Exploration of Medicine

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Open Access Original Article

Right ventricular echocardiographic parameters predict severe sleep apnea syndrome in patients with heart failure

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Academic Editor: Akiko Mammoto, Medical College of Wisconsin, USA
Received: January 31, 2025 **Accepted:** March 31, 2025 **Published:** April 23, 2025

Cite this article: Antit S, Yangui F, Louati M, Boufares A, Abdellatif S, Charfi MR, et al. Right ventricular echocardiographic parameters predict severe sleep apnea syndrome in patients with heart failure. Explor Med. 2025;6:1001311. https://doi.org/10.37349/emed.2025.1001311

Abstract

Aim: The study aimed to determine which right ventricle echocardiography parameters were associated with severe sleep apnea syndrome in heart failure patients with sleep apnea syndrome.

Methods: A cross-sectional monocentric study was conducted, including 85 patients with stable heart failure. All patients underwent home respiratory polygraphy and transthoracic echocardiography with evaluation of the right ventricular echocardiographic parameters and sleep apnea syndrome severity.

Results: The average age of the population was 69 years. The median left ventricular ejection fraction was 38% (33; 52). Sleep apnea syndrome was diagnosed in 71 patients (83.5%), with obstructive sleep apnea in 50 patients (58.8%) and central sleep apnea in 21 patients (24.7%). Severe sleep apnea syndrome was observed in 31% (n = 22) of patients. In the univariate analysis, right ventricle Tei index with a cutoff value > 0.50 (OR = 3; 95% CI = 1.06–8.56; p = 0.035), right ventricle fractional area change < 40 (OR = 4; 95% CI = 1.38–11; p = 0.09) and right ventricle free wall longitudinal strain with a cut-off value > –17 (OR = 3.5; 95% CI = 1.15–9.41; p = 0.002) increased the risk of having severe sleep apnea syndrome. In the multivariate analysis, the right ventricle free wall longitudinal strain was the only independent predictor for severe sleep apnea syndrome [hazard ratio (HR) = 0.892; 95% CI = 0.816–0.979; p = 0.036].

Conclusions: Right ventricle free wall longitudinal strain seems to be a strong predictor of severe sleep apnea syndrome in patients with stable chronic heart failure and sleep apnea syndrome.

Keywords

Sleep apnea syndrome, right ventricular function, echocardiography, heart failure

Introduction

Sleep apnea syndrome (SAS) is a common chronic sleep-related breathing disorder, affecting approximately 5% of middle-aged adults, predominantly men [1, 2]. Currently considered a potentially dangerous disease, SAS poses a growing healthcare concern, increasing economic and social burdens [3]. A well-established link exists between SAS and cardiovascular morbidity and mortality, as it is implicated in a range of cardiovascular conditions, including heart failure (HF), coronary artery disease, and stroke. Additionally, SAS is associated with cardiac rhythm and conduction disorders and a high risk of sudden cardiac death during sleep [4, 5]. SAS includes a wide range of sleep-disordered breathing conditions, from central to mixed apnea, as well as obstructive apnea and hypopnea [6]. Both central and obstructive sleep apneas (OSAs) are frequently observed, with OSA occurring in 17% to 61% of cases and central sleep apnea (CSA) in 35% to 39% [7]. Polysomnography remains the gold standard for diagnosing SAS. Based on the apnea-hypopnea index (AHI), SAS severity is classified into mild, moderate, and severe forms (AHI 5–14, 15–29, and ≥ 30 events/hour, respectively) [3].

Several studies have highlighted an increased incidence of cardiac structural and functional alterations in SAS patients, particularly left ventricular hypertrophy and diastolic dysfunction [8, 9]. However, the effects of SAS on the right ventricle (RV) remain a subject of debate.

The study aimed to identify RV echocardiography parameters that predict severe SAS in patients with stable chronic HF and concomitant SAS.

Materials and methods

Study design and patient enrollment

This was a cross-sectional, descriptive, monocentric study conducted from January to June 2024 in the cardiology and pulmonary departments of the Internal Security Forces Hospital in Tunisia.

The study received approval from the Ethics Committee of the Internal Security Forces Hospital (Ethical approval number: 27/24). Informed consent for participation was obtained from all participants.

Study population

Patients aged over eighteen years with stable HF and on an optimized HF therapy at target dose were included in the study. Non-inclusion criteria were the history of SAS or ongoing continuous positive airway pressure treatment, severe renal failure, severe neurological diseases, or therapy that might affect the respiratory drive, and the non-obtention of the patient's consent. Patients with poor-quality polygraph recordings or suboptimal echogenicity were also excluded. Poor recording quality was defined as a duration of less than six hours or invalid respiratory flow or effort signals.

Clinical evaluation

Clinical and paraclinical data were collected for each patient, including age, gender, cardiovascular risk factors, comorbidities, symptoms, physical examination, anthropometric data (body mass index, neck and waist circumferences), HF etiology, and the current HF medication, polygraphy, and transthoracic echocardiography (TTE) results.

Chronic HF was defined according to 2021 European Society of Cardiology guidelines for the diagnosis and treatment of acute and chronic HF, by the presence of symptoms and/or signs of HF and objective evidence of cardiac dysfunction [10]. Patients were classified into three categories based on left ventricular ejection fraction (LVEF): HF with preserved ejection fraction (LVEF ≥ 50%), HF with mildly reduced ejection fraction (40% ≤ LVEF ≤ 49%), or HF with reduced ejection fraction (LVEF < 40%) according to European guidelines.

Respiratory polygraphy

All our patients underwent an overnight home respiratory polygraphy.

SAS was confirmed according to the International Classification of Sleep Disorders, as outlined by the American Academy of Sleep Medicine guidelines [11].

Apnea was defined as a complete cessation of airflow lasting 10 seconds or longer or a significant decrease in respiratory airflow of at least 95% for at least 10 seconds. Hypopnea was defined as a significant reduction in respiratory airflow of at least 50% for at least 10 seconds or a decrease in respiratory airflow of < 50% for at least 10 seconds accompanied by a drop of oxygen saturation by 3% or more. Respiratory events were classified as obstructive in the presence of thoracoabdominal movements and central in their absence. The AHI was defined as the mean number of apnea and hypopnea episodes per hour of sleep monitoring [11]. SAS was diagnosed when the AHI was superior to or equal to 5 respiratory events per hour of recording time.

Patients were classified based on the AHI values. SAS severity was categorized into three levels: mild SAS (5 ≤ AHI < 15 events per hour), moderate SAS (15 ≤ AHI < 30 events per hour), and severe SAS (AHI ≥ 30 events per hour) [11]. The type of SAS was classified based on the most prevalent events: OSA with ≥ 50% obstructive events per hour, and CSA with ≥ 50% central events per hour.

Echocardiographic evaluation

All patients included underwent a transthoracic two-dimensional conventional Doppler echocardiography, performed using a Philips EPIQ 7C equipped with a 5S1 transducer.

All echocardiographic examinations were conducted by a single operator to limit the variations in measurement acquisition, with simultaneous and continuous electrocardiographic monitoring. Measurements were made following the guidelines established by the American Society of Echocardiography and the European Association of Cardiovascular Imaging [12].

RV systolic function was assessed using several parameters, including right ventricular free wall longitudinal strain (RVFWLS), RV fractional area change (RV FAC), RV Tei index, tricuspid annular plane systolic excursion (TAPSE), and peak systolic velocity of the tricuspid annulus (RV S').

The RVFWLS was assessed by applying dedicated LV strain on the RV, and the results were obtained by averaging the strain measurements from the apical, middle, and basal regions of the RV free wall. The RV FAC was measured in the apical four-chamber view and calculated as the difference in the end-diastolic area (EDA) and end-systolic area (ESA) divided by the EDA using the formula: RV FAC (%) = 100 × (EDA – ESA)/EDA. The RV Tei index was measured in tissue Doppler by summing the isovolumic contraction time (IVCT) and isovolumic relaxation time (IVRT), and relating this to the ejection time (ET) with the formula: RV Tei index = (IVCT + IVRT)/ET. The TAPSE is measured by placing the M-mode cursor through the lateral portion of the tricuspid valve annulus in the apical four-chamber view, with the displacement of the annulus between end-diastole and peak systole being recorded [13].

Endpoints

This study focused on RV echocardiographic parameters to determine their predictive value for severe SAS.

Patients were categorized into two groups according to SAS severity: Group A with severe SAS and Group B with mild to moderate SAS.

Legal and ethical aspects

Informed consent was obtained from all patients, and measures were taken to ensure the confidentiality of their data.

We have no conflicts of interest to declare.

Statistical analysis

Data were recorded and analyzed using IBM SPSS Statistics 23 software.

Baseline characteristics were summarized using mean ± standard deviation or median ± interquartile range (25th–75th percentiles), as appropriate, for continuous data, and counts with percentages for categorical data. The Kolmogorov-Smirnov test was used to assess the normality of continuous variable distributions.

Comparisons of means between independent groups were performed using the Student's *t*-test for independent samples. The Pearson chi-square test was used to compare percentages between independent groups. The determination of threshold values for the studied parameters was conducted by analyzing their receiver operating characteristic (ROC) curves.

Multinomial logistic regression analysis was performed for the multivariate analysis. Ultrasound parameters with a *p*-value < 0.05 in the univariate analysis were included in the multivariate analysis.

In all statistical tests, a *p*-value < 0.05 was considered statistically significant.

Results

We enrolled 85 patients from our outpatient department for stable HF.

A total of 71 patients (83.5%) were diagnosed with SAS. OSA was observed in 50 patients (58.8%) and CSA in 21 patients (24.7%). Mild SAS was more frequent. It was observed in 32 patients (37.6%), moderate SAS in 17 patients (20%), and severe SAS in 22 patients (25.8%). The characteristics of patients with SAS were summarized in Table 1.

Table 1. General characteristics of the study population and comparison of these characteristics between the two groups according to sleep apnea syndrome severity

Parameter	SAS patients	Group A (*n* = 22)	Group B (*n* = 49)	*p*-value*
Age, Me (25%; 75%)	69 (52; 71)	64 (55; 71)	61 (52; 70)	0.480
Sex ratio (Male/Female, %)	56/15 (3.73)	19/3 (6.33)	37/12 (3.08)	0.301
Hypertension (*n*, %)	33 (46.5)	7 (31.8)	26 (53.1)	0.097
Diabetes (*n*, %)	43 (60.6)	16 (72.7)	27 (55.1)	0.160
Dyslipidemia (*n*, %)	31 (43.7)	9 (40.9)	22 (44.9)	0.754
Smoking (*n*, %)	45 (63.4)	13 (59.1)	32 (65.3)	0.615
Chronic kidney disease (*n*, %)	14 (19.7)	4 (18.2)	10 (20.4)	0.827
Chronic obstructive pulmonary disease (*n*, %)	8 (11.3)	3 (13.6)	5 (10.2)	0.672
Body mass index (*n*, kg/m²)	29 ± 4	29 ± 4	28 ± 5	0.643
Heart failure with preserved ejection fraction (*n*, %)	26 (36.6)	7 (31.8)	19 (38.7)	0.574
Heart failure with mildly reduced ejection fraction (*n*, %)	10 (14.1)	3 (13.6)	7 (14.3)	0.942
Heart failure with reduced ejection fraction (*n*, %)	35 (49.3)	12 (54.5)	23 (46.9)	0.553

Significance level at *p*-value < 0.05; * comparison between Group A and Group B; Me (LQ; UQ): data presented as median with lower and upper quartiles

Smoking and diabetes were the most common cardiovascular risk factors, present in 63.4% (*n* = 45) and 60.6% (*n* = 43) of patients. Eight patients (11.3%) had chronic obstructive pulmonary disease.

Medication included beta-blockers in 81.7% (58 patients), diuretics in 62.0% (44 patients), ACE inhibitors in 59.2% (42 patients), spironolactone in 53.5% (38 patients), sodium-glucose cotransporter-2 inhibitors in 42.3% (30 patients) and angiotensin II receptor blockers in 11.3% (8 patients).

The etiology of HF in our study population was ischemic in 59.2% (42 patients), idiopathic in 16.9% (12 patients), arrhythmias in 8.5% (6 patients), hypertensive in 9.9% (7 patients), and others in 5.6% (4 patients).

For the ultrasound parameters, the median value of LVEF was 38% (33; 52). Within this population, 49.3% of patients exhibited reduced ejection fraction, 14.1% had mildly reduced ejection fraction, and 36.6% had preserved ejection fraction.

The comparison of clinical and echocardiographic data between Group A (22 patients: 31%) and Group B (49 patients: 69%) was reported in Tables 1 and 2.

Table 2. Echocardiographic data of the study population and comparison of these data between the two groups according to sleep apnea syndrome severity

Parameter	SAS patients	Group A (*n* = 22)	Group B (*n* = 49)	*p*-value*
End-diastolic diameter of left ventricular (mm)	59 ± 8	61 ± 8	58 ± 8	0.432
Indexed left ventricular mass (g/m²), Me (25%; 75%)	111 (89; 134)	126 (108; 136)	110 (90; 130)	0.252
Left ventricular ejection fraction (%), Me (25%; 75%)	38 (33; 52)	37 (25; 56)	39 (34; 50)	0.645
Global longitudinal strain (%)	–12.8 ± 5	–12.8 ± 5	–13.03 ± 4.52	0.126
Indexed left atrial volume (mL/m²), Me (25%; 75%)	44 (36; 58)	47 (36; 56)	44.6 (35; 58)	0.610
E wave velocity (cm/s)	72 ± 17	74 ± 14	71 ± 19	0.452
A wave velocity (cm/s)	46 ± 20	64 ± 26	64 ± 28	0.928
E/A ratio, Me (25%; 75%)	1.2 (0.7; 1.8)	1.3 (0.7; 2.1)	1.1 (0.7; 1.6)	0.300
Mean E/e' ratio, Me (25%; 75%)	11 (8.5; 15)	13 (9; 14)	10 (7.6; 15)	0.398
Systolic pulmonary arterial pressure (mmHg)	43 ± 16	46 ± 18	41 ± 14	0.125
Right ventricular diameter (mm), Me (25%; 75%)	37 (33; 43)	38 (31; 43)	36 (34; 40)	0.649
Tricuspid annular plane systolic excursion (mm), Me (25%; 75%)	20 (16; 22)	18 (15; 21)	20 (17; 22)	0.434
Right ventricular fractional area change (%), Me (25%; 75%)	44 (34; 49)	37 (28; 44)	45 (39; 49)	0.005
S' velocity of right ventricular (cm/s)	10.7 ± 2	10 ± 2	11 ± 2	0.124
Right ventricular free wall longitudinal strain (%), Me (25%; 75%)	–20 (–21; –17)	–16 (–20; –10)	–20 (–22; –19)	0.008
Right ventricular Tei index, Me (25%; 75%)	0.48 (0.42; 0.56)	0.51 (0.47; 0.59)	0.44 (0.40; 0.63)	0.012
Right atrial surface (cm²), Me (25%; 75%)	17 (15; 22)	18.7 (16; 25)	17 (14; 21)	0.027

Significance level at *p*-value < 0.05; * comparison between Group A and Group B; Me (LQ; UQ): data presented as median with lower and upper quartiles

There were no significant differences between the two groups regarding cardiovascular risk factors, HF phenotype, or left-heart ultrasound parameters, including end-diastolic diameter, indexed left ventricular mass, LVEF, global longitudinal strain, indexed left atrial volume, and mean E/e' ratio (all $p > 0.05$). However, RV FAC ($p = 0.005$), RVFWLS ($p = 0.008$), RV Tei index ($p = 0.012$), and right atrial surface ($p = 0.027$) were significantly higher in severe sleep apnea. Systolic pulmonary arterial pressure, RV diameter, and TAPSE were similar between the two groups.

To further investigate the relationship between right heart ultrasound parameters and severe SAS, ROC curve analysis was performed to determine the cut-off values of echocardiographic parameters associated with severe SAS, as observed in the above comparison.

Univariate analysis revealed that RV FAC < 40 [air under the curve (AUC) = 0.693, $p = 0.010$] increased the likelihood of belonging to Group A by 4 (OR = 4; 95% CI = 1.38–11; $p = 0.09$). For RVFWLS, a cut-off value > –17 (AUC = 0.699, $p = 0.008$) increased the likelihood of belonging to Group A by 3.5 (OR = 3.5; 95% CI = 1.15–9.41; $p = 0.002$). An RV Tei index > 0.50 (AUC = 0.692, $p = 0.010$) increased the likelihood of being classified in Group A by a factor of 3 (OR = 3; 95% CI = 1.06–8.56; $p = 0.035$).

A multivariate study identified RVFWLS [hazard ratio (HR) = 0.892; 95% CI = 0.816–0.979; $p = 0.036$] as an independent predictor of severe SAS after binary logistic regression analysis, as shown in Table 3. A one-point decrease in RVFWLS was associated with an 11% increase in the risk of developing severe SAS.

Table 3. Multivariate analysis

Parameter	*p*-value	HR	95% CI
Right ventricular fractional area change	0.081	0.944	0.884–1.007
Right ventricular free wall longitudinal strain	0.036	0.892	0.816–0.979
Right ventricular Tei index	0.669	1.037	0.975–1.103

HR: hazard ratio

Discussion

This cross-sectional study included 71 patients (83.5%) diagnosed with chronic HF and SAS.

The study revealed a notably high prevalence (83.5%) of SAS in HF patients, underscoring the significant association between these two major public health concerns. This finding raises the question of whether routine screening for SAS should be implemented in HF patients. According to the literature, the prevalence of SAS in HF patients ranges from 46% to 85% [12–17]. Using an AHI threshold of ≥ 5/h, several studies have reported that the prevalence of SAS in HF patients varies between 60% and 85% [14–19]. Using an AHI threshold of ≥ 5/h, several studies have reported that the prevalence of SAS in HF patients varies between 60 and 85% [16, 19–22]. The elevated frequency of SAS in our population can be explained by the high frequency of patients with HF with reduced and moderately reduced ejection fractions.

In patients with chronic HF, the study found that while RVFWLS, RV Tei index, and RV FAC were associated with severe SAS in univariate analysis, only RVFWLS, with a threshold of –17, emerged as an independent predictor of severe SAS in HF patients with SAS. A 1% decrease in RVFWLS was associated with an 11% increase in the risk of developing severe SAS (HR = 0.892; 95% CI = 0.816–0.979; $p = 0.036$).

Several studies have examined right ventricular remodeling and dysfunction in individuals with SAS, exploring the role of RV echocardiographic parameters in predicting the severity of SAS [23]. However, our study is the first to specifically investigate these parameters in patients with chronic HF, addressing a significant gap in the literature. Notably, there is a paucity of research on this topic within the context of HF, and our study contributes valuable insights in this area.

RV strain is highlighted as a more sensitive early imaging marker for assessing the longitudinal function of the RV compared to conventional echocardiographic parameters commonly used in estimating global and regional RV systolic function.

In a study by Tadic et al. [23], significant impairment in RVFWLS was observed in a large number of patients with obstructive SAS, demonstrating a progressive decline with increasing severity of SAS. RVFWLS exhibited more negative values (indicating worse strain) in patients with moderate SAS compared to those with mild SAS [standardized mean difference (SMD): 1.29 ± 0.21, 95% CI: 0.87–1.70, $p < 0.0001$, data from three studies]. Similarly, patients with severe SAS showed more negative RVFWLS values compared to those with mild SAS (SMD: 1.26 ± 0.20, 95% CI: 0.87–1.65, $p < 0.0001$, and data from three studies).

However, this study did not report a statistically significant difference between moderate and severe SAS. Notably, our study is unique in including patients with HF, a population that was excluded from the previous investigation.

Consistent with these findings, Hammerstingl et al. [9] reported a lower RV global longitudinal strain in subjects with higher AHI, showing a positive correlation with SAS severity. Tugcu et al. [5] also found significant correlations between AHI and various RV parameters, and RV mid-free wall strain and strain rate demonstrated the strongest associations. In our study, we specifically focused on RV-free wall strain to exclude the influence of the left ventricle. Notably, after performing binary logistic regression analysis, RVFWLS (HR = 0.892; 95% CI = 0.816–0.979; $p = 0.036$) emerged as an independent predictor for belonging to the severe SAS Group.

RV FAC provides an estimate of overall RV systolic function. In a study by Romero-Corral et al. [24], involving 85 cases of obstructive SAS, a severe decline in RV FAC was observed in the severe SAS group compared to the mild SAS Group (53 ± 7% vs. 57 ± 6%; $p = 0.01$). Consistent with these findings, our investigation revealed a significant association between RV FAC and severe SAS in the univariate analysis. An RV FAC (%) below 40 increased the likelihood of being classified as having severe SAS by a factor of 4 (OR = 4; 95% CI = 1.38–11; $p = 0.09$).

In a previous study conducted by Dursunoglu et al. [25], global right ventricular function was assessed using the RV Tei index in individuals diagnosed with SAS, excluding those with a history of cardiac or lung diseases. Their research revealed a robust correlation between the RV Tei index and AHI ($r = 0.84$; $p < 0.001$), which closely aligns with the findings in our study.

More recently, Akyol et al. [26] established a proportional relationship between the RV Tei index and the severity of obstructive SAS. Some studies have shown that continuous positive airway pressure therapy can improve the RV Tei index. Specifically, RV Tei index measurements taken at baseline and after 6 months of continuous positive airway pressure therapy demonstrated significant improvement in patients with OSA who did not have hypertension [25]. Romero-Corral et al. [24] highlighted altered right and left ventricular function in patients with obstructive SAS, particularly in those with moderate to severe SAS.

In our study, the RV Tei index, with a cutoff value > 0.50 (OR = 3; 95% CI = 1.06–8.56; $p = 0.035$), also demonstrated a correlation with the severity of SAS, though this was observed only in univariate analysis.

The impact of obstructive SAS on RV systolic function (RV S') remains controversial. Altekin et al. [27] in a study involving 79 patients, found no significant variation in RV S' across different severity grades of obstructive SAS, consistent with our findings. However, Shivalkar et al. [28] reported a significant correlation between AHI and RV S'.

In a study by Ibn Hadj Amor et al. [3] assessing right ventricular remodeling and dysfunction in obstructive SAS, TAPSE did not show a significant correlation with SAS severity. Similarly, Tadic et al. [23] observed a reduction in TAPSE in patients with SAS compared to controls, but this parameter proved insufficient for differentiating between SAS severity grades. Our study corroborates these findings.

To our knowledge, this is the first study to assess the role of RV echocardiographic parameters in predicting the severity of SAS in patients with both chronic HF and SAS. However, this study has limitations. It was a single-center study with a small number of patients, which limits the generalization of the results. Further multicenter studies with larger populations are required to confirm our findings.

In conclusion, we found that RVFWLS emerged as the only reliable indicator of SAS severity in individuals with both SAS and HF, underscoring its significant role in assessing SAS severity in this patient group. Therefore, a thorough assessment of RV function is necessary in HF patients with SAS, as it facilitates the detection of those with severe SAS.

Abbreviations

AHI: apnea-hypopnea index

AUC: air under the curve

CSA: central sleep apnea

EDA: end-diastolic area

ESA: end-systolic area

ET: ejection time

HF: heart failure

HR: hazard ratio

IVCT: isovolumic contraction time

IVRT: isovolumic relaxation time

LVEF: left ventricular ejection fraction

OSAs: obstructive sleep apneas

ROC: receiver operating characteristic

RV FAC: right ventricle fractional area change

RV S': peak systolic velocity of the tricuspid annulus

RV: right ventricle

RVFWLS: right ventricular free wall longitudinal strain

SAS: sleep apnea syndrome

SMD: standardized mean difference

TAPSE: tricuspid annular plane systolic excursion

Declarations

Author contributions

S Antit: Conceptualization, Investigation, Methodology. FY: Investigation, Writing—original draft, Writing—review & editing. ML: Writing—original draft, Formal analysis. AB and S Abdellatif: Data curation. MRC: Supervision, Validation. LZ: Validation, Visualization. All authors read and approved the submitted version.

Conflicts of interest

The authors declare that they have no conflicts of interest.

Ethical approval

The study was approved by the Ethics Committee of the Internal Security Forces Hospital (Ethical approval number: 27/24).

Consent to participate

Informed consent to participate in the study was obtained from all participants.

Consent to publication

Not applicable.

Availability of data and materials

The raw data supporting the conclusions of this manuscript will be made available by the authors, without undue reservation, to any qualified researcher.

Funding

Not applicable.

Publisher's note

References

1. Young T, Peppard PE, Gottlieb DJ. Epidemiology of Obstructive Sleep Apnea: A Population Health Perspective. Am J Respir Crit Care Med. 2002;165:1217–39. [DOI] [PubMed]
2. Hedner J, Grote L, Bonsignore M, McNicholas W, Lavie P, Parati G, et al. The European Sleep Apnoea Database (ESADA): report from 22 European sleep laboratories. Eur Respir J. 2011;38:635–42. [DOI] [PubMed]
3. Ibn Hadj Amor H, Touil I, Chebbi R, Bouchareb S, Bouchnak S, Brahem Y, et al. Assessment of right ventricular remodeling and dysfunction in obstructive sleep apnea syndrome: a prospective monocentric study. Sleep Breath. 2022;26:663–74. [DOI] [PubMed]
4. Chetan IM, Domokos Gergely B, Albu A, Tomoaia R, Todea DA. Understanding the role of echocardiography in patients with obstructive sleep apnea and right ventricular subclinical myocardial dysfunction—comparison with other conditions affecting RV deformation. Med Ultrason. 2021;23:213–9. [DOI] [PubMed]
5. Tugcu A, Yildirimtürk O, Tayyareci Y, Demiroglu C, Aytekin S. Evaluation of Subclinical Right Ventricular Dysfunction in Obstructive Sleep Apnea Patients Using Velocity Vector Imaging. Circ J. 2010;74:312–9. [DOI] [PubMed]
6. Cumpston E, Chen P. Sleep Apnea Syndrome. In: StatPearls [Internet]. Treasure Island (FL): StatPearls Publishing; 2025. Available from: http://www.ncbi.nlm.nih.gov/books/NBK564431/
7. Naughton MT. Epidemiology of central sleep apnoea in heart failure. Int J Cardiol. 2016;206:S4–7. [DOI] [PubMed]
8. Baguet J, Nadra M, Barone-Rochette G, Ormezzano O, Pierre H, Pépin J. Early cardiovascular abnormalities in newly diagnosed obstructive sleep apnea. Vasc Health Risk Manag. 2009;5:1063–73. [DOI] [PubMed] [PMC]
9. Hammerstingl C, Schueler R, Wiesen M, Momcilovic D, Pabst S, Nickenig G, et al. Impact of Untreated Obstructive Sleep Apnea on Left and Right Ventricular Myocardial Function and Effects of CPAP Therapy. PLoS One. 2013;8:e76352. [DOI] [PubMed] [PMC]
10. McDonagh TA, Metra M, Adamo M, Gardner RS, Baumbach A, Böhm M, et al.; ESC Scientific Document Group. 2021 ESC Guidelines for the diagnosis and treatment of acute and chronic heart failure: Developed by the Task Force for the diagnosis and treatment of acute and chronic heart failure of the European Society of Cardiology (ESC) With the special contribution of the Heart Failure Association (HFA) of the ESC. Eur Heart J. 2021;42:3599–726. [DOI] [PubMed]
11. The AASM International Classification of Sleep Disorders—Third Edition, Text Revision (ICSD-3-TR) [Internet]. American Academy of Sleep Medicine; c2025 [cited 2023 Nov 12]. Available from: https://aasm.org/clinical-resources/international-classification-sleep-disorders/
12. Rudski LG, Lai WW, Afilalo J, Hua L, Handschumacher MD, Chandrasekaran K, et al. Guidelines for the Echocardiographic Assessment of the Right Heart in Adults: A Report from the American Society of Echocardiography Endorsed by the European Association of Echocardiography, a registered branch of the European Society of Cardiology, and the Canadian Society of Echocardiography. J Am Soc Echocardiogr. 2010;23:685–713. [DOI] [PubMed]
13. DiLorenzo MP, Bhatt SM, Mercer-Rosa L. How best to assess right ventricular function by echocardiography. Cardiol Young. 2015;25:1473–81. [DOI] [PubMed] [PMC]
14. Yumino D, Wang H, Floras JS, Newton GE, Mak S, Ruttanaumpawan P, et al. Prevalence and Physiological Predictors of Sleep Apnea in Patients With Heart Failure and Systolic Dysfunction. J Card Fail. 2009;15:279–85. [DOI] [PubMed]
15. Schulz R, Blau A, Börgel J, Duchna HW, Fietze I, Koper I, et al.; working group Kreislauf und Schlaf of the German Sleep Society (DGSM). Sleep apnoea in heart failure. Eur Respir J. 2007;29:1201–5. [DOI] [PubMed]

16. Kishan S, Rao MS, Ramachandran P, Devasia T, Samanth J. Prevalence and Patterns of Sleep-Disordered Breathing in Indian Heart Failure Population. Pulm Med. 2021;2021:9978906. [DOI] [PubMed] [PMC]
17. Paulino A, Damy T, Margarit L, Stoïca M, Deswarte G, Khouri L, et al. Prevalence of sleep-disordered breathing in a 316-patient French cohort of stable congestive heart failure: Prévalence des syndromes d'apnées du sommeil dans une cohorte française de 316 patients insuffisant cardiaques. Arch Cardiovasc Dis. 2009;102:169–75. [DOI] [PubMed]
18. Arzt M, Woehrle H, Oldenburg O, Graml A, Suling A, Erdmann E, et al.; SchlaHF Investigators. Prevalence and Predictors of Sleep-Disordered Breathing in Patients With Stable Chronic Heart Failure: The SchlaHF Registry. JACC Heart Fail. 2016;4:116–25. [DOI] [PubMed]
19. Salama S, Omar A, Ahmed Y, Abd El Sabour M, Seddik MI, Magdy D. Predictor factors of sleep-disordered breathing in heart failure. Egypt J Bronchol. 2017;11:36–43. [DOI]
20. Herrscher TE, Akre H, Øverland B, Sandvik L, Westheim AS. High Prevalence of Sleep Apnea in Heart Failure Outpatients: Even in Patients With Preserved Systolic Function. J Card Fail. 2011;17:420–5. [DOI] [PubMed]
21. Borrelli C, Gentile F, Sciarrone P, Mirizzi G, Vergaro G, Ghionzoli N, et al. Central and Obstructive Apneas in Heart Failure With Reduced, Mid-Range and Preserved Ejection Fraction. Front Cardiovasc Med. 2019;6:125. [DOI] [PubMed] [PMC]
22. Oldenburg O, Lamp B, Faber L, Teschler H, Horstkotte D, Töpfer V. Sleep-disordered breathing in patients with symptomatic heart failure: a contemporary study of prevalence in and characteristics of 700 patients. Eur J Heart Fail. 2007;9:251–7. [DOI] [PubMed]
23. Tadic M, Cuspidi C. Obstructive Sleep Apnea and Right Ventricular Remodeling: Do We Have All the Answers? J Clin Med. 2023;12:2421. [DOI] [PubMed] [PMC]
24. Romero-Corral A, Somers VK, Pellikka PA, Olson EJ, Bailey KR, Korinek J, et al. Decreased Right and Left Ventricular Myocardial Performance in Obstructive Sleep Apnea. Chest. 2007;132:1863–70. [DOI] [PubMed]
25. Dursunoglu N, Dursunoglu D, Ozkurt S, Gür S, Ozalp G, Evyapan F. Effects of CPAP on right ventricular myocardial performance index in obstructive sleep apnea patients without hypertension. Respir Res. 2006;7:22. [DOI] [PubMed] [PMC]
26. Akyol S, Cortuk M, Baykan AO, Kiraz K, Borekci A, Seker T, et al. Biventricular Myocardial Performance Is Impaired in Proportion to Severity of Obstructive Sleep Apnea. Tex Heart Inst J. 2016;43:119–25. [DOI] [PubMed] [PMC]
27. Altekin RE, Karakas MS, Yanikoglu A, Ozel D, Ozbudak O, Demir I, et al. Determination of right ventricular dysfunction using the speckle tracking echocardiography method in patients with obstructive sleep apnea. Cardiol J. 2012;19:130–9. [PubMed]
28. Shivalkar B, Van de Heyning C, Kerremans M, Rinkevich D, Verbraecken J, De Backer W, et al. Obstructive Sleep Apnea Syndrome: More Insights on Structural and Functional Cardiac Alterations, and the Effects of Treatment With Continuous Positive Airway Pressure. J Am Coll Cardiol. 2006;47:1433–9. [DOI] [PubMed]