

OSA and Heart Health

Cardiovascular Risk, Evidence, and Care

Audience: APRN | **Contact hrs:** 1.5 | **Pharmacology hrs:** 0.0

Expected revision: October 4, 2028

Pharmacology hours are included in the total contact hours.

The cardiovascular question behind the snoring

A patient with recurrent atrial fibrillation reports feeling rested. Another has blood pressure that remains above goal despite several medications. A third uses continuous positive airway pressure for four hours, removes the mask, and sleeps another four. Each encounter raises a different question: who needs evaluation, what treatment can reasonably achieve, and whether prescribed treatment is actually covering the period of risk. Obstructive sleep apnea belongs in cardiovascular assessment, but it should neither explain every abnormal finding nor become a promise that one device will prevent every cardiac event (Yeghiazarians et al., 2021; Patil et al., 2019).

Obstructive sleep apnea (OSA) produces recurrent partial or complete upper-airway obstruction during sleep. The cardiovascular relevance extends beyond oxygen saturation: respiratory effort, arousal, autonomic changes, and the duration of nightly exposure also matter. Associations with hypertension, arrhythmia, coronary disease, and heart failure are well supported. The effect of treating OSA on those conditions depends on the outcome studied, patient selection, and treatment actually used. A lower apnea-hypopnea index is not automatically evidence of fewer myocardial infarctions (Yeghiazarians et al., 2021; Sánchez-de-la-Torre et al., 2023).

Purpose, audience, and learning outcomes

This activity develops advanced practice registered nurses' ability to integrate adult OSA assessment and treatment into cardiovascular care. Diagnostic, prescribing, and device-management decisions remain subject to professional authorization and local pathways. U.S. medication labeling is identified explicitly and is not a worldwide prescribing authorization. The five clinical cases are fictional teaching examples, not patient records.

After completing this activity, the learner should be able to: explain the principal mechanisms linking OSA to cardiovascular stress; select an appropriate

diagnostic pathway and interpret the limits of sleep-study metrics; differentiate established clinical benefits from uncertain cardiovascular outcome benefits; prioritize care when OSA coexists with hypertension, arrhythmia, or heart failure; compare PAP, oral-appliance, weight-management, and specialist procedural options; and formulate a follow-up plan that verifies treatment effectiveness, medication safety, and patient understanding.

The practical starting point is to separate three questions. Is OSA present? Does the patient have another cause of hypoxemia, sleepiness, or cardiovascular instability? Which treatment goals matter for this person? Keeping those questions distinct prevents a screening score from becoming a diagnosis, an association from becoming a causal conclusion, and an equipment prescription from being mistaken for effective treatment (Kapur et al., 2017; Caples et al., 2021).

From airway collapse to cardiovascular stress

Upper-airway patency reflects anatomy and neuromuscular control. Adipose tissue around the pharynx, a crowded oropharynx, mandibular position, nasal obstruction, and reduced dilator-muscle effectiveness can increase susceptibility to collapse. Obesity is important, but a person without obesity can have clinically important OSA. Aging, menopause, alcohol near bedtime, and selected sedating medications may influence risk or presentation. Avoid the stereotype that only an obviously sleepy man with a large neck warrants assessment (Yeghiazarians et al., 2021; Kapur et al., 2017).

During an obstructive event, inspiratory effort continues against a narrowed or closed airway. Oxygen may fall, carbon dioxide may rise, and increasingly negative pressure develops within the chest. An arousal helps restore airflow, but produces an abrupt autonomic transition rather than restorative sleep. Repetition can expose the circulation to intermittent hypoxia and reoxygenation, recurrent

An airway event becomes a cardiovascular exposure

Simplified pharyngeal cross-sections; not patient images or a scale drawing

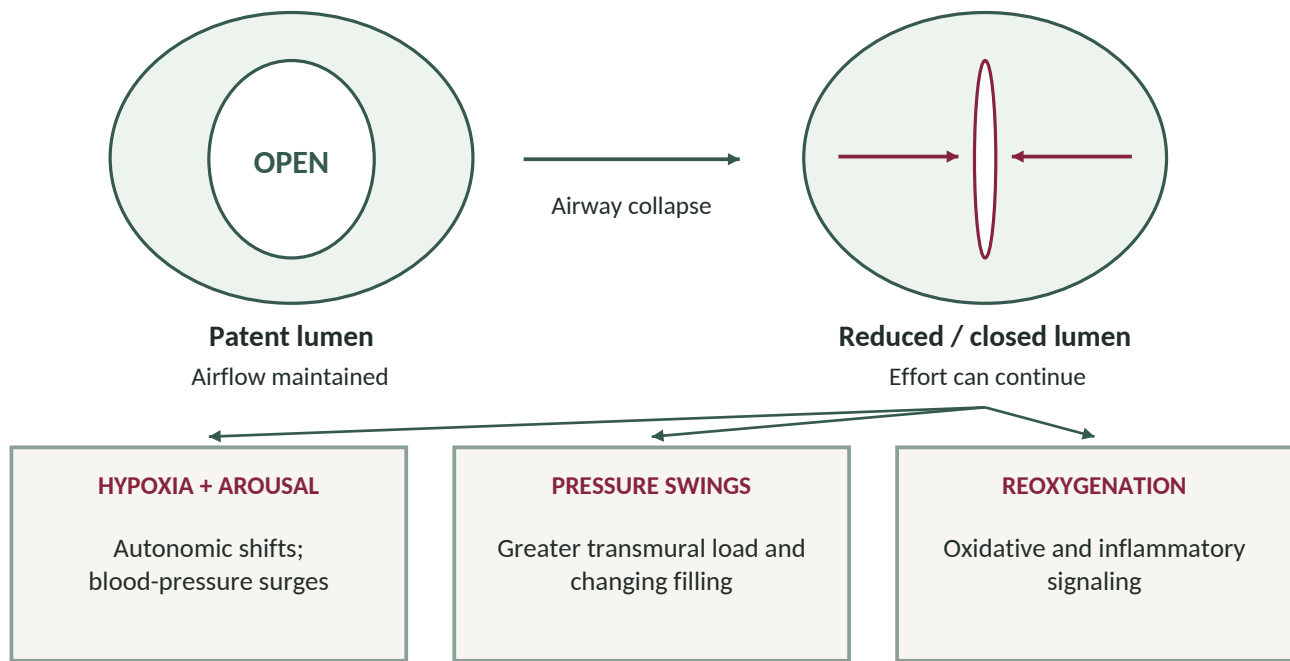


Figure 1. Recurrent upper-airway collapse creates interacting mechanical and physiologic exposures. The drawing simplifies the airway lumen and does not portray individual anatomy. Content: Yeghiazarians et al. (2021); Mehra et al. (2022).

pressure loading, and sleep fragmentation. Figure 1 separates the airway event from its downstream cardiovascular effects (Yeghiazarians et al., 2021; Mehra et al., 2022).

Intermittent hypoxia is not the whole mechanism

Hypoxia and arousal can activate sympathetic pathways, increasing vasoconstriction and altering heart rate and blood pressure. Reoxygenation can promote oxidative stress; inflammatory signaling and impaired endothelial function offer plausible pathways toward vascular injury. These mechanisms reinforce one another, but laboratory demonstrations do not quantify the cardiovascular risk attributable to OSA in an individual who also has hypertension, diabetes, smoking exposure, and obesity. Mechanistic plausibility strengthens a hypothesis; it does not replace a clinical outcome trial (Yeghiazarians et al., 2021; Mehra et al., 2022).

The pressure surrounding the heart is clinically relevant. Left ventricular transmural pressure is the difference between intracavitary pressure and the pressure outside the ventricle. More negative intrathoracic pressure can increase that gradient and therefore the load against which the left ventricle

ejects. Venous return and right ventricular filling can also change. Hypoxic pulmonary vasoconstriction adds a potential right-sided load, while ventricular interaction may impair left ventricular filling. These effects help explain why merely improving the oximeter value does not reproduce all effects of opening the airway (Yeghiazarians et al., 2021; Gottlieb et al., 2014).

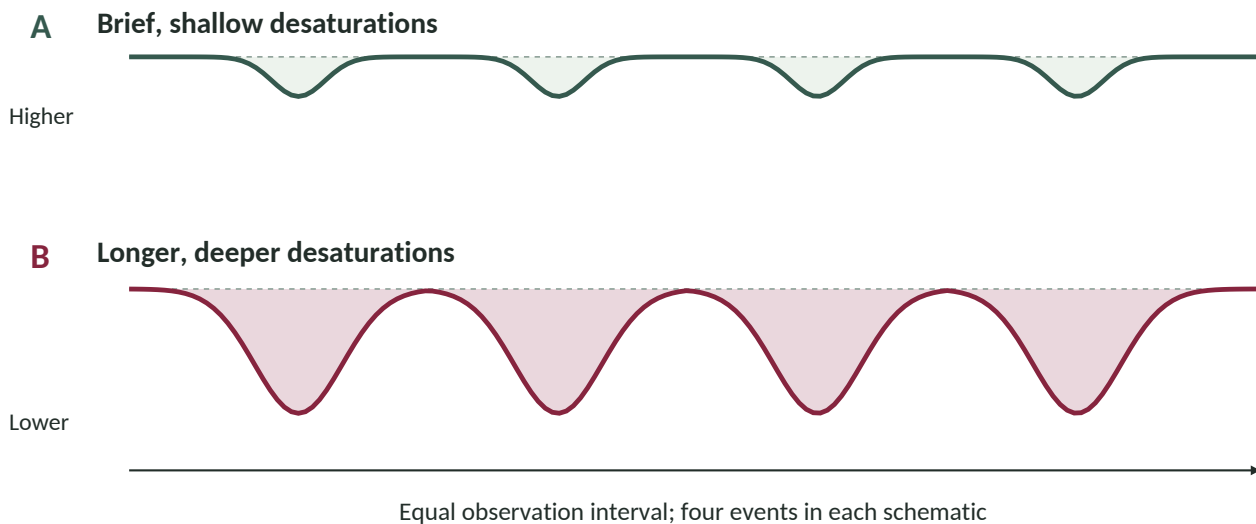
Autonomic behavior is not simply a sustained fast heart rate. Vagal influences during an apnea may produce bradycardia, followed by sympathetic acceleration at termination. In a susceptible myocardium, repeated autonomic shifts, atrial stretch, and altered repolarization can favor arrhythmogenesis. Clinical interpretation still requires the actual rhythm, symptoms, timing, medications, and structural heart disease; a nocturnal rhythm abnormality is not automatically caused by OSA (Mehra et al., 2022).

A bidirectional relationship

Heart failure may worsen sleep-disordered breathing rather than merely result from it. Fluid accumulated in the legs can redistribute toward the neck when the patient lies down, increasing upper-airway vulnerability. Pulmonary congestion and unstable

The same event count can conceal different hypoxic exposure

Schematic saturation patterns only; not patient recordings or numerical data



AHI counts events. Hypoxic burden reflects the depth and duration of event-related desaturation; neither replaces the full clinical assessment.

Figure 2. AHI alone does not capture event depth or duration. Hypoxic-burden methods quantify event-related desaturation, but are not a universal standalone treatment threshold. Original schematic based on Azarbarzin et al. (2019).

ventilatory control may also contribute to central events. Consequently, a change in the breathing pattern can be a reason to reassess congestion, cardiac status, and medication exposure—not just to increase airway pressure (Yeghiazarians et al., 2021; Badr et al., 2025).

Research measures such as muscle sympathetic nerve activity, spectral heart-rate variability, and post-exercise heart-rate recovery can illuminate autonomic dysfunction. They are not interchangeable, and they are affected by fitness, medications, rhythm, breathing, and measurement technique. They should not be ordered as a routine panel to diagnose OSA or certify cardiovascular protection from PAP. For routine care, the actionable evidence remains a suitable sleep study, cardiovascular assessment, symptom review, and verification of treatment response (Mehra et al., 2022; Kapur et al., 2017).

Recognize risk and choose the right test

Ask about witnessed pauses, choking or gasping, snoring, morning headaches, nocturia, fragmented sleep, and impaired daytime function. Distinguish sleepiness—the tendency to fall asleep—from fatigue or lack of energy. Ask specifically about unintended sleep while driving or operating equipment. A low

Epworth Sleepiness Scale score does not exclude OSA, especially in a patient referred through cardiovascular rather than sleep services. A bed partner's observations can be useful when the patient is unaware of the events (Yeghiazarians et al., 2021; Kapur et al., 2017).

The American Heart Association identifies resistant or poorly controlled hypertension, pulmonary hypertension, and recurrent atrial fibrillation after cardioversion or ablation as important settings for OSA screening. Evaluation is also relevant when heart failure and symptoms or suspicion of sleep-disordered breathing coexist. Screening here means identifying who needs further assessment; it does not mean that every person with cardiovascular disease should receive a home test without clinical triage (Yeghiazarians et al., 2021).

Questionnaires support referral, not diagnosis

A structured screening tool can standardize questions, but neither a questionnaire nor a consumer wearable establishes the diagnosis. Overnight oximetry can show concerning desaturation, yet cannot reliably identify whether the cause is obstruction, central apnea, hypoventilation, or cardiopulmonary disease. A

Table 1. Sleep-study language that changes interpretation

Measure	Interpretation
AHI severity	Mild: 5 to <15; moderate: 15 to <30; severe: ≥30 events/hour of sleep.
Hypopnea scoring	AASM-recommended scoring includes ≥30% airflow reduction for ≥10 seconds with ≥3% desaturation or arousal. Some reports use a ≥4% desaturation rule.
REI versus AHI	Monitoring time and sleep time are not equivalent denominators; document which was used.
Oxygen profile	Review baseline, nadir, duration of desaturation, and whether low saturation persists between events.

Sources: Kapur et al., 2017; Azarbarzin et al., 2019.

normal clinic saturation does not describe ventilation during sleep. Select testing after considering comorbid disease, medications, insomnia, and the question the test must answer (Kapur et al., 2017; Mokhlesi et al., 2019).

Polysomnography records sleep and physiologic signals and remains the standard diagnostic test. A technically adequate home sleep apnea test is appropriate for selected uncomplicated adults with a high clinical likelihood of moderate-to-severe OSA. Polysomnography is preferred for significant cardiorespiratory disease, suspected awake or sleep-related hypoventilation, potential respiratory muscle weakness, chronic opioid use, previous stroke, or severe insomnia. These are not trivial exclusions: a simplified study may miss the disorder that most needs treatment (Kapur et al., 2017).

A negative, inconclusive, or technically inadequate home study should lead to polysomnography when OSA remains a diagnostic concern. Do not repeatedly substitute home testing for a study that can resolve uncertainty. If an initial polysomnogram is negative but suspicion remains high, a repeat study may be appropriate. The diagnostic pathway should document both the result and whether it adequately addressed the clinical question (Kapur et al., 2017).

Interpret the denominator and the hypoxemia

The apnea–hypopnea index (AHI) counts apneas and hypopneas per hour of sleep. A respiratory event index (REI) from many home systems uses monitoring time rather than measured sleep time. Awake time in the denominator can dilute the apparent event frequency. Respiratory disturbance index (RDI) is not simply

another name for home-study REI; it may include respiratory effort–related arousals as well as apneas and hypopneas. Read the laboratory’s definitions rather than comparing labels alone; Table 1 summarizes key distinctions (Kapur et al., 2017).

The conventional severity bands are useful but do not capture all physiologic burden. Two people with the same AHI may have different event lengths, desaturation depths, arousal burdens, sleep-stage distributions, and cardiovascular vulnerability. Sleep apnea–specific hypoxic burden incorporates the depth and duration of event-related desaturations. Cohort studies link it to cardiovascular mortality, but it is not yet a universally standardized replacement for diagnostic criteria or a stand-alone treatment-selection rule. Figure 2 illustrates this distinction without presenting synthetic curves as patient recordings (Azarbarzin et al., 2019).

Persistent hypoxemia between events, daytime hypercapnia, marked obesity, or unexplained right-heart findings should broaden the differential. Obesity hypoventilation syndrome requires obesity and awake arterial carbon dioxide tension of at least 45 mmHg after excluding other causes. In a low-to-moderate suspicion setting, serum bicarbonate below 27 mmol/L can help exclude it; a value at or above 27 warrants assessment of arterial carbon dioxide rather than a diagnosis by bicarbonate alone. With high clinical suspicion, measure arterial blood gases directly. Diuretics and other causes of metabolic alkalosis can complicate interpretation (Mokhlesi et al., 2019).

Hypertension: a treatable overlap, not a single-cause diagnosis

OSA can contribute to nocturnal blood-pressure surges and loss of normal nighttime dipping. In apparent resistant hypertension, first verify measurement technique, appropriate cuff size, medication use, and out-of-office pressure. Assess volume status and other secondary causes. Labeling OSA as the explanation should not interrupt evaluation for primary aldosteronism, kidney disease, interfering drugs, or inadequate treatment. An OSA diagnosis and another cause of resistant hypertension can coexist (Carey et al., 2018).

CPAP lowers blood pressure modestly on average, with variation by baseline pressure, OSA phenotype, and actual use. The HIPARCO randomized trial in resistant hypertension demonstrated an approximately 3 mmHg between-group improvement in 24-hour mean pressure after 12 weeks, not normalization in every participant. The appropriate message is that effective OSA treatment may contribute to control alongside antihypertensive therapy, sodium and weight management, activity, and attention to adherence (Martínez-García et al., 2013).

Measure outcomes consistently. Compare home or ambulatory measurements obtained with a similar technique and medication regimen, and review symptoms of hypotension as treatment and weight change. A patient may feel better before pressure improves, or pressure may remain high despite excellent PAP use. Neither observation alone proves PAP failure. Conversely, a lower office reading does not establish adequate nighttime airway treatment (Carey et al., 2018; Patil et al., 2019).

CASE 1 • Resistant pressure without prominent sleepiness

Marcus, 58, has home readings averaging 154/92 mmHg despite three appropriately selected antihypertensive agents, including a diuretic. His spouse reports loud snoring and breathing pauses. He scores 6 on the Epworth scale and says a sleep study is unnecessary because he is not tired.

The low sleepiness score does not cancel the cardiovascular and witnessed-breathing clues. The APRN confirms cuff technique, medication access and actual use, and arranges an appropriate sleep evaluation while continuing the resistant-hypertension workup. Subsequent

polysomnography shows predominantly obstructive events with an AHI of 36/hour.

After treatment begins, the follow-up plan includes objective PAP use, residual events and leak, home blood pressure, and the patient's own treatment goals. The explanation is specific: opening the airway may help blood pressure, but it does not replace his medications or establish that OSA is the only cause of hypertension (Carey et al., 2018; Kapur et al., 2017; Martínez-García et al., 2013).

Vascular, metabolic, and myocardial signals

Coronary disease and vascular function

OSA frequently coexists with coronary artery disease. Intermittent hypoxia, sympathetic surges, and pressure loading provide biologically plausible contributors to myocardial oxygen supply–demand imbalance. Observational studies associate OSA with coronary events and adverse outcomes, but shared factors such as obesity, hypertension, diabetes, age, and smoking complicate causal attribution. A cardiovascular risk marker can be clinically useful even when the fraction of risk directly reversible by OSA treatment remains uncertain (Yeghiazarians et al., 2021; McEvoy et al., 2016).

Endothelial function, arterial stiffness, and carotid intima–media thickness have been used as surrogate measures of vascular injury. Some treatment studies show favorable changes, while findings differ with participant selection, treatment duration, and adherence. These measurements are not equivalent to coronary plaque regression or prevention of stroke. In routine practice, assess symptoms and established cardiovascular risk factors rather than ordering vascular surrogate tests simply to demonstrate that CPAP is working (Yeghiazarians et al., 2021).

Reports linking OSA with aortic dilation or aneurysm progression require similarly careful interpretation. A possible association does not justify universal aortic imaging or a new surveillance interval based solely on AHI. Established aortic disease, inherited aortopathy, family history, examination findings, and symptoms should determine the vascular pathway. Acute chest or back pain with concerning features requires urgent assessment; OSA should never provide a reassuring explanation for a possible acute aortic syndrome (Isselbacher et al., 2022).

Insulin resistance, lipids, and the liver

Intermittent hypoxia and sleep fragmentation can influence glucose regulation through sympathetic activation, altered insulin sensitivity, and inflammatory pathways. Obesity and visceral adiposity contribute to both OSA and metabolic disease, so associations do not neatly separate into a single causal direction. Treating the airway may improve selected metabolic measures, but it is not a dependable substitute for diabetes care or a reason to stop glucose monitoring (Chirinos et al., 2014; Yeghiazarians et al., 2021).

Weight treatment and PAP address overlapping but different mechanisms. In a randomized comparison of CPAP, weight loss, or both, weight loss contributed important metabolic benefits that CPAP alone did not reproduce. Thus, normalizing the device-reported AHI does not establish that dyslipidemia, inflammation, or adiposity has resolved. Continue appropriate lipid-lowering therapy, nutritional care, physical activity, and diabetes treatment, and assess their outcomes separately from PAP adherence (Chirinos et al., 2014).

The terminology metabolic dysfunction–associated steatotic liver disease (MASLD) replaces much of the older nonalcoholic fatty liver disease framework. OSA and nocturnal hypoxemia may coexist with hepatic steatosis and fibrosis, but a sleep diagnosis cannot stage liver disease, exclude alternative causes, or demonstrate that airway treatment reverses fibrosis. Patients with metabolic risk need the appropriate liver-risk assessment and follow-up rather than reassurance based on better sleep. Animal intermittent-hypoxia findings should not be presented as proof of a treatment effect in humans (Rinella et al., 2023; Kanwal et al., 2024).

Do not turn research biomarkers into routine OSA tests

BNP and NT-proBNP are markers used in the assessment of cardiac wall stress and heart failure, not diagnostic tests for OSA. Obesity can suppress concentrations, while renal dysfunction, age, atrial fibrillation, and other conditions alter interpretation. A low value in a patient with obesity and exertional dyspnea should not end a clinically warranted heart-failure evaluation. Likewise, a change after PAP initiation does not by itself establish a change in cardiovascular prognosis (Heidenreich et al., 2022).

High-sensitivity cardiac troponin identifies myocardial injury when elevated above the assay's reference limit, but the clinical diagnosis depends on the trajectory and surrounding evidence. Myocardial infarction requires evidence of ischemia in addition to acute injury. Do not attribute chest discomfort, new electrocardiographic changes, or a rising troponin to nocturnal hypoxemia without the appropriate acute evaluation. A patient can have OSA and an acute coronary syndrome at the same time (Nedungadi, 2026).

The same reasoning applies to an asymptomatic patient asking for a 'heart damage blood test' because the sleep study was abnormal. Explain what the proposed test can answer and whether the result would change management. Cardiovascular examination, blood pressure, risk assessment, and symptom-directed investigations are more useful than an unvalidated OSA biomarker panel. Avoid offering an apparently precise laboratory result as a certificate of either cardiovascular safety or irreversible damage (Heidenreich et al., 2022; Nedungadi, 2026).

Atrial fibrillation, other arrhythmias, and stroke

Atrial stretch, autonomic instability, hypoxia, and remodeling provide a substrate and potential triggers for atrial fibrillation (AF). OSA may be underrecognized in AF because many patients are not markedly sleepy. The AF guideline states that screening for OSA may be reasonable given its prevalence, while the role of treating sleep-disordered breathing to maintain sinus rhythm remains uncertain. This is a deliberate distinction between finding a treatable comorbidity and promising a particular rhythm outcome (Joglar et al., 2024; Mehra et al., 2022).

Observational studies often associate PAP use with less AF recurrence after cardioversion or ablation. Randomized evidence is less consistent and has important limitations, including sample size and exclusion of some symptomatic or higher-risk patients. Present OSA treatment as part of comprehensive risk-factor management, not as a guaranteed method of preventing recurrent AF. Weight, alcohol exposure, blood pressure, exercise, and the patient's rhythm-management plan remain relevant (Joglar et al., 2024).

A reduction in AF burden, a successful ablation, or improved PAP adherence does not independently

determine whether anticoagulation is indicated. Anticoagulation decisions follow thromboembolic risk and the applicable AF pathway, with bleeding risk and patient circumstances considered. The belief that correcting an overnight trigger removes daytime embolic risk is an unsafe shortcut. Document the rationale for any authorized antithrombotic decision separately from the sleep-treatment plan (Joglar et al., 2024).

CASE 2 • Better sleep does not settle anticoagulation

Elena, 69, has hypertension, prior AF ablation, and moderate OSA. Her PAP download shows consistent use and low residual event estimates. She has not noticed palpitations for several months and asks whether she can stop her prescribed anticoagulant.

The APRN acknowledges the improvement but separates symptom control from stroke prevention. Lack of perceived palpitations does not exclude AF, and successful sleep treatment does not erase her underlying thromboembolic risk. The current anticoagulation plan is maintained while the responsible prescriber reviews the full AF risk assessment, bleeding history, renal function, and treatment preferences.

The teaching message is not that PAP has failed. It is that different treatments serve different purposes: PAP treats upper-airway obstruction, the rhythm strategy addresses AF, and anticoagulation addresses an assessed embolic risk. Improvement in one domain does not automatically remove the need for another treatment (Joglar et al., 2024; Mehra et al., 2022).

Sleep-associated bradyarrhythmias should prompt evaluation for sleep apnea when clinically appropriate. Nocturnal sinus slowing alone is not an indication for permanent pacing. Treating a reversible sleep-related contributor may reduce some nocturnal rhythm disturbances, but syncope, symptomatic bradycardia, high-grade conduction disease, sustained ventricular arrhythmia, or hemodynamic instability requires its own urgent assessment. OSA is not a reason to defer electrophysiology evaluation or to attribute every overnight monitor alarm to a benign reversible cause (Mehra et al., 2022).

Stroke also creates diagnostic and care challenges. Weakness, communication difficulty, impaired cognition, and reduced dexterity may interfere with

mask use or symptom reporting. A history of stroke favors laboratory polysomnography rather than a simplified initial home pathway. Adapt education, caregiver involvement, and interface support to function. Suspected recurrent acute neurologic deficits require emergency stroke assessment, not a trial of nighttime respiratory treatment (Kapur et al., 2017; Mehra et al., 2022).

Heart failure, pulmonary pressure, and the apnea phenotype

OSA may coexist with left ventricular hypertrophy, impaired relaxation, left atrial enlargement, and abnormalities of myocardial deformation. Obesity and hypertension can produce similar findings. A preserved ejection fraction does not exclude clinically important heart failure, and a mildly abnormal strain measurement does not prove an OSA-induced cardiomyopathy. Echocardiography should answer the clinical question using the complete structural, functional, and hemodynamic context. Figure 3 illustrates the relationships without treating them as inevitable stages (Yeghiazarians et al., 2021; Heidenreich et al., 2022).

In heart failure with reduced ejection fraction (HFrEF), continue guideline-directed cardiac treatment and evaluate sleep-disordered breathing when indicated. CPAP may improve sleep quality and daytime sleepiness when OSA is present. Small studies showing improved ejection fraction or sympathetic measures do not establish a mortality benefit. An increase in ejection fraction after treatment can be encouraging without proving that PAP alone produced the change or that standard heart-failure therapy is no longer needed (Heidenreich et al., 2022).

Heart failure with preserved ejection fraction (HFpEF) is no longer accurately described as having no effective disease-directed therapy. Contemporary guidance recommends SGLT2 inhibitors for eligible symptomatic patients to reduce the composite risk of heart-failure hospitalization or cardiovascular death, with the trial benefit driven principally by fewer heart-failure hospitalizations. Address congestion and relevant comorbidities. OSA treatment complements this plan; it is not a replacement for a contemporary HFpEF pathway (McDonagh et al., 2023).

Repeated loading affects both sides of the circulation

Conceptual heart-lung schematic; association does not establish an individual cause

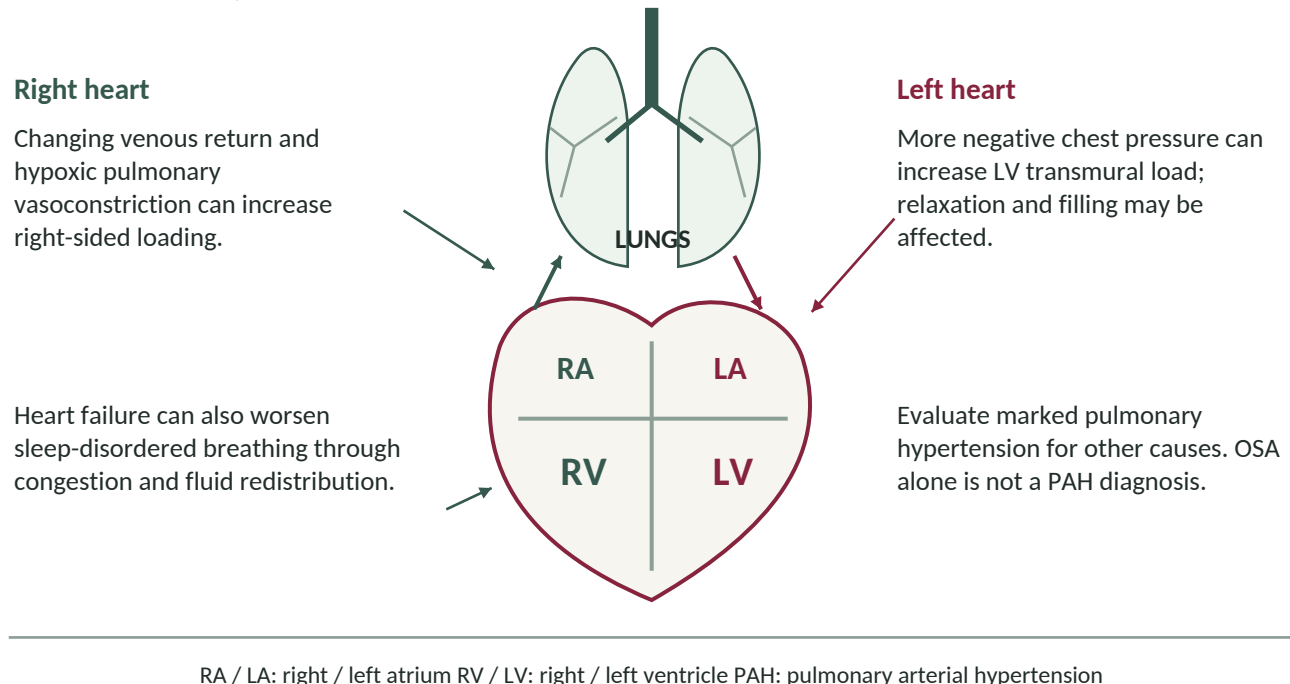


Figure 3. OSA and heart failure have bidirectional relationships. Marked pulmonary hypertension requires etiologic evaluation rather than automatic attribution to OSA. Content: Yeghiazarians et al. (2021); Humbert et al. (2022); Heidenreich et al. (2022).

OSA and central sleep apnea are different problems

In OSA, airflow obstruction occurs despite respiratory effort. Central sleep apnea (CSA) involves reduced or absent respiratory effort during events and may reflect unstable ventilatory control, medication effects, or another condition. Both patterns may coexist, and central events can become apparent during PAP treatment. A device label or a report of ‘apnea’ is insufficient to select a ventilatory mode. Review the actual phenotype, cardiac function, symptoms, and the quality of the available data (Badr et al., 2025).

The distinction is especially important for adaptive servo-ventilation (ASV). SERVE-HF identified increased mortality with the studied ASV approach in symptomatic systolic heart failure with predominantly central apnea. ADVENT-HF, using a different ASV approach, did not show improved cardiovascular outcomes or the same safety signal. These results should not be generalized to ordinary CPAP treatment of obstructive apnea, nor should different devices be assumed interchangeable (Cowie et al., 2015; Badr et al., 2025).

The 2025 AASM CSA guideline conditionally supports several treatments according to etiology and

emphasizes reassessment of underlying disease. ASV consideration in HFrEF is restricted to experienced centers with close monitoring and shared decision-making. Device-specific contraindications still matter: some ASV labeling contraindicates use in symptomatic chronic heart failure with LVEF at or below 45% and moderate-to-severe predominant CSA. An APRN should not resolve that tension by initiating ASV empirically; specialist review must reconcile the current guideline, the patient’s phenotype, and the exact device labeling (Badr et al., 2025; ResMed, n.d.).

Pulmonary hypertension is not automatically pulmonary arterial hypertension

Pulmonary hypertension is an umbrella hemodynamic condition; pulmonary arterial hypertension is a specific subgroup, not another name for every elevated pulmonary pressure. Current ESC/ERS guidance does not regard isolated nocturnal OSA as a usual explanation for substantial pulmonary hypertension. Look for left-heart disease, chronic lung disease, hypoventilation with daytime hypercapnia, chronic thromboembolic disease, or another cause. An echocardiographic pressure estimate suggests probability but does not itself establish the full hemodynamic diagnosis (Humbert et al., 2022).

CASE 3 • The pressure estimate needs a broader explanation

Thomas, 66, has severe OSA, obesity, increasing edema, and exertional dyspnea. Echocardiography reports right ventricular enlargement and an elevated estimated pulmonary artery systolic pressure. A note attributes all findings to sleep apnea; his awake bicarbonate is 31 mmol/L.

The APRN does not accept OSA as a complete explanation. Assessment addresses volume status, left ventricular and valvular disease, pulmonary disease, thromboembolic risk, and possible hypoventilation. The bicarbonate result raises the question of chronic hypercapnia but is not diagnostic, particularly in a patient using diuretics. Arterial carbon dioxide measurement and appropriate specialist-directed testing clarify the phenotype.

Treatment follows the confirmed contributors. Adding pulmonary arterial hypertension medication solely because an echocardiogram estimates a high pressure would be inappropriate. Worsening oxygenation, respiratory distress, syncope, or hemodynamic instability would require urgent care rather than routine sleep-clinic follow-up (Humbert et al., 2022; Mokhlesi et al., 2019).

What cardiovascular outcome trials actually show

The central evidence question is no longer whether any randomized cardiovascular trial has been performed. Large trials have tested CPAP added to usual care in patients with established cardiovascular disease and OSA, often without severe sleepiness. SAVE did not demonstrate a reduction in its composite cardiovascular endpoint in the intention-to-treat comparison, despite improvement in sleepiness and quality-of-life measures. Average nightly use was limited. The result informs secondary prevention in a defined trial population; it does not show that symptomatic OSA should be left untreated (McEvoy et al., 2016).

An individual participant data meta-analysis combined SAVE, ISAACC, and RICCADSA, totaling 4,186 participants. The intention-to-treat hazard ratio for recurrent major cardiac or cerebrovascular events was 1.01, with a 95% confidence interval of 0.87–1.17. A modeled on-treatment analysis associated use of at least four hours daily with lower risk: hazard ratio 0.69, 95% confidence interval 0.52–0.92. Figure 4

displays these results as different questions, not as competing randomized answers (Sánchez-de-la-Torre et al., 2023).

Intention-to-treat preserves the comparison created by randomization: what happened when patients were assigned to the treatment strategy? An adherence analysis asks what happened among people with specified treatment exposure and therefore needs additional adjustment. Better adherence may travel with health behaviors, access, disease characteristics, and other unmeasured influences. Sophisticated adjustment reduces some bias but does not recreate a trial that randomized patients to sustained all-night use. The adherence finding supports efforts to improve effective treatment without justifying a guarantee of fewer events (Sánchez-de-la-Torre et al., 2023).

The four-hour threshold is an analytic convention, not a biological finish line or a universal definition of adequate treatment. A patient who sleeps eight hours but uses PAP for four remains untreated for a substantial part of the night. Conversely, a device may run while the patient is awake. The relevant clinical questions are how much of actual sleep is covered and whether obstruction, hypoxemia, symptoms, and tolerability have improved (Patil et al., 2019; Caples et al., 2021).

Research is moving toward physiologic and clinical phenotypes that might identify greater benefit. A 2026 machine-learning analysis of SAVE explored heterogeneous treatment effects, but it was a secondary analysis requiring independent validation before clinical use. Its predictions are not an established rule for withholding or selecting CPAP. Similarly, hypoxic burden is promising for risk characterization but should not be promoted as a fully validated prescription algorithm (Cohen et al., 2026; Azarbarzin et al., 2019).

A balanced explanation is therefore possible: PAP reliably treats obstruction when used effectively and can improve important patient-centered outcomes; it may help blood pressure; protection against recurrent major cardiovascular events has not been established for every OSA population. Continue proven cardiovascular prevention while choosing OSA treatment with the patient. Uncertainty about one endpoint is not the same as absence of any worthwhile treatment benefit (Patil et al., 2019; McEvoy et al., 2016).

Cardiovascular outcomes: keep the analyses separate

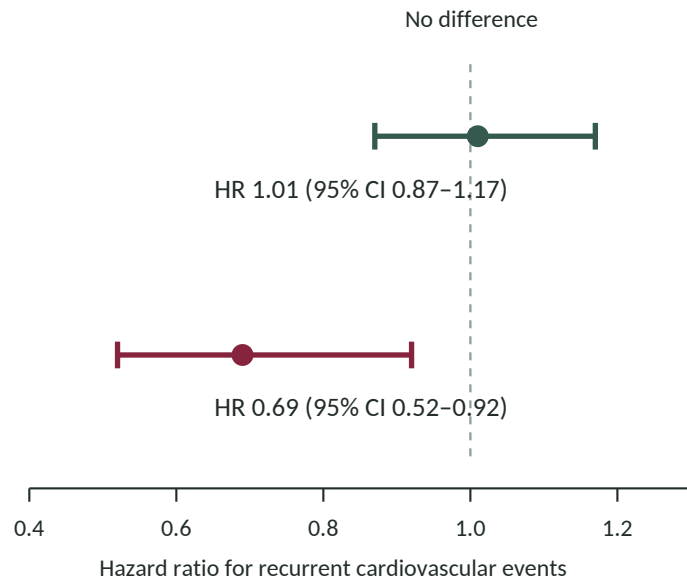
Individual-participant meta-analysis of three trials; 4,186 participants

Randomized assignment

Intention-to-treat

Use ≥ 4 hours/night

Modeled adherence analysis



Neutral randomized result; favorable adherence association. The second analysis is not a randomized comparison of adherent versus nonadherent patients.

Figure 4. Points and horizontal lines show hazard ratios and 95% confidence intervals from Sánchez-de-la-Torre et al. (2023). The adherence estimate used a marginal structural model; residual confounding remains possible. This is an original rendering of reported numerical results.

Choose a treatment the patient can use effectively

PAP is a treatment process, not an equipment order

Positive airway pressure pneumatically splints the upper airway. AASM recommendations strongly support PAP for excessive sleepiness and conditionally support it for impaired sleep-related quality of life or comorbid hypertension. CPAP or auto-adjusting PAP is generally used for routine adult OSA; selection and initiation depend on comorbidity and the clinical setting. Home APAP initiation pathways studied in uncomplicated OSA should not automatically be extended to patients with major cardiopulmonary disease or suspected hypoventilation (Patil et al., 2019).

Early education and troubleshooting make the prescribed treatment more usable. Ask the patient to demonstrate mask application and device operation. Review leak, nasal symptoms, dryness, pressure discomfort, aerophagia, claustrophobia, and skin injury. Address the actual barrier: an unsuitable interface calls for refitting, not a lecture about motivation. A patient with limited hand function may

need a simpler fastening system or caregiver assistance. Check equipment availability, replacement supplies, electricity, travel arrangements, and the cost of follow-up (Patil et al., 2019).

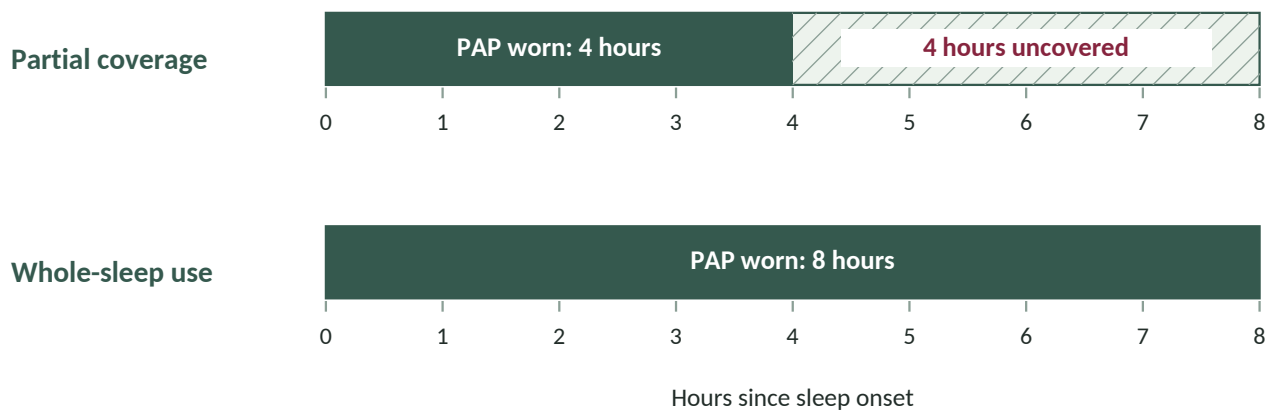
Review downloads in context. Usage duration, residual event estimates, pressure behavior, and leak can help identify a problem, but device-generated event labels are not equivalent to a complete sleep study. Large leaks may impair treatment and event estimation. Persistent symptoms with apparently reassuring data require review of actual sleep time, medication effects, other sleep disorders, and cardiovascular or respiratory disease. Do not escalate pressure reflexively in response to a number without considering the event type and the patient (Caples et al., 2021).

CASE 4 • Four hours on the device, eight hours in bed

Priya, 55, has hypertension and severe OSA. Her download shows approximately four hours of use nightly. She explains that nasal dryness wakes her around 02:00, after which she removes the mask and sleeps until 06:00. The device-reported residual AHI is low during recorded use.

Treatment needs to cover the sleep period

Illustrative eight-hour sleep window; the goal is use during all sleep, including naps



A low residual device AHI describes the period the device is used—not the untreated remainder of the night. Review total sleep time, use, leak, residual events, and symptoms together.

Figure 5. Four hours is a common research adherence threshold, not a physiologic guarantee of protection for a longer sleep period. Effective treatment requires usable equipment and follow-up. Content: Patil et al. (2019); Caples et al. (2021); Sánchez-de-la-Torre et al. (2023).

The first response is to address dryness, interface fit, and comfort with the sleep-treatment team, then reassess whether therapy covers the entire sleep period. The low residual AHI describes treated time; it does not establish that the untreated second half of the night is safe. The patient is encouraged to use the prescribed therapy during naps as well. The agreed goal is practical: restore tolerable whole-night use and review symptoms, leak, residual events, and home blood pressure. Calling her ‘compliant’ based on one threshold would conceal a remediable gap in treatment exposure. Figure 5 makes this coverage problem visible (Patil et al., 2019; Caples et al., 2021).

Alternatives should be verified, not assumed effective

A custom, titratable mandibular advancement device is an option for selected adults who prefer an alternative or cannot tolerate CPAP. A qualified dental professional should assess suitability, titrate the device, and monitor dental or jaw effects, working with the sleep clinician. A store-bought snoring product is not an equivalent treatment. Follow-up sleep testing is important because reduced snoring or a better subjective night does not necessarily establish

adequate control of obstructive events (Ramar et al., 2015; Caples et al., 2021).

In the CRESCENT randomized trial, a mandibular advancement device was noninferior to CPAP for reduction of 24-hour mean arterial pressure over six months in selected patients with hypertension, increased cardiovascular risk, and moderate-to-severe OSA. This supports a meaningful alternative when selected and followed appropriately. It does not establish equivalent elimination of apnea in all patients, nor prove that the two treatments prevent myocardial infarction or stroke equally well (Ou et al., 2024).

Positional treatment may help a documented position-dependent phenotype, but should not be selected merely because the patient reports less snoring on the side. Surgical evaluation, including discussion of upper-airway procedures or hypoglossal nerve stimulation, may be appropriate after individualized assessment. Eligibility depends on anatomy, disease pattern, body habitus, prior treatment, and the specific procedure or device. Bariatric evaluation may also be relevant. None of these pathways should omit

objective reassessment of residual OSA (Kent et al., 2021; Caples et al., 2021).

Supplemental oxygen is not an interchangeable replacement for PAP in ordinary obstructive apnea. It may improve measured saturation while obstruction, respiratory effort, and arousals continue. In HeartBEAT, CPAP reduced blood pressure whereas nocturnal oxygen did not produce the same benefit. Oxygen can have a role for another established indication, but persistent desaturation on apparently effective PAP requires evaluation for residual obstruction, hypoventilation, lung disease, or heart failure rather than unsupervised oxygen addition (Gottlieb et al., 2014; Mokhlesi et al., 2019).

Weight treatment and the new medication pathway

Weight management should be offered without delaying effective airway treatment. Sustainable nutrition, activity suited to cardiovascular status, and behavioral support address adiposity and metabolic risk. Some patients need obesity pharmacotherapy or metabolic surgery. Discuss these options without presenting OSA as a failure of willpower. Weight loss can reduce disease severity, but improvement varies and recurrence is possible; do not discontinue PAP solely because a weight target or a quieter night has been achieved (Chirinos et al., 2014; Caples et al., 2021).

In the United States, tirzepatide under the Zepbound labeling is approved for moderate-to-severe OSA in adults with obesity, together with a reduced-calorie diet and increased physical activity. This is not a general indication for every patient with OSA. Table 2 summarizes selected prescribing checkpoints. SURMOUNT-OSA included separate trials in participants not using PAP and those using PAP at baseline. Tirzepatide improved AHI and several related measures over 52 weeks; the trials did not establish an OSA-specific reduction in myocardial infarction or stroke (U.S. Food and Drug Administration, 2024; Malhotra et al., 2024).

The placebo-adjusted AHI reductions were approximately 20.0 and 23.8 events/hour in the two trials. These are mean treatment effects, not predicted individual outcomes and not guaranteed remission. A medication that reduces weight and airway-event burden can still leave clinically important residual OSA. Explain that prescribed PAP should continue

until reassessment supports a different plan, rather than asking the patient to infer treatment success from appetite suppression or weight loss alone (Malhotra et al., 2024; Caples et al., 2021).

Prescribing safety includes gastrointestinal intolerance, dehydration-related kidney injury, gallbladder disease, and pancreatitis concerns. The label does not recommend use with severe gastroparesis. Review concurrent insulin or sulfonylurea therapy because hypoglycemia risk can increase, and do not combine with another tirzepatide product or GLP-1 receptor agonist. Pregnancy requires discontinuation; oral hormonal contraception needs a nonoral method or additional barrier protection for four weeks after initiation and after each dose increase. Patients should inform procedural teams because delayed gastric emptying is relevant to aspiration risk. Use the actual product instructions rather than assuming all device volumes are identical (Eli Lilly and Company, 2026).

CASE 5 • The weight is falling, but the treatment plan still matters

Rosa, 47, has obesity, hypertension, and an AHI of 42/hour. She uses PAP and begins authorized tirzepatide treatment. Several months later, she has lost weight and asks whether she can stop PAP immediately. She also reports persistent vomiting after a recent dose increase and dizziness on standing.

The immediate issue is possible volume depletion and medication intolerance, not whether she has reached an attractive weight-loss milestone. The APRN assesses hydration, blood pressure, renal risk, and the need for prompt clinical review and an authorized medication adjustment. Severe abdominal pain, inability to maintain intake, syncope, or other concerning findings require escalation.

Once the acute problem is addressed, the sleep plan is reassessed objectively. Weight loss alone does not show that obstruction or hypoxemia has resolved. Rosa leaves with separate instructions for medication safety, cardiovascular monitoring, and continued airway treatment pending sleep-clinician reassessment (Eli Lilly and Company, 2026; Caples et al., 2021).

Table 2. Tirzepatide: selected U.S. prescribing checkpoints

Checkpoint	Clinical detail
Initiation and titration	2.5 mg subcutaneously weekly for 4 weeks, then 5 mg weekly. Further increases are 2.5 mg after at least 4 weeks on the current dose.
OSA maintenance	10 mg or 15 mg weekly; maximum 15 mg weekly. The 2.5 mg dose is for initiation, not maintenance.
Do not use	Personal/family history of medullary thyroid carcinoma, MEN2, or known serious hypersensitivity to the product.
Before and during treatment	Review GI tolerance, hydration, glucose-lowering therapy, pregnancy potential, relevant oral drugs, and the exact delivery device.

Sources: Eli Lilly and Company, 2026, sections 1–5 and 8.

Close the loop: function, physiology, and cardiovascular care

Follow-up should ask whether the treatment is available, usable, effective during actual sleep, and worthwhile to the patient. Review sleepiness and driving safety, sleep-related quality of life, treatment use, residual events, leak, blood pressure, weight, and relevant cardiac symptoms. A normal residual device AHI does not explain persistent dyspnea, edema, chest discomfort, or reduced exercise tolerance. Separate the domains so that improvement in one does not hide deterioration in another (Caples et al., 2021; Patil et al., 2019).

Routine repeat sleep testing is not generally needed for an asymptomatic patient doing well on PAP. Testing becomes useful with recurrent or persistent symptoms, clinically significant weight change, concern about hypoxemia or hypoventilation, unexplained device data, or relevant cardiovascular changes. Non-PAP interventions also require confirmation of effectiveness. The question is not simply whether another test can be ordered, but whether it can resolve a treatment decision that the available data cannot answer (Caples et al., 2021).

Use teach-back for the decisions most likely to be misunderstood: when the device should be worn, whom to call for intolerance, why cardiovascular medications remain necessary, and what findings need urgent evaluation. Ask the patient to describe the plan in their own words. Someone who says ‘four hours protects me for the whole night’ or ‘my AF is cured because I sleep better’ has revealed a specific

educational need that a generic handout may not address (Patil et al., 2019; Joglar et al., 2024).

Document a closed-loop handoff among sleep medicine, cardiology, primary care, dental sleep care, and the patient when relevant. Include the diagnostic method and phenotype, important hypoxemia or hypercapnia, cardiac function when it affects device selection, treatment and actual use, unresolved barriers, and responsibility for the next review. A referral is not complete until the result is reviewed and the plan is communicated. An adherence download is not the same as a comprehensive cardiovascular assessment (Caples et al., 2021; Badr et al., 2025).

Clinical synthesis

OSA is a clinically important cardiovascular comorbidity with plausible injurious mechanisms, heterogeneous expression, and effective treatments for airway obstruction and patient-centered outcomes. The safest approach avoids two extremes: dismissing OSA because a patient is not sleepy, and promising that correcting AHI will remove cardiovascular risk. Choose the right diagnostic test, identify central apnea or hypoventilation when present, support treatment throughout sleep, and continue evidence-based cardiovascular prevention. Measure what each intervention is intended to change, and revise the plan when symptoms, physiology, or the patient’s circumstances change (Yeghiazarians et al., 2021; Patil et al., 2019; Sánchez-de-la-Torre et al., 2023).

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