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Review Article

Phrenic nerve stimulation as a novel therapeutic approach for heart failure with central sleep apnea: a systematic review

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Abstract
Heart failure (HF) is a chronic condition associated with significant morbidity and mortality. Phrenic nerve stimulation (PNS) is an innovative therapeutic approach targeting HF patients with central sleep apnea (CSA), a condition linked to worsened cardiac outcomes. This systematic review evaluated the efficacy and safety of PNS, focusing on its impact on clinical outcomes such as the apnea-hypopnea index (AHI), central apnea index (CAI), left ventricular ejection fraction (LVEF), and sleep quality. A comprehensive literature search of studies published between 2014 and 2023 identified five relevant studies, following PRISMA guidelines and utilizing the Newcastle-Ottawa Scale for quality assessment. Results from current studies consistently demonstrated that PNS significantly reduces CSA severity, improves cardiac function, and enhances sleep quality, with minimal adverse events and high patient satisfaction. While these findings highlight PNS as a promising additional treatment for HF patients with CSA especially for patients who do not improve despite optimal guideline-directed medical therapy (GDMT), further large-scale randomized trials are needed to confirm its long-term efficacy and safety.

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Introduction

Heart failure (HF) is a complex medical condition caused by any structural or functional issue that impairs the heart's ability to fill with or pump blood.[1] HF remains a significant clinical challenge, affecting millions worldwide and leading to significant morbidity, frequent hospitalizations, and elevated mortality rates. Central sleep apnea (CSA) is a sleep disorder characterized by the brain's failure to send appropriate signals to the muscles controlling breathing, leading to pauses or reductions in respiratory effort during sleep. Unlike obstructive sleep apnea (OSA), which results from physical airway blockages, CSA is associated with various medical conditions, particularly heart failure.[2]

The prevalence of CSA in HF ranges from 25% to 40% and is linked to increased mortality and poor prognosis.[3] Recurrent apneic events in CSA lead to disrupted sleep, chronic fatigue, and decreased daytime functioning, contributing to a diminished quality of life for these individuals.[4] Furthermore, the occurrence of CSA in heart failure patients is linked to increased hospitalizations, elevated healthcare costs, and higher mortality rates.[5]

The recurrent episodes of apnea and hypopnea in CSA lead to intermittent hypoxia and hypercapnia, triggering a cascade of pathophysiological responses. These responses include sympathetic nervous system activation, inflammation, and oxidative stress, which can further impair cardiac function and exacerbate heart failure.[6] Various treatment modalities exist for CSA in HF, such as phrenic nerve stimulation, which has shown promise in improving CSA.[4] Adaptive servo-ventilation has been studied for its effects on CSA in HF patients, showing potential benefits in improving sleep structure.[7] This method involves the electrical stimulation of the phrenic nerve to activate the diaphragm, promoting regular diaphragmatic contractions and mitigating apneic events during sleep.[8]

The remedē System, approved by the FDA, uses transvenous phrenic nerve stimulation (PNS) to treat moderate to severe CSA in adults.[9] PNS stabilizes respiratory patterns, reducing the frequency and severity of CSA episodes, which improves oxygenation and decreases nocturnal hypoxia—crucial for heart failure patients.[10] Additionally, PNS improves cardiac function by controlling breathing patterns and reducing sympathetic activation and oxidative stress, thus alleviating cardiac burdens associated with HF.[11] Finally, PNS enhances quality of life by improving sleep quality and reducing daytime symptoms, leading to a better overall quality of life for heart failure patients with CSA.[12]

This systematic review aims to comprehensively evaluate the efficacy and safety of phrenic nerve stimulation in heart failure patients, particularly those suffering from CSA, by analyzing data from clinical studies and providing insights into its potential role as a therapeutic modality.

Methods

A comprehensive search was conducted across multiple databases to find related studies about Phrenic Nerve Stimulation as a novel therapy in heart failure patients. We used the Preferred Reporting Items for Systematic Reviews and Meta-Analysis (PRISMA) Guidelines to report our findings.[13] The research protocol was registered at PROSPERO (ID: CRD42024604614).

Searching Strategy

A literature search was conducted on 4 online databases (Pubmed, Web of Science, Science Direct, and ProQuest) on June 22, 2024 using the keywords "(phrenic nerve stimulation) AND (heart failure)". All retrieved articles were imported into Rayyan for duplicate removal. The titles and abstracts of the remaining articles were screened independently by the authors to exclude irrelevant studies. Discrepancies were resolved through discussion. Full texts of potentially relevant articles were then reviewed based on pre-established eligibility criteria.

Eligibility Criteria

Specific inclusion and exclusion criteria were established to ensure a comprehensive and relevant analysis. The inclusion criteria encompassed studies involving adult patients (aged 18 years and above) diagnosed with heart failure who also experienced central sleep apnea. Only studies that utilized phrenic nerve stimulation as an intervention and reported its efficacy and safety in treating heart failure were considered. Eligible studies included randomized controlled trials, non-randomized trials, and observational studies. Furthermore, only articles published in English were included to maintain consistency in data interpretation. Conversely, the exclusion criteria aimed to filter out irrelevant or non-applicable studies. This included studies that did not involve heart failure patients or used interventions other than phrenic nerve stimulation. Non-human studies were excluded to focus on clinically relevant human data. Additionally, reviews, case reports, opinion pieces, and articles not published in English were excluded to maintain a high standard of evidence and ensure clarity in data analysis.

Data Extraction

Data were extracted independently by three authors using a standardized extraction form. The extracted data included authors, publication year, study location, study design, sample size, patient characteristics, intervention details, primary and secondary outcomes, and adverse events. The data were compiled into an online spreadsheet and cross-verified by other authors to ensure accuracy. Disagreements were resolved through consensus.

Quality Assessment

The risk of bias in the included studies was assessed using the Newcastle-Ottawa Scale (NOS). The assessment was independently performed by three authors, with disagreements resolved by discussion. The risk of bias was categorized as high, medium, or low based on the number of stars attributed to each study.

Table 1. Quality assessment of selected studies based on the Newcastle-Ottawa scale (NOS).

Author, Year	Selection	Comparability	Exposure	Overall Grade
Costanzo, *et. al.*, 2018	****	**	***	9
Scwarts, *et. al.*, 2021	***	*	**	6
Hill, *et. al.*, 2023	****	*	**	7
Prtratz, *et. al.*, 2021	**	**	***	7
Zhang, *et. al.*, 2015	**	*	***	6

Literature Selection

A total of 1227 articles were identified after initial literature search performed on 4 databases [104 articles from PubMed/MEDLINE, 181 from Science Direct, 316 from Web of Science, and 625 from ProQuest (Figure 1)]. 303 duplicates were removed before screening. A total of 924 articles were identified through screening of titles and abstracts from initial results. Full-text review of all the articles was then completed. All articles were published in 2014 - 2023. Of the 5 articles included, a total of 203 patients underwent device implantation.

Results

The following baseline and study characteristics were collected for each selected article: first author, publication year, nation, and study patient characteristics (Table 2). Efficacy outcome measures included the apnea-hypopnea index (AHI), central apnea index (CAI), percent of sleep with O_2 saturation <90% (T90), sleep efficiency, and Epworth Sleepiness Scale (ESS) (Table 3).

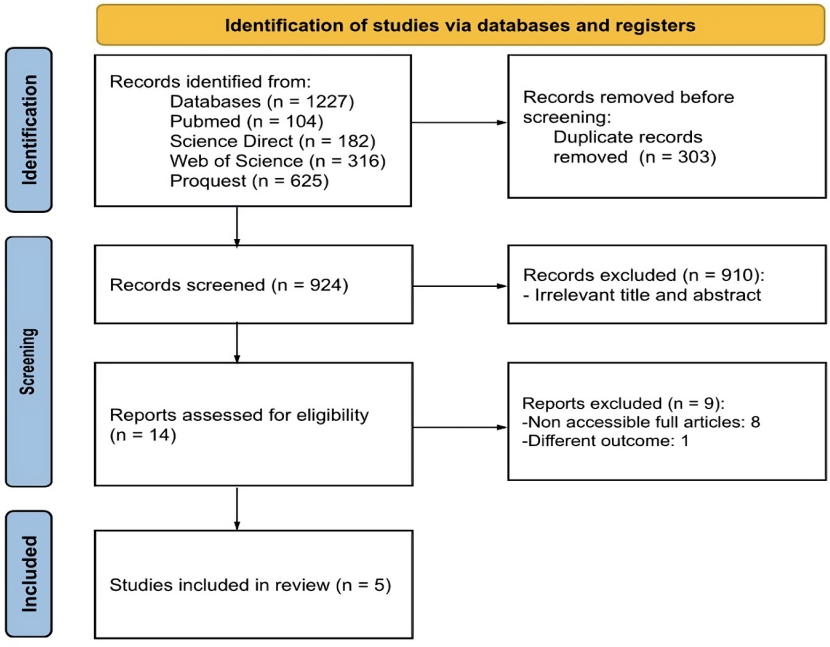

Figure 1. PRISMA flowchart of literature search.

Table 2. Characteristics of selected studies (n=5) and subjects.

Author, Year	Country	Study Design	Sample Size	Age (Year)	Male (%)	BMI (kg/m^2)	NYHA class (%)	LVEF (%)	Type of HF	AHI (events/ h)	CAI (events/ h)	Follow up (mo)
Costanzo, *et. al.*, 2018	USA	Prospective, RCT, MC	96	67±12	87	30.7	I: 19 II: 43 III: 39	34.5	HFrFF	47.1	26.2	6
Schwarts, *et. al*, 2021	Not specified	RCT	151	67.1	89	30.6	NA	40.71	HFmrFF	44.7	26.2	6 & 12
Hill*, et. a*l, 2023	USA Germany and Poland	RCT	75	67±8	93	31	I: 20 II: 48 III: 32	34.6	HFrFF	45.6	24.3	12
Potratz*, et. al.,* 2021	Germany	Prospective	24	67.1± 11.2	92	34.6	II: 62 III: 38	42.4	HFmrFF	38.1	18	6
Zhang, *et. al*., 2015	China	Prospective, SC	8	51.5± 4	75	24.6	III: 62.5 IV: 37.5	37.3	HFrFF	31.2	29.4	6

Values are presented as number or mean; RCT: Randomized Controlled Trial; MC: Multicenter; SC: Single Center; BMI: Body Mass Index; NYHA: New York Heart Association; LVEF: Left Ventricular Ejection Fraction; AHI: Apnea Hypopnea Index; CAI: Central Apnoea Index.

Table 2 shows that CSA in heart failure patients often occurs in elderly men who are obese (BMI ≥ 30kg/m^2) and especially those who have several comorbidities such as cardiovascular disease and metabolic disease.[9-10,13-14] The majority of patients are heart failure patients with Left Ventricular Ejection Fraction (LVEF) ≤ 45.[9-10,14-16] 3 out of 5 studies had a majority of patients with EF ≤ 40, which is included in the HFrEF (heart failure with reduced ejection fraction) category[9,14-15,] while the other 2 studies used samples with a mean LVEF of 40.71 and 42.4, respectively.[10,16] Most of them are included in NYHA class II-III.[9-10,15-16]

Table 3. Results and conclusion of selected studies (n=5).

Author, Year	Medication status	Comorbidities	Intervention	Adverse Effect	Outcomes	Conclusion
Costanzo, et. al., 2018	Patients had to be medically stable for 30 days on GDMT prior to baseline assessments and have a qualifying polysomnogram. Medications used such as: ACEi or ARB, statins, beta-blocker, antiplatelet, MRA, loop diuretic, thiazide, digoxin and CCB.	history of AF, HT, CAD, DM, stroke, renal impairment	Unilateral TPNS vs no TPNS	33% patients reported non-serious therapy-related discomfort through 12 months	Reduces CSA severity, improvement LVEF and AHI, fewer arousals, less hypoxaemia, and improvement in REM sleep and QoL	TPNS reduces CSA severity in patients with HF
Schwarts, et. al, 2021	Patients included are medically stable for 30 days prior to all baseline testing including not using any PAP therapy. Medications not specified.	AF, CAD, history of jaw/ neck surgery	TPNS; active and deferred 6-mo therapy	One-third patients complaint discomfort	Reduces AHI to <20/h and CAI to ≤2/h, reductions in daytime sleepiness and fatigue	TPNS improved AHI, CAI, QoL of the patients with CSA

Hill, et. al, 2023	Patients who were medically stable on GDMT for >30 days. Medications not specified.	DM, AF, concomitant cardiac device, HT, CAD	TPNS; active and deferred 6-mo therapy	Not reported	Reductions in daytime sleepiness and fatigue, resolution of insomnia/fragmented sleep and snoring	TPNS improved QOL, sleep quality, and reduced daytime sleepiness
Potratz, et. al., 2021	Patients included already received treatment with optimal guideline-based HF medication and cardiac devices for at least 6 months. Medications not specified.	Not reported.	TPNS	No serious adverse events	Significant decrease in AHI and CAI, significant improvement in 6MWD and hypoxemic burden	TPNS can reduce hypoxemic burden and improve physical capacity
Zhang,et. al., 2015	All the patients were on standard therapy for HF based on their ejection fraction and medically stable prior to the procedure. Medications used such as: digoxin, diuretics, beta-blocker, nitroglycerin and ACEi.	Cardiomyopathy, AF	TPNS	1 out of 8 patients have dislodgement	Significant decrease in AHI and CAI, improvement in 6MWD, and statistical elevation of LVEF	TPNS is safe and feasible for HF patients with CSA

TPNS: Transvenous Phrenic Nerve Stimulation; AF: Atrial Fibrillation; HT: Hypertension; CAD: Coronary Artery Disease; DM: Diabetes Mellitus; CSA: Central Sleep Apnoea; GDMT: Guideline-Directed Medical Therapy; ACEi: ACE Inhibitors; ARB: Angiotensin Receptor Blocker; MRA: Mineralocorticoid Receptor Antagonist; CCB: Calcium Channel Blocker; AHI: Apnea Hypopnea Index; CAI: Central Apnoea Index; LVEF: Left Ventricular Ejection Fraction; 6MWD: Six-Minute Walk Distance; REM: Rapid Eye Movement; QoL: Quality of Life; HF: Heart Failure.

As presented in Table 3, patients included in these studies were already on GDMT for HF and medically stable prior to TPNS intervention.[9,10,14,16] All the studies maintained a standard approach where PNS was implemented as an adjunctive therapy rather than a replacement for GDMT. The study on PNS for treating CSA in HF patients demonstrates that PNS significantly reduces the AHI and CAI.[9-10,14,16] PNS also improves sleep quality and oxygenation, thereby improving the global well-being of patients.[9,14-15] There is also improvement in physical performance that is measured using 6MWD.[10,16] During the 6 and 12 months follow-up, some studies report one-third of patients complaining of discomfort after PNS implantation.[9,15] In other studies, there is no serious adverse effect or mortality has been mentioned, but one of them had dislodgement and the lead was subsequently repositioned after follow-up.[10,15-16]

Discussion

Despite advancements in HF treatments, many patients remain symptomatic and experience compromised quality of life, leading to high readmission rates.[17] Sleep-disordered breathing, particularly CSA, significantly contributes to the high morbidity and mortality in HF patients.[9] CSA in these patients is driven by hyperventilation, circulatory delay, and cerebrovascular reactivity. Hyperventilation is triggered by increased chemosensitivity and stimulation of lung receptors due to pulmonary interstitial congestion.[18] Fluid movement during sleep exacerbates this congestion, causing chronic hyperventilation and reduced $PaCO_2$ levels, which leads to periodic breathing cessation or known as central apnea.[19] Additionally, underlying cardiac disease activates peripheral chemoreceptors, triggering an exaggerated response to the decreased CO_2 levels, resulting in apnea. This apnea increases CO_2 levels, leading to hyperventilation and creating a cyclical pattern known as Cheyne-Stokes respiration. CSA in HF often manifests as Cheyne-Stokes Respiration (CSR), a cyclical pattern of waxing and waning breathing. This is driven by the delayed circulation time between the lungs and the brain, leading to a lag in the

feedback mechanism that regulates breathing. When CO_2 levels drop too low, it dampens the brain's drive to breathe, causing apnea. The subsequent rise in CO_2 from apnea stimulates hyperventilation, perpetuating the cycle.[18] Recognizing CSA in HF patients is crucial for providing intensive therapy to improve prognosis and quality of life.

Treating CSA in HF patients involves various strategies aimed at enhancing both cardiac function and respiratory stability. The primary approach typically involves optimizing HF medications. Additionally, device-based therapies such as cardiac resynchronization therapy (CRT) have been shown to improve CSA by boosting cardiac efficiency and reducing episodes of sleep-disordered breathing.[9] Beyond these methods, Continuous Positive Airway Pressure (CPAP) and PNS are two other treatment options, each with its unique benefits and considerations.[20-21]

CPAP is a widely used therapy for CSA, primarily effective in patients with both obstructive and central sleep apnea. It works by providing a constant stream of air through a mask, keeping the airways open and preventing apnea episodes. Studies have shown that CPAP can improve sleep quality, reduce daytime sleepiness, and enhance overall cardiovascular outcomes in HF patients. However, adherence to CPAP therapy can be a challenge due to discomfort and the cumbersome nature of the equipment.[18,20]

PNS involves implanting a device that stimulates the phrenic nerve, which controls the diaphragm, thereby helping to regulate breathing patterns during sleep. The study on PNS for treating CSA in HF patients shows that PNS significantly reduces the AHI, enhances sleep quality, and improves oxygenation, making it a promising alternative therapy, especially for those who struggle with CPAP adherence. Unlike CPAP, PNS has shown higher tolerance and compliance rates among patients. PNS also have minimal serious adverse events over a 12-month follow-up. The therapy also improves cardiac structure and systolic function, reduces the hypoxemic burden (SaO_2 < 90%), and enhances physical performance, as measured by the 6MWD.[10,16] Significant reductions in central respiratory events, including decreases in AHI and the CAI, were observed, though obstructive apnea index (OAI) and LVEF did not differ significantly. However, the left atrial diameter showed notable improvements. These findings support PNS as an effective and safe option as an additional therapeutic option on top of established GDMT for HF patients with CSA specifically for patients who do not improve with standard treatment or who experience side effects from primary treatments such as CPAP. Further randomized, controlled trials are needed to evaluate its long-term efficacy and safety, especially in relation to other implanted cardiac devices and its broader applicability, including for those with coexisting obstructive apnea.[20-21]

Conclusion

Despite optimal GDMT, CSA remains prevalent across all stages of HF. Phrenic nerve stimulation presents a promising alternative for improving both cardiac and sleep outcomes, particularly in patients who cannot tolerate CPAP. This review highlights that PNS significantly reduces apnea events, enhances sleep quality, improves oxygenation and cardiac structure, and increases physical performance. PNS is well tolerated, with a high success rate and minimal serious side effects. However, further large-scale, randomized controlled trials are needed to establish its long-term efficacy and safety in this population.

List of Abbreviations

6MWD	Six-Minute Walk Distance
AHI	Apnea–Hypopnea Index
AF	Atrial Fibrillation
ARB	Angiotensin Receptor Blocker
BMI	Body Mass Index
CAI	Central Apnea Index
CAD	Coronary Artery Disease
CCB	Calcium Channel Blocker
CPAP	Continuous Positive Airway Pressure
CRT	Cardiac Resynchronization Therapy
CSA	Central Sleep Apnea
DM	Diabetes Mellitus
EF	Ejection Fraction
ESS	Epworth Sleepiness Scale
GDMT	Guideline-Directed Medical Therapy
HF	Heart Failure
HFmrEF	Heart Failure with Mildly Reduced Ejection Fraction
HFrEF	Heart Failure with Reduced Ejection Fraction
LVEF	Left Ventricular Ejection Fraction
NOS	Newcastle–Ottawa Scale
NYHA	New York Heart Association
OSA	Obstructive Sleep Apnea
PNS	Phrenic Nerve Stimulation
PRISMA	Preferred Reporting Items for

	Systematic Reviews and Meta-Analyses
QoL	Quality of Life
RCT	Randomized Controlled Trial
REM	Rapid Eye Movement
TPNS	Transvenous Phrenic Nerve Stimulation

Ethical Clearance
Not applicable.

Publication Approval
All authors have reviewed and approved the final version of the manuscript and consent to its publication in the Indonesian Journal of Cardiology.

Authors Contributions
C.A., R.A.F.K., and A.Z.F.R. contributed to the conception and design of the study, literature search, data extraction, analysis, and interpretation of data. All authors have reviewed and approved the final manuscript and agree to be accountable for all aspects of the work.

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Conflict of Interest
The authors declare no conflicts of interest related to the research, authorship, or publication of this article.

Availability of Data and Materials
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Generative AI and AI-Assisted Technologies in the Writing Process
The authors affirm that artificial intelligence (AI) tools were employed exclusively for language refinement, grammar correction, and formatting. No AI tools were used to generate, analyze, or alter the scientific content, data interpretation, or conclusions of this manuscript.

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