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1. I do not have any potential conflicts to disclose.

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2. I wish to disclose the following potential conflicts of interest:

Type of Potential Conflict	Details of Potential Conflict
Grant/Research Support	
Consultant	Inspire, Idorsia, Resmed
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ESRS EXAMINATION PREPARATION SERIES • PART 1 OF 2

CPAP Therapy for Obstructive Sleep Apnoea

Evidence, indications and guideline-based practice: NICE • ATS • ERS

A knowledge-focused review for candidates sitting the ESRS examination. Practical technique, adherence coaching, ASV and NIV are covered in the companion workshop.

Learning Objectives

1	Physiological principles	Explain the evidence base for CPAP as a pneumatic splint, and its effects beyond the upper airway
2	Indications	Apply NICE NG202, ATS and ERS/AASM criteria for who should be offered CPAP, including mild disease
3	Adequate use	Define adequate CPAP use and its evidence-based dose-response relationship
4	Clinical outcomes	Critically appraise landmark cohort and outcome trials — Marin, MERGE, SAVE, RICCADSA, Xu et al. — and formal society recommendations
5	Special populations	Summarise OHS, COPD–OSA overlap, and phenotype-based approaches to predicting treatment benefit

Why CPAP Works: The Pathophysiological Rationale

The problem: airway collapse

- Repetitive collapse of the pharyngeal airway during sleep, driven by loss of dilator muscle tone, anatomical narrowing, and unfavourable loop gain / arousal threshold
- Produces apnoeas, hypopnoeas and flow limitation → intermittent hypoxia, arousals, sympathetic activation
- Clinical consequences: fragmented sleep, excessive daytime sleepiness, and elevated cardiometabolic risk

The fix: pneumatic splinting

- CPAP delivers continuous positive pressure to the upper airway via a mask interface
- Acts as a pneumatic splint, increasing intraluminal (transmural) pressure so the airway resists collapse throughout the respiratory cycle
- Passive collapse is prevented once intraluminal pressure exceeds the airway's critical closing pressure
- Result: elimination or major reduction of apnoeas/hypopnoeas, restoration of sleep architecture, and reduction in AHI toward normal

The Physiological Signature of Obstruction

Recognising the pattern on airflow and thoraco-abdominal traces is core physiology, and shows precisely what CPAP corrects.

Patent airway: in-phase movement

- Ribcage and abdomen rise and fall together (synchronously) with each breath
- Airflow trace shows a regular, unobstructed rhythm
- Reflects unimpeded airway patency and normal respiratory mechanics

Obstruction: paradoxical movement

- Ribcage and abdomen move out of phase (paradoxically) as respiratory effort continues against a closed airway
- Airflow falls or ceases despite ongoing effort — the hallmark of an OBSTRUCTIVE (not central) event
- Event is typically terminated by a cortical arousal, restoring airway patency and airflow

Where the Evidence Comes From

Sullivan, Issa, Berthon-Jones & Eves (1981)

- The original description of CPAP: nasal pressure of 4.5–10 cmH₂O abolished sleep-related upper-airway collapse in 5 patients with severe OSA
- Established that increased intraluminal pressure acting as a pneumatic splint is the key physiological effect of nasal CPAP

Schwab et al. (1996) — imaging the effect

- Upper-airway imaging shows that increasing nasal CPAP progressively enlarges the pharyngeal airway
- The enlargement is more pronounced in the latero-lateral diameter than the antero-posterior diameter

Beyond the Airway: Delivery Route and Systemic Effects

Why nasal delivery is preferred

- Nasal pressure delivery exerts a direct stabilising effect on the velopharynx (retropalatal segment) — a key site in OSA pathogenesis
- Oral or oronasal delivery may be less potent, or even destabilise the velopharyngeal airway, risking persistence of respiratory events despite treatment (Ebben 2012)
- Persistent obstructive events under PAP can compromise adherence (Borel 2013) — so nasal masks are preferred first-line, with full-face masks as second choice if nasal fails

Systemic effects beyond splinting

- Increased intrapulmonary pressure raises end-expiratory lung volume (functional residual capacity)
- This may exert a caudal pull on the central airways, further reducing upper-airway collapsibility
- Other proposed effects: decreased work of breathing, improved stability of central respiratory drive, and improved cardiac function via reduced preload and afterload
- These cardiac mechanisms matter most in CHF and CSA-Cheyne–Stokes patients — covered in depth in the Part 2 workshop

Before CPAP: General Measures for Mild OSA

General measures — such as dietary intervention and abstaining from alcohol or sedatives — should be considered before starting a specific therapy such as CPAP

Measures that improve OSA itself

- Weight loss — reduces upper-airway fat loading and can lower AHI
- Reduced alcohol intake — alcohol lowers dilator muscle tone and arousal threshold
- Improved nasal patency — reduces upstream resistance and mouth breathing
- Positional therapy — avoiding supine sleep in position-dependent OSA

General sleep hygiene measures

- Reduce caffeine, alcohol and nicotine near bedtime
- Keep a regular sleep–wake schedule and avoid daytime napping
- Wind down with a relaxing pre-bed routine; avoid screens in bed
- Optimise the sleep environment — temperature, noise and light

NICE NG202: Indications for CPAP

NICE guideline NG202 (2021) updates the 2008 CPAP technology appraisal (TA139) and is organised by OSAHS severity

Mild OSAHS

- Offer fixed-level CPAP if symptoms affect quality of life or usual daytime activities
- Given alongside lifestyle advice
- Consider auto-CPAP (APAP) instead of fixed CPAP if the patient needs high pressure only at certain times, cannot tolerate fixed CPAP, or cannot use telemonitoring for technical reasons

Moderate or Severe OSAHS

- Offer fixed-level CPAP, in addition to lifestyle advice, regardless of symptom severity threshold used for mild disease
- Same auto-CPAP alternative criteria apply
- This tier carries forward the original 2008 CPAP technology appraisal recommendation

Mild OSA and CPAP: What Does the Trial Evidence Show?

MERGE trial (Wimms et al., 2019, *Lancet Respir Med*)

- Multicentre RCT of CPAP vs standard care in mild OSA (AHI 5–15/h), n = 233, followed for 3 months
- Median CPAP usage was only around 4 hours per night — a real-world, modest adherence level
- Despite this, CPAP produced statistically significant improvements across multiple SF-36 quality-of-life domains and reduced Epworth Sleepiness Scale scores by about 3 points versus no change with standard care

Why this matters for the exam

- MERGE directly countered the older assumption that mild OSA patients gain little and adhere poorly — adherence was comparable to more severe disease in several studies (Weaver, 2019)
- The ESRS textbook nuance still holds: CPAP is the mainstay for moderate-severe OSA, while oral appliances have specific evidence of benefit in mild-to-moderate disease

NICE NG202: Telemonitoring Requirements

For every patient started on CPAP

- Offer telemonitoring with CPAP for up to 12 months from initiation, across both severity tiers
- Consider continuing telemonitoring beyond 12 months where it continues to add value

Why this matters for the exam

- Telemonitoring is woven into NICE's core recommendation
- NICE's rationale leans partly on cost — telemonitoring is recommended in part because it adds little or no cost given modern device capability; outcome-trial evidence for telemonitoring itself is less settled
- The ERS statement on advanced telemedicine (e-Sleep) reaches a similar practical conclusion, but as a statement of current practice rather than a mandatory recommendation

ERS Guideline on Non-CPAP Therapies: CPAP as the Benchmark

Published 2021, updating the 2011 ERS statement on non-CPAP therapies into a full clinical guideline. Its core design compares alternative therapies AGAINST CPAP using PICO questions and GRADE methodology.

CPAP vs myofunctional therapy

Task force suggests CPAP over myofunctional therapy (conditional recommendation, low-quality evidence)

CPAP vs maxillo-mandibular osteotomy (MMO)

Task force suggests either MMO or CPAP may be used (conditional recommendation, very-low-quality evidence) — one of few areas of near-equipose

CPAP vs positional therapy

Positional therapy considered only for positional OSA; CPAP remains preferred where adherence and severity favour it

ATS: The Importance of Mask Selection

The Importance of Mask Selection on CPAP Outcomes for OSA: An Official ATS Workshop Report (2020)

Interface matters clinically

Mask choice is not a comfort afterthought — interface selection is a determinant of effective pressure delivery, leak, and downstream adherence

Nasal vs oronasal (full-face)

Oronasal masks are associated with higher effective pressure requirements and, in several studies, lower adherence than nasal interfaces or nasal pillows — a finding the AASM's 2019 PAP treatment guideline also concurs with

Individualised fitting

Structured, individualised mask fitting and early review is called for, given the direct link between fit/leak and both efficacy and comfort

Defining Adequate CPAP Use

4+

hours per night

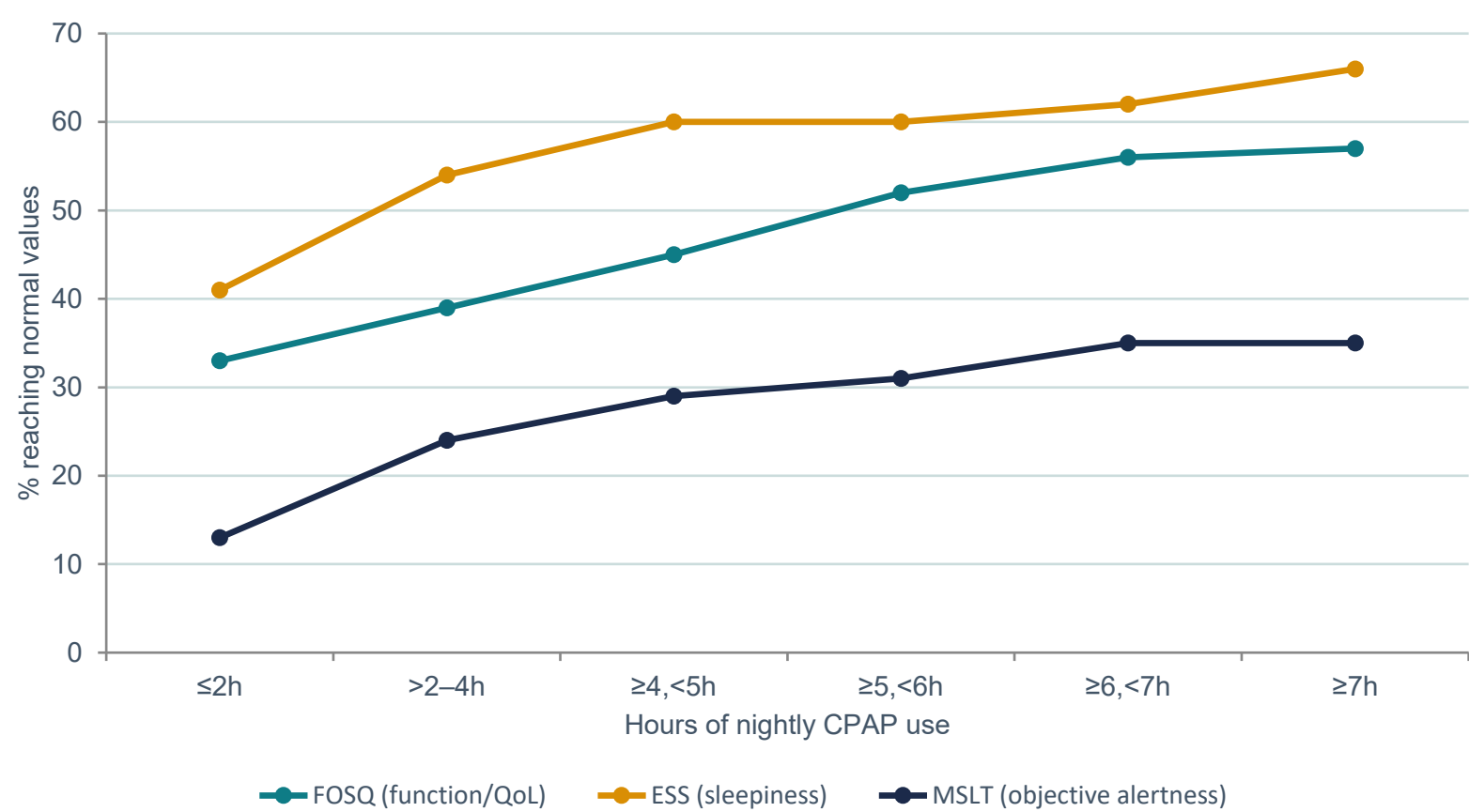
The widely used minimum threshold for “adequate” CPAP adherence, referenced across US and European sources, including the ERS/ESRS statement on driving risk in OSA

Why a single number is a simplification

- Originally derived from US Medicare reimbursement criteria (≥ 4 h/night on $\geq 70\%$ of nights over a 30-day window), not from a physiological dose-response threshold
- Dose-response data (Weaver, 2007) show benefits for sleepiness and quality of life continue to accrue with MORE than 4 hours — see the next slide
- ATS's CPAP Adherence Tracking Systems statement (2013) addresses how objective device data should define and monitor usage

How Much CPAP Is Enough? The Dose-Response Curve

Weaver et al. (Sleep, 2007) related nightly CPAP usage hours to the proportion of patients reaching NORMAL values on three outcome measures.



- Every outcome keeps improving well past 4 hours/night — there is no true efficacy “ceiling” at that figure
- Subjective measures (ESS, FOSQ) improve earlier and more steeply than objective alertness (MSLT)
- Supports counselling patients toward maximal nightly use, not just the minimum reimbursement threshold

CPAP, Sleepiness and Driving Risk

ATS: Sleep Apnea, Sleepiness, and Driving Risk (2013)

- Official ATS Clinical Practice Guideline addressing assessment and management of crash risk in noncommercial drivers with OSA
- Excessive sleepiness in OSA is a modifiable driving-risk factor — CPAP is the primary modifying intervention
- Antonopoulos et al. (2011) meta-analysis: CPAP reduces road traffic accidents and improves driving-simulator performance across pooled observational and controlled data

ERS/ESRS Statement on Sleepiness & Driving

- Addresses driver licensing for patients with OSA under the EU Directive framework introduced in 2014
- CPAP treatment prevents most OSA-related motor-vehicle collisions, associated costs and deaths
- Minimum acceptable adherence for licensing purposes: more than 4 hours of use per night
- Recommends assessing BOTH objective severity (AHI) and subjective sleepiness (ESS), since the two correlate poorly

Untreated OSA and Cardiovascular Risk

Marin et al., 2005, Lancet

- Observational cohort of 1,651 middle-aged men, mean follow-up 10.1 years
- Untreated severe OSA (AHI ≥ 30) carried significantly higher risk of fatal (OR 2.87) and non-fatal (OR 3.17) cardiovascular events vs healthy controls
- CPAP-treated severe OSA patients had event rates statistically similar to healthy controls — the foundational evidence that treatment normalises risk

AASM Clinical Practice Guideline (Patil et al., 2019)

- STRONG recommendation: use PAP (vs no therapy) for adults with excessive sleepiness
- CONDITIONAL recommendations: PAP for impaired sleep-related quality of life, and for comorbid hypertension
- The task force explicitly found insufficient/inconclusive evidence to recommend PAP specifically for cardiovascular risk or mortality reduction, or for neurocognitive benefit

CPAP and Cardiovascular Events: SAVE and RICCADSA

SAVE (McEvoy et al., 2016, NEJM)

- RCT of CPAP vs usual care for SECONDARY cardiovascular prevention in moderate-severe OSA
- Excluded severe daytime sleepiness (ESS > 15) and very severe overnight hypoxaemia (SpO2 < 80% for more than 10% of recording time)
- Mean CPAP adherence only ~3.3 hours/night
- No significant reduction in the composite cardiovascular event endpoint

RICCADSA (Peker et al., 2016, AJRCCM)

- RCT in patients with coronary artery disease and non-sleepy OSA (ESS < 10, AHI ≥ 15)
- Overall trial NEGATIVE for its primary composite cardiovascular endpoint
- A per-protocol/adherence analysis suggested benefit specifically in those using CPAP > 4 hours/night

Phenotyping Cardiovascular Risk: Beyond the AHI

Xu et al., Thorax 2023 — Hong Kong OSA cohort

- n = 1,544 patients with OSA; mean follow-up 8.3 years; cluster analysis by clinical and polysomnographic features
- 14.9% of the eligible cohort experienced a major adverse cardiovascular event (MACE) over follow-up
- Overall MACE risk was NOT predicted by AHI itself
- Percentage of sleep time with $\text{SpO}_2 < 90\%$ (TST90) and both awake and nocturnal heart rate were independent predictors of MACE
- CPAP use did not reduce MACE risk in the cohort overall — BUT a subgroup (younger, more obese, more severe OSA by AHI and hypoxic burden, more cardiovascular risk factors) showed fewer MACEs with CPAP use

Who benefits most? A practical targeting framework

Symptom benefit is relevant • Adherence is important • Cardiovascular impact of untreated disease is likely • Cardiovascular benefit from treatment is plausible

Beyond Sleepiness: Cardiometabolic and Quality-of-Life Effects

Blood pressure

The ANDANTE individual-patient-data meta-analysis of 34 RCTs (n=7,456) found CPAP produced a modest overall reduction in office BP and a larger reduction in 24-hour ambulatory BP; the effect was driven mainly by patients with uncontrolled hypertension, and younger patients with uncontrolled BP benefited most

Symptom burden

Consistent, well-replicated improvements in Epworth Sleepiness Scale scores and disease-specific quality-of-life measures with effective, adherent use (see MERGE and Weaver dose-response data)

Hard cardiovascular events

Evidence for reduction in major adverse cardiovascular events with CPAP is less consistent across large randomised trials (SAVE, RICCADSA) than for symptomatic outcomes

CPAP vs oral appliance

Schwartz et al. (2018) meta-analysis: CPAP is significantly more effective than mandibular advancement devices at lowering AHI, but CPAP compliance was ~1.1 h/night lower — explaining why QoL/cognitive/functional outcomes showed no significant difference between the two despite CPAP's efficacy edge

Effective Pressure as a Phenotyping Marker

Landry et al. (2017, Sleep)

- The therapeutic CPAP pressure required to control a patient's OSA is a surrogate marker for their upper-airway collapsibility
- A patient's effective pressure therefore carries predictive information about how they might respond to alternative, non-CPAP treatments

Lee, Seay, Walters, Scalzitti & Dedhia (2019)

- If a patient's effective CPAP pressure exceeds certain thresholds, this predicts a lower chance of success with mandibular advancement devices or hypoglossal nerve stimulation
- In practice: a very high required pressure is itself a clinical signal, not just a titration outcome

Obesity Hypoventilation and COPD–OSA Overlap

NICE NG202 explicitly covers OSAHS, obesity hypoventilation syndrome (OHS) and COPD–OSAHS overlap syndrome together as related conditions along a spectrum of sleep-disordered breathing

Obesity Hypoventilation Syndrome (OHS)

- ATS's official Clinical Practice Guideline (2019) covers evaluation and management of OHS specifically
- Many OHS patients have coexisting severe OSA — CPAP can be effective first-line therapy in this subgroup, reserving NIV for those with persistent hypoventilation or CPAP failure
- Diagnosis requires confirming daytime hypercapnia in the context of obesity, not sleep study findings alone

COPD–OSAHS Overlap Syndrome

- Coexistence of COPD and OSAHS carries a disproportionately elevated risk of nocturnal hypoxaemia and cardiovascular complications compared with either disease alone
- NICE covers assessment and management of overlap syndrome within NG202's remit
- CPAP (or NIV where hypoventilation coexists) remains central to management alongside standard COPD therapy — NIV settings covered in the Part 2 workshop

ATS: Weight Management as an Adjunct to CPAP

The Role of Weight Management in the Treatment of Adult Obstructive Sleep Apnea: An Official ATS Clinical Practice Guideline (2018)

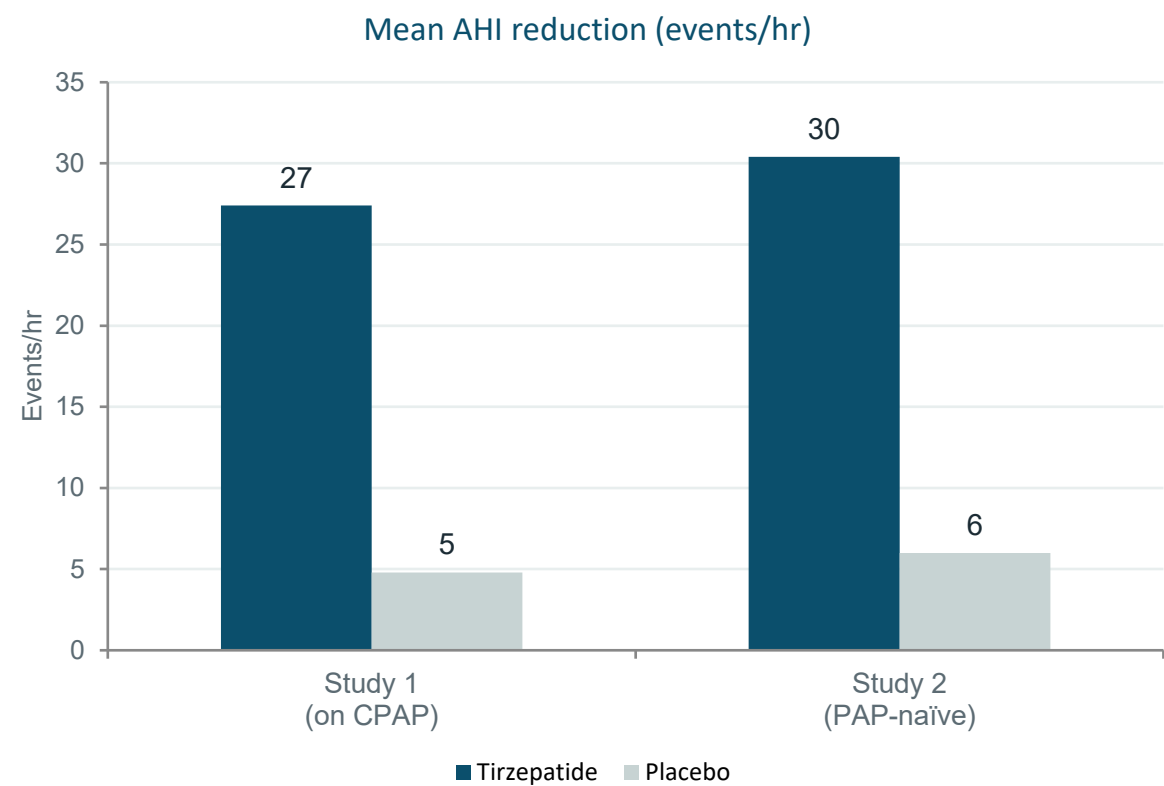
Positions structured weight-loss interventions (behavioural, dietary, and where appropriate pharmacological or surgical) as recommended adjuncts to primary OSA therapy in patients with overweight or obesity

Weight loss can reduce AHI and, in some patients, reduce the CPAP pressure required — but is not positioned as a routine substitute for CPAP in moderate-severe disease; only a minority (roughly 10%) see clinically meaningful change

Reinforces the mechanistic theme: CPAP treats the mechanical consequence of OSA nightly, while weight management addresses an underlying contributor

Weight-loss pharmacotherapy: SURMOUNT-OSA

Two-part, double-blind RCT of tirzepatide (GIP/GLP-1) up to 52 weeks — Malhotra A. et al., NEJM 2024



Also demonstrated:

- Mean body weight reduction of 18.1% (Study 1) and 20.1% (Study 2) vs 1.3% / 2.3% placebo
- Significant reductions in systolic blood pressure
- Improved patient-reported sleep impairment and disturbance
- Reduced hypoxic burden and hsCRP concentration

Cross-Guideline Comparison

Topic	NICE (NG202)	ATS	ERS / ERS-ESRS
Document type	Clinical guideline (graded recs)	Clinical practice guidelines & official statements (topic-specific)	Clinical guidelines & statements (mixed; check type per document)
CPAP indication basis	Symptom-gated in mild; offered in moderate/severe	AASM (Patil 2019): strong for sleepiness, conditional for QoL/HTN, insufficient evidence for CV prevention	Framed via comparative evidence vs alternative therapies
Adherence threshold	Referenced via telemonitoring pathway, not a single hour cut-off	Formal tracking via 2013 statement on adherence systems	>4 h/night cited for driving-licence adequacy
Telemonitoring	Recommended for 12 months, consider beyond	Not the primary focus of a single ATS document	Described as beneficial practice (e-Sleep), not mandated

Fourteen Facts Worth Memorising

1

NG202 amends TA139 for MILD OSAHS only — moderate/severe recommendations carry over from 2008

2

Sullivan et al. (1981): 4.5–10 cmH₂O abolished collapse in the first 5 CPAP patients

3

CPAP enlarges the pharynx more in the latero-lateral than antero-posterior dimension (Schwab 1996)

4

Nasal delivery stabilises the velopharynx directly; oral/oronasal delivery may not (Ebben 2012)

5

Marin et al. (2005): untreated severe OSA — OR 2.87 fatal, 3.17 non-fatal CV events vs controls over 10 years

6

AASM (Patil 2019): PAP is **STRONGLY** recommended for sleepiness only; **CONDITIONAL** for QoL/hypertension; insufficient evidence for CV-event prevention

7

4 h/night adherence threshold originated from US reimbursement policy, not a physiological ceiling

8

Benefit keeps rising well past 4 h/night (Weaver 2007) — 4 h is a floor, not a target

9

MERGE (Wimms 2019): significant QoL/ESS benefit in mild OSA despite ~4h median adherence

10

SAVE (McEvoy 2016) and RICCADSA (Peker 2016): both formally **NEGATIVE** for their primary cardiovascular endpoint

11

Xu et al. (2023): AHI does not predict MACE; TST90 and heart rate do

12

Landry (2017): effective CPAP pressure is a surrogate marker for upper-airway collapsibility

13

ATS OHS CPG (2019) requires daytime hypercapnia for diagnosis, not sleep study alone

14

ERS/ESRS driving statement: CPAP prevents most OSA-related collisions; EU directive dates to 2014

Summary

- CPAP works by pneumatically splinting the upper airway above its critical closing pressure — an effect first described by Sullivan et al. in 1981 and since imaged, quantified and extended to systemic mechanisms (lung volume, cardiac preload/afterload)
- NICE, ATS/AASM and ERS converge on CPAP as first-line therapy for symptomatic OSA, but differ in document structure and in exactly how mild disease, telemonitoring and cardiovascular prevention are framed
- The observational cohort literature (Marin 2005) suggested large cardiovascular benefit from treating severe OSA; modern RCTs (SAVE, RICCADSA) were formally negative on hard endpoints, and current guidance (AASM 2019) reflects that tension explicitly
- Phenotyping — by hypoxic burden, heart rate, or effective CPAP pressure — is displacing AHI-alone as the lens for predicting who benefits most from treatment
- Practical technique — titration method, adherence coaching, ASV and NIV — is covered in the companion Part 2 workshop

Key Source Documents

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Questions & Discussion

Good luck in the ESRS examination.

Continue with Part 2: CPAP, ASV and NIV — A Clinical Workshop