIV + structured weight-management programme; assess pulmonary hypertension/cardiac comorbidity.

Do not treat nocturnal desaturation with oxygen alone without addressing hypoventilation.

# Common trainee pitfalls

❌ Diagnosing OHS from obesity + nocturnal desaturation alone.

❌ Using ESS to rule out OSA/OHS.

❌ Assuming all obese patients with daytime hypercapnia have OHS without excluding COPD, neuromuscular, chest-wall, drug and endocrine causes.

❌ Giving oxygen alone to a hypoventilating patient without assessing ventilatory support.

❌ Calling CPAP failure before checking adherence, leak, mask/interface and pressure.

❌ Forgetting driving/occupational safety.

❌ Treating PAP as the whole treatment—weight management and comorbidity optimisation remain essential.

# Take-home messages

1. OSAHS = sleep-related upper-airway obstruction; OHS = obesity + awake daytime hypercapnia + sleep-disordered breathing after exclusion of other causes.
2. AHI ≥30/h defines severe OSAHS; symptoms and clinical risk still matter.
3. CPAP is first-line for moderate/severe OSAHS and stable OHS with severe OSA.
4. NIV is preferred for OHS with predominant hypoventilation/non-severe OSA and is first-line in acute ventilatory failure.
5. Weight management is disease-modifying and should run alongside PAP.
6. Objective monitoring + adherence support determine real-world success.
7. Always address driving safety, cardiovascular/metabolic disease and long-term follow-up.

# Key references & further reading

NICE NG202 (2021): Obstructive sleep apnoea/hypopnoea syndrome and obesity hypoventilation syndrome in over 16s.

European Respiratory Society: Obesity hypoventilation syndrome — definition, pathophysiology and management review.

BTS/ICS guideline: Ventilatory management of acute hypercapnic respiratory failure in adults — OHS recommendations.

BTS: Sleep Apnoea resources and Position Statement on Driving and Obstructive Sleep Apnoea.

NICE TA139: Continuous positive airway pressure for OSAHS.

Always check the latest local sleep/NIV pathway, device policy and current DVLA guidance before clinical implementation.