CO-MORBIDITIES IN HEART FAILURE

SLEEP APNEA

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HOW TO DIAGNOSE SLEEP APNEA
• **SLEEP-DISORDERED BREATHING**

Umbrella term for different pathological entities that are associated with restless sleep

• **APNEA**

Cessation of oral or nasal air flow for >10sec associated with a drop in O2 saturation by more than 4 points

• **HYPOPNEA**

Reduction of oral or nasal air flow by <50% for >10sec associated with a drop in O2 saturation by more than 4 points

• **AHI**

Number of apneas or hypopneas per hour
CLASSIFICATION OF SLEEP APNEA

• Up to 5 events/h is usually defined as normal,

• 5–15/h as **mild SDB**

• 15–30/h as **moderate SDB**,

• and >30/h as **severe SDB**.

• The number and severity of oxygen desaturations may also be used as a metric of the severity of SDB.

• >50% of events are obstructive are labelled as predominantly **OSA**,

• >50 % of events are central, such a patient is labelled as predominantly **CSA**.
SYMPTOMS

• Witnessed phases of apnea of at least 10 sec duration
• Restless sleep
• Excessive daytime sleepiness
• Morning headache
• Vertigo, particularly after getting up
• Waking up with a dry mouth/ sore throat
• Nocturnal sweating
• Nocturia
• Sleepiness while driving
• Depression, forgetfulness, mood changes
• Decreased interest in sex, impotence, erectile dysfunction
A *polysomnography* is a multi-parametric sleep study that monitors
- respiratory airflow
- oxygen saturation (via pulse oximetry)
- thoracic and abdominal respiratory effort
- rhonchopathy
- heart activity (via ECG)
- skeletal muscle behaviour (via electromyography)
- electrical brain activity (via electroencephalography)
- eye movement (via electro-oculography) during sleep.

A *polygraphy* only includes the recordings of
- respiratory airflow
- oxygen saturation
- thoracic and abdominal movement.

*Sensitivity and specificity of 90–100 %*
Sample PSG Report

- Events by sleep stage & position

**Respiratory Summary – Pre-Treatment:**

<table>
<thead>
<tr>
<th>Types of Respiratory Events</th>
<th>Number</th>
<th>Index</th>
</tr>
</thead>
<tbody>
<tr>
<td>Obstructive Apneas</td>
<td>65</td>
<td>22.3/hr</td>
</tr>
<tr>
<td>Mixed Apneas</td>
<td>0</td>
<td>0.0/hr</td>
</tr>
<tr>
<td>Central Apneas</td>
<td>0</td>
<td>0.0/hr</td>
</tr>
<tr>
<td>Total Apneas</td>
<td>65</td>
<td>22.3/hr</td>
</tr>
<tr>
<td>Total Hypopneas*</td>
<td>48</td>
<td>16.5/hr</td>
</tr>
<tr>
<td>Apneas + Hypopnea*</td>
<td>113</td>
<td>38.9/hr</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Respiratory Effort Related Arousal (RERA) Events</th>
<th>Total</th>
<th>Index</th>
</tr>
</thead>
<tbody>
<tr>
<td>Total:</td>
<td>24</td>
<td>8.3</td>
</tr>
<tr>
<td>Non-REM:</td>
<td>23</td>
<td>8.3</td>
</tr>
<tr>
<td>REM:</td>
<td>1</td>
<td>6.7</td>
</tr>
<tr>
<td>Supine:</td>
<td>24</td>
<td>8.3</td>
</tr>
<tr>
<td>Lateral:</td>
<td>N/A</td>
<td>N/A</td>
</tr>
<tr>
<td>Prone:</td>
<td>N/A</td>
<td>N/A</td>
</tr>
</tbody>
</table>

**Oxygen Saturation Summary – Pre-Treatment:**

- Mean SaO2: 95.2%
- % TST SaO2 < 90%: 2.3%
- % TST SaO2 < 99%: 1.7%
- Minutes SaO2 < 90%: 4.0
- Minutes SaO2 <= 88%: 5.5

- Lowest SaO2: 79.0%
- # Desaturation 4% or >: 91
- Desaturation Index: 31.3
- NREM Desaturations Index: 28.6
- REM Desaturations Index: 80.0
CENTRAL VS. OBSTRUCTIVE SLEEP APNEA
OSA is primarily caused by an obstruction of the upper respiratory tract that results in repeated interruptions of the normal breathing process during sleep. Patients with OSA are often anatomically predisposed to smaller pharyngeal breathing tracts as a result of obesity, enlarged tonsils, adenoids, or tissue irregularities. Rostral fluid shift during sleep in HF also causes OSA.
The central form of apnoea is caused by a dysfunction of the ventilatory control system in HF, neurological diseases, renal failure or opioids use.

*J Am Coll Cardiol. 2017 February 21; 69(7): 841–858*
RELATIONSHIP BETWEEN OBSTRUCTIVE SLEEP APNOEA AND HEART FAILURE

Nat Rev Cardiol. 2016 Jul;13(7):389-403
RELATIONSHIP BETWEEN CENTRAL SLEEP APNOEA AND HEART FAILURE

Arousal → CSA

$P_aO_2$ and $P_aCO_2$ oscillation → Cardiovacular sympathetic neuron cyclic activation

$\uparrow$ Sympathetic activation $\downarrow$ Vagal tone

$\uparrow$ RAA system

$\uparrow$ Blood pressure, heart rate, and $VO_2$ oscillation

$\uparrow$ Left ventricular remodelling

Fluid retention → Heart failure

Apnoea threshold

Respiratory centre alterations

$\downarrow$ $P_aCO_2$

$\uparrow$ $P_aCO_2$ sensitivity

$\uparrow$ Ventilatory rate

$\uparrow$ Stretch receptor stimulation

$\uparrow$ Pulmonary venous pressure

Pulmonary congestion

Nat Rev Cardiol. 2016 Jul;13(7):389-403
OBSTRUCTIVE SLEEP APNEA AND THE RISK OF SUDDEN CARDIAC DEATH
A LONGITUDINAL STUDY OF 10,701 ADULTS

![Graph showing the risk of sudden cardiac death (SCD) over years for different AHI levels.](image)

<table>
<thead>
<tr>
<th>Sleep parameters</th>
<th>HR</th>
<th>95% CI</th>
<th>P value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Apnea-hypopnea index (per 10)</td>
<td>1.03</td>
<td>(0.98, 1.08)</td>
<td>0.281</td>
</tr>
<tr>
<td>Mean nocturnal O₂ saturation (per 10%)</td>
<td>1.49</td>
<td>(0.96, 2.28)</td>
<td>0.073</td>
</tr>
<tr>
<td>Lowest nocturnal O₂ saturation (per 10%)</td>
<td>1.14</td>
<td>(1.01, 1.27)</td>
<td>0.029</td>
</tr>
</tbody>
</table>

J Am Coll Cardiol. 2013 August 13; 62(7):
In men, severe obstructive sleep apnoea-hypopnoea significantly increases the risk of fatal and non-fatal cardiovascular events.

Lancet 2005; 365: 1046–53
N=2717 pts with moderate-to-severe OSA and coronary or cerebrovascular disease
The primary composite end point was death from cardiovascular causes, myocardial infarction, stroke, or hospitalization for unstable angina, heart failure, or transient ischemic attack

CPAP significantly reduced snoring and daytime sleepiness and improved health-related quality of life and mood

SLEEP APNEA DIAGNOSIS IN HF
PREVALENCE OF MODERATE TO SEVERE SLEEP APNEA IN LV DYSFUNCTION

<table>
<thead>
<tr>
<th></th>
<th>LVSD Asymptomatic</th>
<th>LVDD Asymptomatic</th>
<th>HFrEF</th>
<th>HFpEF</th>
<th>ADHF</th>
</tr>
</thead>
<tbody>
<tr>
<td>AHI &gt; 15/h</td>
<td>66%</td>
<td>25%</td>
<td>53%</td>
<td>47%</td>
<td>78%</td>
</tr>
<tr>
<td>OSA</td>
<td>55%</td>
<td>21%</td>
<td>20%</td>
<td>23%</td>
<td>47%</td>
</tr>
<tr>
<td>CSA</td>
<td>11%</td>
<td>4%</td>
<td>33%</td>
<td>24%</td>
<td>31%</td>
</tr>
</tbody>
</table>

J Am Coll Cardiol. 2017 February 21; 69(7): 841–858
RELATIVE PREVALENCE AND IMPORTANCE OF OSA AND CSA ACCORDING TO HEART FAILURE SEVERITY.

Circ J 2012; 76: 2305 – 2317
PREVALENCE AND PHYSIOLOGICAL PREDICTORS
OF SLEEP APNEA IN PATIENTS WITH HEART
FAILURE AND SYSTOLIC DYSFUNCTION

Table 3. Multivariable Odds Ratios for OSA and CSA

<table>
<thead>
<tr>
<th>Variables</th>
<th>OSA</th>
<th>CSA</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Adjusted Odds Ratio (95%CI)</td>
<td>P Value</td>
</tr>
<tr>
<td>Age (per 10-y increase)</td>
<td>1.52 (1.08 – 2.14)</td>
<td>.017</td>
</tr>
<tr>
<td>Male sex</td>
<td>4.95 (1.74 – 14.07)</td>
<td>.003</td>
</tr>
<tr>
<td>Body mass index (per 5-kg/m² increase)</td>
<td>1.58 (1.08 – 2.33)</td>
<td>.019</td>
</tr>
<tr>
<td>Atrial fibrillation</td>
<td>2.44 (0.56 – 10.55)</td>
<td>.231</td>
</tr>
<tr>
<td>PCO₂, wake (per 1-mmHg decrease)</td>
<td>1.02 (0.94 – 1.11)</td>
<td>.569</td>
</tr>
<tr>
<td>Thiazide and loop diuretic use</td>
<td>2.25 (0.87 – 5.82)</td>
<td>.094</td>
</tr>
</tbody>
</table>
NOCTURNAL ROSTRAL FLUID SHIFT

Circulation. 2010;121:1598-1605
PROGNOSTIC SIGNIFICANCE OF CENTRAL APNEAS THROUGHOUT A 24-HOUR PERIOD IN PATIENTS WITH HEART FAILURE

J Am Coll Cardiol 2017;70:1351–64)
INFLUENCE OF OBSTRUCTIVE SLEEP APNEA ON MORTALITY IN PATIENTS WITH HEART FAILURE

n—164 pts, LVEF < 45%

Table 3: Multivariate Hazards Ratios for Mortality Rate

<table>
<thead>
<tr>
<th>Variables</th>
<th>Hazard Ratio</th>
<th>p Value</th>
<th>95% Confidence Interval</th>
</tr>
</thead>
<tbody>
<tr>
<td>Untreated OSA</td>
<td>2.81</td>
<td>0.029</td>
<td>1.11 - 7.10</td>
</tr>
<tr>
<td>LVEF</td>
<td>0.93</td>
<td>0.006</td>
<td>0.88 - 0.98</td>
</tr>
<tr>
<td>NYHA functional class</td>
<td>2.30</td>
<td>0.037</td>
<td>1.04 - 5.08</td>
</tr>
<tr>
<td>Age</td>
<td>1.05</td>
<td>0.005</td>
<td>1.02 - 1.09</td>
</tr>
</tbody>
</table>

J Am Coll Cardiol 2007;49:1625–31
SLEEP DISORDERED BREATHING AND POST-DISCHARGE MORTALITY IN PATIENTS WITH ACUTE HEART FAILURE

N=1117 hospitalized AHF, LVE<45%, 31% had CSA, 47% OSA
TREATMENT RECOMMENDATIONS
TREATMENT OF SDB IN HEART FAILURE
LIFESTYLE MEASURES

• Weight loss, elastic stockings and exercise

• Positional therapy: using a wedge or cushion or sewing a pocket filled with tennis balls on the back of a pyjama shirt can discourage sleep in the supine position.

• Alcohol, sedatives, narcotics, and muscle relaxants should be avoided.

• Oral appliances, worn during sleep may be effective in select patients with OSA and retrognathism, particularly if the SDB is mild or positional.

• Although surgical methods of ameliorating SDB have not been specifically tested in HF, there may be a limited role for such intervention in carefully selected cases with OSA and a BMI >35 kg/m²
Positional sleep apnoea was defined as a >50% reduction in the AHI between the supine and the lateral position.
### SUMMARY OF STUDIES EVALUATING THE IMPACT OF MEDICAL TREATMENT ON THE APNOEA–HYPOPNOEA INDEX OF CHF PATIENTS

<table>
<thead>
<tr>
<th>Study</th>
<th>Type of study</th>
<th>No. of patients</th>
<th>LVEF (%)</th>
<th>Treatment</th>
<th>AHI (number of events per hour)</th>
<th>AI (number of events per hour)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Walsh et al.(^{15})</td>
<td>OO</td>
<td>9</td>
<td>&lt;30</td>
<td>Captopril 75 mg/day 4 weeks</td>
<td>35 ± 7</td>
<td>20 ± 5</td>
</tr>
<tr>
<td>Soln et al.(^{16})</td>
<td>OO</td>
<td>7</td>
<td>18.9 ± 1.3</td>
<td>Diuretics: 6 patients ACE inhibitor: 4 patients Carvedilol: 2 patients Nitrates: 2 patients 1 to 6 months</td>
<td>38.5 ± 7.7 (central)</td>
<td>18.1 ± 5.8 (central)</td>
</tr>
<tr>
<td>Javaheri et al.(^{17})</td>
<td>CO and DB</td>
<td>12</td>
<td>19 ± 6</td>
<td>Acetazolamide 3.5 mg/kg/day 6 days</td>
<td>55 ± 24</td>
<td>34 ± 20</td>
</tr>
<tr>
<td>Tamura et al.(^{18})</td>
<td>OO</td>
<td>5</td>
<td>36 ± 8.6</td>
<td>Carvedilol 2.5 to 20 mg/day 6 months</td>
<td>28.8 ± 7.5 (central)</td>
<td>12.4 ± 9.1 (central)</td>
</tr>
<tr>
<td>Bucca et al.(^{19})</td>
<td>OO</td>
<td>15</td>
<td>65.85 ± 1.7</td>
<td>Furosemide 40 mg/day Spironolactone 200 mg/day 3 days</td>
<td>74.89 ± 6.95 (obstructive)</td>
<td>57.17 ± 5.4</td>
</tr>
<tr>
<td>Tamura et al.(^{20})</td>
<td>OO</td>
<td>19</td>
<td>32 ± 7.4</td>
<td>Carvedilol 2.5 to 20 mg/day 6 months</td>
<td>34 ± 13 (central)</td>
<td>14 ± 13</td>
</tr>
</tbody>
</table>

*ESC Heart Failure 2018; 5: 222–230*
INFLUENCE OF CRT ON DIFFERENT TYPES OF SLEEP DISORDERED BREATHING

![Graph 1: AHI (events/hour) for different types of sleep disorders](image1.png)

![Graph 2: AHI (events/hour) by response to CRT](image2.png)
CARDIOVASCULAR EFFECTS OF CPAP IN PATIENTS WITH HEART FAILURE AND OBSTRUCTIVE SLEEP APNEA

N=24pts, LVEF<45%, NYHA: II-III, 1-month therapy

SLEEP APNEA TESTING AND OUTCOMES IN A LARGE COHORT OF MEDICARE BENEFICIARIES WITH NEWLY DIAGNOSED HEART FAILURE

Percent of Cohort Alive

- Tested, Diagnosed, Treated, N=258
- Not Tested, Not Treated, N=30,065

Hazard ratio = .33 (95% CI = .21-.51), P < .0001

Percent of Cohort Alive

- Tested, Diagnosed and Treated, N=258
- Tested, Diagnosed and Not treated, N=295

Hazard ratio = .49 (95% CI = .29-.84), P=0.009
CPAP FOR CENTRAL SLEEP APNEA AND HEART FAILURE CANPAP TRIAL

N=358 pts, LVEF<40, NYHA: II-III

The effects are sustained with long-term therapy

CPAP FOR CENTRAL SLEEP APNEA AND HEART FAILURE
CANPAP TRIAL

N=358 pts, LVEF<40, NYHA: II-III

CPAP patients were divided post hoc into those whose apnea-hypopnea index was or was not reduced below 15

ADAPTIVE SERVO VENTILATION (ASV)

ADAPTIVE SERVO-VENTILATION THERAPY FOR PATIENTS WITH CHRONIC HEART FAILURE SAVIOR-C

N=213 pts, LVEF <40%, NYHA ≥II

Circ J 2015; 79: 981 – 990
N=1325 patients
NYHA II-IV
LVEF≤45%
AHI≥ 15/h
with >50% central events
Adherence to therapy
the use of ASV ≥ 3 hours per night.
The target was to reduce the AHI < 10/ h

The primary end point in the time-to-event analysis was the first event of death from any cause, lifesaving cardiovascular intervention or unplanned hospitalization for worsening heart failure

ADAPTIVE SERVO-VENTILATION FOR CENTRAL SLEEP APNEA IN SYSTOLIC HEART FAILURE SERVE-HF

B Death from Any Cause

C Death from Cardiovascular Causes

Hazard ratio, 1.28 (95% CI, 1.06–1.55) 
P=0.01

Hazard ratio, 1.34 (95% CI, 1.09–1.65) 
P=0.006

Treated SDB had a better outcome than untreated severe SDB after adjustment for confounding factors. Subgroup analysis that included only OSA showed a similar result after adjustment.

European Journal of Heart Failure (2012) 14, 1009–1019
PERSPECTIVES AND NEEDS

• Many patients with central sleep apnea have a variable proportion of events that are obstructive and it can be hard to accurately distinguish between obstructive and central event subtypes. Prognosis and variations in treatment response may reflect differences in the pathophysiology of these events, which are not well characterized.

• New studies may benefit from careful identification of sub-groups most likely to respond to the intervention, larger sample size (allowing detection of clinically relevant effects across stratum), and incorporation of methods for improving treatment adherence over long periods of observation.

• There is a need to understand whether CSA-CSR itself is an adaptive or harmful condition in heart failure and whether chronic use of pressure-based devices (CPAP or ASV) adversely affects cardiac function in patients with heart failure.

• The most common diagnostic metric used for characterizing sleep apnea severity, the apnea hypopnea index (AHI), does not strongly predict adverse health outcomes or response to treatment.

• Small proportion of patients may develop central apneas when exposed to positive pressure therapy, a disorder termed “complex sleep apnea.”
DESIGN OF THE EFFECT OF ADAPTIVE SERVO-VENTILATION ON SURVIVAL AND CARDIOVASCULAR HOSPITAL ADMISSIONS IN PATIENTS WITH HEART FAILURE AND SLEEP APNOEA: THE ADVENT-HF TRIAL

- ADVENT-HF is a multicentre, multinational, randomized, parallel-group, open-label trial with blinded assessment of endpoints of standard medical therapy for HFrEF alone vs. with the addition of ASV in patients with HFrEF and SDB.

- Patients with a history of HFrEF undergo echocardiography and polysomnography.

- Those with a LVEF ≤45% and SDB (apnoea–hypopnoea index ≥15) are eligible. SDB is stratified into OSA with ≥50% of events obstructive or CSA with >50% of events central.

*European Journal of Heart Failure (2017) 19, 579–587*
PHENOTYPES IN OBSTRUCTIVE SLEEP APNEA: A DEFINITION, EXAMPLES AND EVOLUTION OF APPROACHES

Data levels
- Risk factor / environment
- Clinical
- Pathophysiologic
- Biologic
- Genetic/omic

Component examples
- Allergens
- Sleep patterns
- Alcohol
- Medications
- Obesity
- CV d/o
- Age
- Metabolic d/o
- Cancer
- Sex
- Neurocognition
- PSG patterns
- Muscle responsiveness
- UA anatomy
- Lung volumes
- Ventilatory drive
- Sleep stability
- Arousalability
- Neurohormonal changes
- Inflammation
- Fibrinolytic imbalance
- Oxidative stress
- Endothelial dysfunction
- Age
- Pharmacogenomics
- Epigenetics
- GWA
- miRNA
- ncDNA

Potential clinical relevance (selected examples)
- Lifestyle
  - Modifiable factors (weight loss)
- Clinical phenotypes
  - Integrated care
  - Risk stratification (EDS, elderly)
  - Comprehensive guidelines
- Intermediate phenotypes
  - Therapeutic targets (oxygen, sedatives)
  - Diagnostic (PALM)
  - Therapy response (CCC)
- Biomarkers
  - Diagnostic (IL-6, IL-10)
  - Therapeutic targets
  - Sequelae predisposition
- Genetic risk assessment
  - OSA risk
  - Sequelae predisposition
  - Response to therapy (miRNAs & resistant HTN)

Sleep Med Rev. 2017 October; 35: 113–123
EMERGING THERAPIES
RANDOMIZED CONTROLLED TRIAL OF AN ORAL APPLIANCE (SOMNODENT) FOR SLEEP-DISORDERED BREATHING AND CARDIAC FUNCTION IN PATIENTS WITH HEART FAILURE

Clinical Cardiology. 2018;41:1009–1012
# Effects of Nocturnal Oxygen Therapy in Patients With Chronic HF and CSA

**CHF-HOT Study**

<table>
<thead>
<tr>
<th>Table 4: Effects of nocturnal oxygen on sleep, QOL, cardiac function, and arrhythmias</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Groups</strong></td>
</tr>
<tr>
<td></td>
</tr>
<tr>
<td><strong>AHI (events/h)</strong></td>
</tr>
<tr>
<td>HOT</td>
</tr>
<tr>
<td>Control</td>
</tr>
<tr>
<td><strong>CAI (events/h)</strong></td>
</tr>
<tr>
<td>HOT</td>
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<tr>
<td>Control</td>
</tr>
<tr>
<td><strong>OAI (events/h)</strong></td>
</tr>
<tr>
<td>HOT</td>
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<tr>
<td>Control</td>
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<tr>
<td><strong>HI (events/h)</strong></td>
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<tr>
<td>HOT</td>
</tr>
<tr>
<td>Control</td>
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<tr>
<td><strong>ODI (dips/h)</strong></td>
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<tr>
<td>HOT</td>
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<tr>
<td>Control</td>
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<tr>
<td><strong>PaCO₂ (mmHg)</strong></td>
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<tr>
<td>HOT</td>
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<tr>
<td>Control</td>
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<tr>
<td><strong>SAS (Mets)</strong></td>
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<tr>
<td>HOT</td>
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<tr>
<td>Control</td>
</tr>
<tr>
<td><strong>HR (bpm)</strong></td>
</tr>
<tr>
<td>HOT</td>
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<tr>
<td>Control</td>
</tr>
<tr>
<td><strong>LVEF (%)</strong></td>
</tr>
<tr>
<td>HOT</td>
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<tr>
<td>Control</td>
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<tr>
<td><strong>CTR (%)</strong></td>
</tr>
<tr>
<td>HOT</td>
</tr>
<tr>
<td>Control</td>
</tr>
<tr>
<td><strong>BNP (pg/ml)</strong></td>
</tr>
<tr>
<td>HOT</td>
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<tr>
<td>Control</td>
</tr>
<tr>
<td><strong>ANP (pg/ml)</strong></td>
</tr>
<tr>
<td>HOT</td>
</tr>
<tr>
<td>Control</td>
</tr>
<tr>
<td><strong>NE (pg/ml)</strong></td>
</tr>
<tr>
<td>HOT</td>
</tr>
<tr>
<td>Control</td>
</tr>
<tr>
<td><strong>PVCs (b/h)</strong></td>
</tr>
<tr>
<td>HOT</td>
</tr>
<tr>
<td>Control</td>
</tr>
<tr>
<td><strong>% PVCs (%)</strong></td>
</tr>
<tr>
<td>HOT</td>
</tr>
<tr>
<td>Control</td>
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</tbody>
</table>
ACETAZOLAMIDE IMPROVES CENTRAL SLEEP APNEA IN HEART FAILURE

Because metabolic acidosis induced by acetazolamide, the difference between the prevailing Pco2 and the apneic threshold Pco2 increased. As a result of this, acetazolamide decreased the likelihood of developing sleep apnea.

The remede system is an implantable device which transvenously stimulates a nerve causing diaphragmatic contraction similar to normal breathing.

N=151 pts with AHI>20/h
PHRENIC NERVE STIMULATION FOR THE TREATMENT OF CENTRAL SLEEP APNEA

N=47 pts, 79%HF with CSA

55% reduction in apnea-hypopnea index from baseline to 3 months.
Central apnea index, oxygenation, and arousals significantly improved.

*J Am Coll Cardiol HF 2015;3:360–9*
UPPER-AIRWAY STIMULATION FOR OBSTRUCTIVE SLEEP APNEA
STAR-TRIAL

![Diagram showing upper-airway stimulation for obstructive sleep apnea with STAR-TRIAL results.](image)

**N Engl J Med 2014;370:139-49**
Key topics and self-care skills to include in patient education and the professional behaviours to optimize learning and facilitate shared decision making

<table>
<thead>
<tr>
<th>Recommendations</th>
<th>Class</th>
<th>Level</th>
<th>Ref</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sleep apnoea</td>
<td>III</td>
<td>B</td>
<td>473</td>
</tr>
</tbody>
</table>

Adaptive servo-ventilation is not recommended in patients with HFrEF and a predominant central sleep apnoea because of an increased all-cause and cardiovascular mortality.
## Recommendations for Treatment of Sleep Disorders

<table>
<thead>
<tr>
<th>COR</th>
<th>LOE</th>
<th>Recommendations</th>
<th>Comment/Rationale</th>
</tr>
</thead>
<tbody>
<tr>
<td>IIIa</td>
<td>C-LD</td>
<td>In patients with NYHA class II–IV HF and suspicion of sleep-disordered breathing or excessive daytime sleepiness, a formal sleep assessment is reasonable.(^{200,201})</td>
<td><strong>NEW:</strong> Recommendation reflects clinical necessity to distinguish obstructive versus central sleep apnea.</td>
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<td><a href="#">See Online Data Supplement G.</a></td>
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</tbody>
</table>

| IIb | B-R | In patients with cardiovascular disease and obstructive sleep apnea, CPAP may be reasonable to improve sleep quality and daytime sleepiness.\(^{204}\) | **NEW:** New data demonstrate the limited scope of benefit expected from CPAP for obstructive sleep apnea.                                      |
|     |     | [See Online Data Supplement G.](#)                                                |                                                                                                                                                    |

## Recommendations for Treatment of Sleep Disorders (Continued)

<table>
<thead>
<tr>
<th>COR</th>
<th>LOE</th>
<th>Recommendations</th>
<th>Comment/Rationale</th>
</tr>
</thead>
<tbody>
<tr>
<td>III: Harm</td>
<td>B-R</td>
<td>In patients with NYHA class II–IV HFrEF and central sleep apnea, adaptive servo-ventilation causes harm.(^{203})</td>
<td><strong>NEW:</strong> New data demonstrate a signal of harm when adaptive servo-ventilation is used for central sleep apnea.</td>
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<td><a href="#">See Online Data Supplement G.</a></td>
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## Recommendations

Purposeful weight loss via healthy dietary intervention and physical activity for the purposes of improving health-related QOL or managing comorbidities such as diabetes mellitus, hypertension, or sleep apnea may be reasonable in obese patients with HF.

<table>
<thead>
<tr>
<th>COR</th>
<th>LOE</th>
<th>Referenced Guideline</th>
<th>References</th>
</tr>
</thead>
<tbody>
<tr>
<td>IIb</td>
<td>C</td>
<td>6, 8, 29</td>
<td>202, 228, 230–236</td>
</tr>
</tbody>
</table>
TAKE HOME MESSAGES

Sleep breathing disorder is highly prevalent in HF (>50%)

Patients with more severe HF are more likely to suffer from mixed or CSA

Sleepiness scales are nor reliable in HF

AHI>5 indicates the presence of SBD

Guideline recommended therapy is the 1st step

CPAP, BiPAP and ASV may be beneficial in OSA

CPAP improves LVEF and 6-MWD but not prognosis in CSA
THANK YOU