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Sleep-disordered breathing in patients with decompensated heart failure

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Abstract

Sleep-disordered breathing (SDB) has a higher prevalence in patients with heart failure than in the general middle-aged population. Obstructive sleep apnea (OSA), one of the forms of SBD, promotes poorly controlled hypertension, coronary events, and atrial fibrillation events that can lead to acutely decompensated heart failure (ADHF), and evidence suggests that untreated OSA increases mortality in patients with heart failure. Cheyne–Stokes respiration and central sleep apnea (CSA) have long been associated with heart failure and, in many patients, can coexist with OSA. In this article, we propose a systematic approach to diagnose and treat OSA in patients with ADHF based on current evidence.

Keywords

Acutely decompensated heart failure; Obstructive sleep apnea; Central sleep apnea; Sleep-disordered breathing; Cheyne; Stokes respiration

Introduction

An association between severe heart failure and a unique form of breathing irregularity: Cheyne–Stokes respiration (CSR) was described over two centuries ago. However, it has been only in the past three decades that intensive epidemiological and experimental work uncovered an intriguing reciprocal relationship between sleep-disordered breathing (SDB) and cardiovascular disease. Central sleep apnea (CSA), a rare form of SDB in the general population, is highly prevalent in patients with systolic heart failure, where it seems to be a result of heart failure and portends a poor prognosis. In these patients with heart failure, central sleep apnea often manifests in a crescendo decrescendo pattern of ventilation followed by central apnea, described as Cheyne–Stokes respiration (CSA-CSR). Obstructive Sleep Apnea Hypopnea Syndrome (or Obstructive Sleep Apnea-OSA) is a cause of hypertension, left ventricular hypertrophy, and early atherosclerosis. OSA also worsens ischemic heart disease and cardiac arrhythmias, and is an independent risk factor for the development of heart failure [1]. That is, in otherwise healthy individuals, OSA can be a cause of cardiac dysfunction, and in patients with existing cardiovascular disorders or heart failure, OSA may accelerate the

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progression to acutely decompensated heart failure (ADHF) [2–4]. To date, no substantive study of adequate size has systematically evaluated either the prevalence or importance of SDB in the setting of decompensated heart failure. It is likely that the aforementioned cardiovascular consequences of SDB contribute to the decompensated state and poor outcomes of acute heart failure syndromes. Indeed prospective epidemiological data regarding the cardiovascular outcomes of SDB [4], while still limited, show increased incidence of fatal and non-fatal cardiovascular events in patients with SDB.

Sleep disordered breathing: a new component in the management of heart failure

Heart failure is the only cardiovascular disease with increasing incidence and mortality [5]. Approximately 5 million Americans have heart failure, with an annual incidence of 10 per 1000 in individuals over 65 years, an already rising demographic segment. Hospital discharges for heart failure increased 175% between 1979 and 2004. For 2007, the annual cost of heart failure was estimated at $33 billion. Heart failure is the most frequent Medicare diagnosis with most of its cost related to hospitalizations [5,6]. Treatment options for decompensated heart failure are limited [7,8]. Identification and treatment of highly prevalent co-morbidities with known detrimental effects, carries a high potential for positive impact [9].

SDB, broadly categorized into central and obstructive sleep apnea, has far higher prevalence in patients with heart failure than in the general middle-aged population [10–12]. Recent data suggest a higher prevalence of OSA than previously described with an estimated prevalence of 38–53% of patients with stable heart failure [13–16]. This increasing incidence is likely related to the rise in obesity and aging in the general population. OSA promotes poorly controlled hypertension, coronary events, and atrial fibrillation; all are associated with ADHF [17,18]. It is likely, therefore, that OSA would be very prevalent in patients presenting with ADHF. Given the new evidence of increased mortality in patients with heart failure and untreated OSA [19], it is critical to establish a systematic approach to diagnose and treat OSA in patients with ADHF. However, such an approach is not part of the current standard of care [20,21]. In the subsequent portion of this review, we will first discuss the effect of decompensated heart failure on SDB, followed by the role of SDB in worsening existing cardiac dysfunction. Finally, we will discuss treatment of SDB in patients with cardiac dysfunction, with focus on the setting of decompensated heart failure.

The effect of decompensated heart failure on sleep-disordered breathing

As cardiac function deteriorates, several pathophysiological factors develop and contribute to both of respiratory control instability and reduced upper airway patency; subsequently promoting both obstructive and central respiratory events during sleep.

Decompensated heart failure induces respiratory control instability and central sleep apnea

During sleep, arterial carbon dioxide level ($PaCO_2$) is the main stimulus for ventilation; and respiration ceases if $PaCO_2$ falls below a tightly regulated level called the apnea threshold. Patients with heart failure have a pattern of chronic hyperventilation characterized by close proximity between their eupneic level of $PaCO_2$ (steady-state level of ventilation) and their apnea threshold [22]. The mechanism of this chronic hyperventilation in patients with heart failure is thought to be related to pulmonary interstitial congestion [23]. In fact, animal models had increased respiratory control instability and central apnea in response to increased left atrial pressure [24], a typical condition in patients with ADHF. Any slight increase in ventilation during sleep, as occurs with arousal or changes in sleep stages [25–28], will result in a drop in $PaCO_2$ below the apnea threshold precipitating apneic events [29]. Given the inertia in the

respiratory control system, breathing does not resume until an excessive chemical stimulus (hypercapnea) has accumulated producing an "overshoot" of ventilation that is likely to drop $PaCO_2$ again below the apnea threshold and create periodic breathing and CSR (see article by Badr in this supplement). Furthermore, patients with heart failure have reduced cerebral blood flow response to changes in $PaCO_2$, which reduces the ability of the respiratory control center to dampen the overshoot and undershoot in the ventilatory response to carbon dioxide [30]. The mechanism of central apnea in patients with congestive heart failure is discussed in detail in another article in this supplement.

The role of increased filling pressure in the pathogenesis of SDB in patients with heart failure is quite intriguing, and only minimally understood. As discussed above, increased pulmonary vascular congestion is thought to contribute to hyperventilation, and increased respiratory control instability leading to central sleep apnea. Recent studies are also suggesting that cervical venous congestion may equally destabilize the upper airway and potentially produce obstructive events as well [31]. It is known that central sleep apnea may result from upper airway instability [32], and that upper airway obstruction may follow central apnea [33]. Therefore, worsening of heart failure may lead to both increased central and obstructive apneas.

Decompensated heart failure exacerbates obstructive sleep apnea

Obstructive sleep apneas occur when the balance between the upper airway constricting and dilating forces shifts toward the constricting forces. The two main collapsing factors of the upper airway are the intra-luminal negative pressure generated by the diaphragm during inspiration and the extra-luminal pressure from the tissue surrounding the airway [34]. The mechanism of OSA in patients with stable heart failure is probably similar to that in the general population and is strongly related to obesity [12]. Additional influences on the upper airway patency, such as pharyngeal edema or cervical venous congestion may contribute to the genesis of OSA, or worsening of underlying OSA in patients with decompensated heart failure [31]. Furthermore, upper airway collapse is common at the end of central apneas because of central influences on the upper airway [33]. The respiratory control instability in patients with severe heart failure may further destabilize the upper airway producing concomitant obstructive events in predisposed patients [24].

Mixed respiratory sleep disorder and decompensated heart failure

Since CSA is a very common manifestation of respiratory control instability in heart failure, and OSA is highly prevalent in patients with cardiovascular diseases, it is very likely that the two types of apnea, obstructive and central, may coexist in the same patient. Furthermore, respiratory control instability may cause changes in the upper airway tone producing obstruction during central apneas [33]. In fact, increased filling pressures during the course of one night in patients with severe chronic heart failure and OSA may give rise to increased central apneas by the end of the same night [25,26].

Consequences of SDB in decompensated heart failure

In patients with SDB, the recurrence, throughout the night, of apnea or hypopnea followed by a recovery phase induces a cyclic pattern of intermittent hypoxia-reoxygenation, arousal, increased inspiratory effort, sympathetic activation, and surges in blood pressure. These perturbations are strikingly similar in both central and obstructive sleep disorders and are associated with the same cardiovascular consequences.

Intermittent hypoxia and sympathetic activation

During apnea, in both OSA and CSA, hypoxia stimulates the chemoreceptors which mediate an increase in sympathetic activity [27]. This sympathetic activation produces vasoconstriction

and a surge in blood pressure [28], increasing the afterload for a failing heart. With the recurrence of apnea-recovery and the associated hypoxia-reoxygenation, a cyclic pattern of sympathetic activation and blood pressure surges recurs throughout the sleep period. Interestingly, the increase in sympathetic activity and blood pressure persists during the daytime [35]. This memory effect or plasticity in the neurocirculatory response is unique to the stimulus of intermittent hypoxia and explains the causal link between SDB and hypertension [36]. This sympathetic overactivity in patients with heart failure and SDB induces ventricular irritability and consistently resolves with treatment of SDB [37,38].

Increased respiratory effort

The mechanical perturbations are more profound in OSA than they are in CSA, and appear to have more severe implications for patients with heart failure. When an obstructive apnea or hypopnea occurs, the resulting hypoxia stimulates the respiratory centers, which generate repetitive vigorous inspiratory effort attempting to overcome the collapsed airway. Subsequently, the pressure inside the thoracic cavity becomes increasingly negative reaching several folds the normal inspiratory negative pressure and can have serious effects on the heart via several mechanisms. First, the negative intra-thoracic pressure augments the gradient between the intra-ventricular pressure and the intrathoracic pressure resulting in increased left ventricular work and wall stress during systole, thus increasing afterload [39]. Second, while varying effects on central venous pressure during apnea are reported in patients with OSA, an increase in venous return to the right ventricle is likely [40]. This may serve to increase right ventricular preload and may cause a shift in the inter-ventricular septum reducing the left ventricular end diastolic volume. Finally, the negative intrathoracic pressure may affect the balance of forces governing the transudation of fluid into the interstitial space. In fact, pulmonary edema following repeated upper airway obstruction has been reported in humans and reproduced in experimental animals [41].

In central apnea, the large breaths occur during the recovery phase after an arousal terminates the apnea, usually without major accompanying upper airway obstruction and therefore, with less profound negative swings in the intrathoracic pressure. Changes in the venous return and transmural cardiac pressure, however, may still occur. The sympathetic activation associated with CSA (also due to hypoxia) appears to be more important in mediating the cardiovascular effects of CSA than these less profound pressure changes [27].

OSA as a cause of decompensation of heart failure

The deleterious hemodynamic effects of OSA create the conditions to decompensate and perpetuate heart failure. Significant sympathetic activation is a hallmark of OSA [42,43]. It is also widely accepted that increased sympathetic activity is detrimental in patients with existing heart failure [23,44,45]. Sympathetic activation is possibly the mechanism by which OSA exacerbates hypertension [35,46], coronary artery disease (CAD) [47–49], and arrhythmia [50,51] leading to acute heart failure [17].

The causative relation between OSA and hypertension is particularly important in this context [52–55], since OSA can also worsen blood pressure control [56,57]. Patients with OSA have increase risk of developing atrial fibrillation [58,59] and of poor control of atrial fibrillation after cardioversion [59]. Additionally, patients with OSA have increased risk of developing coronary disease [60] and have worse outcomes of their CAD [61,62]. Hypertension and CAD are the most established causes of chronic heart failure [63]. Furthermore, hypertension, CAD, and atrial fibrillation, all etiologically linked to OSA, are also causes of decompensated heart failure [17]. It is therefore not unexpected that OSA is associated with increased mortality in patients with heart failure [19]. It is in this setting of already progressing heart failure, that OSA may further complicate the clinical course producing further decompensation.

In the long-term, OSA may accelerate the progression of coexistent cardiovascular disorders toward heart failure [4]. However, strong evidence now supports a role for OSA in producing cardiac dysfunction, independently of preexisting cardiovascular morbidity. Endothelial dysfunction is strongly associated with OSA [64–66] and may accelerate coronary artery disease [67–71]. Animals exposed to intermittent hypoxia develop left ventricular hypertrophy, probably independent changes in afterload [41,72]. Similarly, patients with OSA appear to develop a unique pattern of left ventricular remodeling [73–75], which improves with Continuous Positive Airway Pressure (CPAP) [76]. Importantly, several studies showed a pattern of asymptomatic cardiac dysfunction in patients with OSA and no apparent cardiovascular disease [75,77]. These left and right ventricular abnormalities were reversible with CPAP. Additionally, epidemiologic evidence shows, that sudden death, cardiac events, and arrhythmia are all strongly associated with OSA [58,59,78].

Figure 1 reviews the reciprocal relationship between severe heart failure and SDB. The various mechanisms by which SDB may contribute to the pathophysiology of acute heart failure syndromes are presented. This scheme is based on known pathways and its contribution to acute heart failure is biologically plausible on multiple levels. In particular, the interrelationship between obstructive and central sleep apnea and the vicious cycle of acute heart failure and SDB should be noted. Thus, while SDB should be diagnosed and treated in chronic heart failure patients, the importance to do so may be even greater in patients with acute heart failure since the perturbations caused by SDB may be immediately critical and potentially life threatening.

Prevalence and presentation of SDB in patients with heart failure

Estimates of the prevalence of SDB in patients with heart failure differ between studies, due to the varying definitions used. Studies in patients with stable heart failure indicate a staggering prevalence that ranges between 50 and 70% [11]. Although, there are no available studies evaluating the prevalence of SDB in patients with decompensated heart failure, this prevalence is thought to be even higher [79] than in patients with stable heart failure. The distribution of OSA versus CSA in patients with heart failure may depend on the population studied [80]. Patients with advanced or decompensated heart failure are generally expected to have predominantly CSA, while stable chronic outpatients may exhibit a predominance of OSA. However, the recent prevalence studies suggest that the occurrence of CSA is declining [37, 81]. On the other hand, all these recent studies consistently demonstrate a very high prevalence of OSA, in 38–53% of patients with stable heart failure [13–16]. This increasing incidence is supported by the increase in obesity [82] in the general population.

Patients with SDB may report no specific symptoms. This, combined with the significant overlap between symptoms of SDB and symptoms of heart failure, creates difficulty in identifying SDB in patients with chronic heart failure based on history alone. For example, symptoms such as fatigue, tiredness, sleepiness, reduced physical activity level, and impaired cognitive function may be due to either SDB or heart failure and do not aid in determining which patients to screen for SDB. Given the severe impact that untreated SDB has on patients with heart failure [19], it is imperative to maintain a very high index of suspicion for this diagnosis. To date, there is no consensus on a cost-effective approach to conduct surveillance of SDB in heart failure patients. Such an approach will, most likely include the use of validated questionnaires and portable screening devices [83].

Risk factors for OSA in the general population include obesity, increased neck circumference, male sex, and post-menopause in women [84]. These risk factors remain the same in patients with heart failure [12]. The typical symptoms of OSA, snoring, excessive daytime sleepiness, or poor sleep quality, also occur in patients with heart failure. However, their sensitivity and specificity for the diagnosis of SDB may be reduced by the heart failure state [85]. Other

significant presentations in this population probably include worsening heat failure, recurrence of atrial fibrillation, ventricular arrhythmia, nocturnal angina, or stroke.

The form of CSA found in the general population, idiopathic central sleep apnea, has no known risk factors. In patients with heart failure, CSA is very common, and several risk factors have been suggested: reduced systolic function, male sex, advanced age, atrial fibrillation, and reduced daytime $PaCO_2$ [12]. Usually, these patients present with fatigue, insomnia, and poor sleep continuity. Table 1 summarizes the clinical features of the SDB syndromes and their risk factors.

Approach to the management of SBD in patients with ADHF

To date, very little is known about the role of detection of SDB in hospitalized patients with ADHF. Moreover, there are no studies addressing the role of treatment of SDB in the inpatient setting in this population. The clinician is increasingly likely to encounter patients with ADHF and one form or the other of SDB on the hospital ward.

Treatment of OSA and the failing heart

Positive airway pressure and OSA in patients with stable heart failure—Nasal continuous positive airway pressure (CPAP) is the standard treatment for OSA in the general population. CPAP acts as an "air splint" for the upper airway, preventing collapse and episodes of apnea and hypopnea. Thus, CPAP improves sleep quality, reduces daytime sleepiness, improves cognitive function, and results in reduction of the daytime and nocturnal blood pressure in patients with OSA and normal heart function [86]. CPAP and other forms of positive airway pressure exert additional effects on the cardiovascular function in patients with heart failure. Within few hours of administration, CPAP reduces myocardial muscle energy consumption without decreasing cardiac contractile efficiency [87] and reduces respiratory and cardiac muscles workload [39]. After few weeks of administration, CPAP improves left ventricular ejection fraction, and left ventricular transmural pressure [88]. CPAP also results in reduction of left ventricular hypertrophy and improvement in cardiac work index [76], along with improvement in pulmonary and right ventricular pressure and morphology. Reduced Sympathetic activation is another important effect of CPAP in patients with heart failure and OSA [89,90]. Thus, CPAP may improve systolic function through the following mechanisms: (1) it increases the intrathoracic pressure with subsequent reduction in the left ventricular transmural pressure and improvement in the ejection fraction. (2) It reduces sympathetic activation by improving hypoxia. (3) It abolishes the nocturnal variations of blood pressure triggered by apneic episodes. These direct cardiac effects occur in conjunction with improvement in sleep quality. However, large epidemiological studies are still needed to confirm the impact of CPAP on mortality in general and in heart failure patients.

Bilevel positive airway pressure (Bilevel PAP), a modality that delivers two different levels of pressure during inspiration and expiration, is also used to treat OSA. There are no convincing data supporting the superiority of bilevel PAP over CPAP in the outpatient setting. In fact, there has been a theoretical concern that the use of bilevel PAP may promote episodes of central apnea during sleep in patients with mixed CSA–OSA, by increasing ventilation in these patients who have close proximity between eupneic CO_2 and the apnea threshold during sleep [91, 92]. Despite this concern, bilevel PAP is generally considered equivalent to CPAP in the treatment of OSA [93]. The immediate and short-term cardiovascular effects of CPAP are far more studied than those of bilevel. While CPAP was shown to improve systolic function in patients with heart failure and OSA [94,95], bilevel PAP has not been evaluated in this setting.

Positive airway pressure and decompensated heart failure—Positive airway pressure, particularly CPAP is an effective treatment of respiratory insufficiency in patients

with severe decompensated heart failure regardless of underlying upper airway obstruction. In this setting, CPAP is a form of non-invasive ventilation and can improve gas exchange, increase left ventricular ejection fraction (LVEF), and reduce left ventricular filling pressure [96,97]. In a small study of patients with ADHF, CPAP improved ejection fraction and mitral regurgitation [98]. Furthermore, the use of positive pressure ventilation reduces the need for intubation in patients with cardiogenic pulmonary edema by improving the oxygenation and cardiac output [99,100]. These effects of CPAP were consistently present and probably regardless of underlying OSA [101]. Physiological studies confirm that short-term application of CPAP in patients with heart failure and OSA, improves general sympathetic activity [102–104], cardiac sympathetic tone [102–104], cardiac vagal tone [105], cardiac afterload [88], and ventricular arrhythmia [38]. Within several weeks of treatment with CPAP, patients with heart failure and OSA may have an improvement in ejection fraction and functional outcome [94, 95]. Therefore, application of CPAP in patients with acute heart failure and underlying OSA is likely to improve cardiac and autonomic function in the immediate term with outcomes measurable within hours to days. Considering that patients with ADHF have 5–20% mortality [17,106] and a staggering readmission rate [107], the effects of CPAP are likely to be very beneficial in the immediate post-discharge period in patients with ADHF. However, the beneficial effects of expedited treatment of OSA in patients with heart failure in the immediate post-discharge phase have not been evaluated in randomized controlled studies.

The acute cardiac effects of bilevel PAP are likely similar to those of CPAP in the setting of ADHF [108,109]. One non-randomized trial in stable outpatients with chronic heart failure and no OSA demonstrated a significant improvement on LVEF and other hemodynamic markers after one hour of administration of bilevel PAP. They attributed the effect to reduction in afterload [110]. Other studies comparing CPAP and bilevel PAP for the treatment of acute heart failure, demonstrated that bilevel PAP was more effective in reducing respiratory load than CPAP [97,111].

In conclusion, it is likely that in the setting of ADHF, either CPAP or bilevel PAP will improve pulmonary edema and reduce the need for intubation and mechanical ventilation. These effects are likely to be even more beneficial in patients with ADHF and concomitant OSA. Nevertheless, intermediate and long-term studies evaluating the effect and safety of in-hospital diagnosis and treatment of OSA in patients with ADHF remain lacking.

Patients with OSA who are already treated with CPAP or bilevel PAP, and develop ADHF, treatment with positive pressure should continue in the hospital setting. There is no evidence that titrating the pressure again in the hospital is beneficial in these patients. In patients with acute heart failure and without the diagnosis of OSA in whom the disorder is highly suspected, a sleep medicine consultation, or empirical CPAP is strongly advised.

Approach to central sleep apnea in decompensated heart failure

Optimization of medical therapy of heart failure improves and can significantly ameliorate CSA [112]. The benefit of treating CSA in patients with chronic heart failure remains debatable [113,114]. In a recent randomized, single blinded, multi-center trial, CPAP improved oxygenation and ejection fraction but failed to improve transplantation-free survival or to decrease the number of hospitalizations [37]. It should be noted that this study applies only to outpatients with chronic heart failure and CSA. In patients with ADHF, respiratory control instability is heightened, subsequently CSA is likely to be more severe than during the stable course of heart failure [112]. In patients with respiratory insufficiency due to ADHF, CPAP is an effective treatment that can avert respiratory failure. Generally, in patients with acutely decompensated heart failure, with or without CSA or OSA, CPAP, or bilevel PAP is probably beneficial.

Adaptive pressure support servo ventilation (ASV) is a modality that delivers positive expiratory airway pressure along with variable pressure support based on the detection of CSA-CSR with an automatic back-up respiratory rate. In a small prospective, randomized, multicenter trial this mode of ventilation significantly improved compliance and left ventricular function after 6 months when compared with CPAP [115]. The use of ASV in the hospitalized setting has not been evaluated yet.

Nocturnal supplemental oxygen may also improves central apnea, eliminating apnea related hypoxia, but it lacks some of the benefits of positive pressure such as improvement in cardiac function or water redistribution in the congestive lung [116–119]. The inability of oxygen to relieve the obstructive events in patients with the mixed form is another disadvantage. Overall, oxygen therapy has not been adequately studied as a treatment for CSA in heart failure, although its administration to patients presenting with acute heart failure syndromes is empirically encouraged. Added dead space breathing, as a respiratory stimulant, is a treatment modality that is insufficiently studied in the clinical setting [120].

Acetazolamide and theophylline have been evaluated for the treatment of CSA; however, safety data regarding their use in patients with heart failure are not adequately reassuring. With the former agent, urinary potassium wasting leading to hypokalemia and increased arrhythmia risk is a real concern. In the case of theophylline, its stimulatory affect on the heart may produce tachycardia and theoretically increase the risk of life threatening arrhythmias. Both should be avoided in the setting of heart failure.

Complications of positive pressure ventilation

Possible adverse reactions to nasal interfaces for CPAP, bilevel PAP, or servo ventilation are very mild and include nasal congestion, upper respiratory tract dryness, epistaxis, skin abrasion, conjunctivitis, claustrophobia, chest discomfort, aerophagia, and rarely pneumothorax. The use of humidifiers helps to prevent nasal dryness and epistaxis and may help to reduce resistance to the airflow. A proper mask fitting is an important step, to avoid leaks that can cause conjunctivitis and to prevent skin abrasion due to excessive pressure from the mask. Attention to the pressure administered and the patient-device interface is critical in determining immediate acceptance of and long-term compliance with the device. These side effects are as important in the inpatient setting as they are in the outpatient setting. The ability to appropriately administer positive airway pressure in the inpatient setting is a complex process that requires training and close follow-up.

Conclusion

The distinction between central and obstructive disorders in patients with heart failure may be difficult due to the complex physiological interaction between the mechanisms leading to either disorder. A mixed pattern of SDB is present in many patients with heart failure, resulting in central, obstructive, and mixed apneas and hypopneas during the course of one night. Patients with acute heart failure syndromes with or without SDB may benefit from treatment with CPAP, which improves pulmonary edema and cardiac output in patients with high preload. Patients with acute heart failure and OSA should certainly be treated with CPAP to eliminate upper airway obstruction. Physiological evidence supports the use of some form of positive airway pressure in patients with acute heart failure and CSA. Randomized controlled trials in the area of management of SDB in patients with ADHF remain conspicuously lacking.

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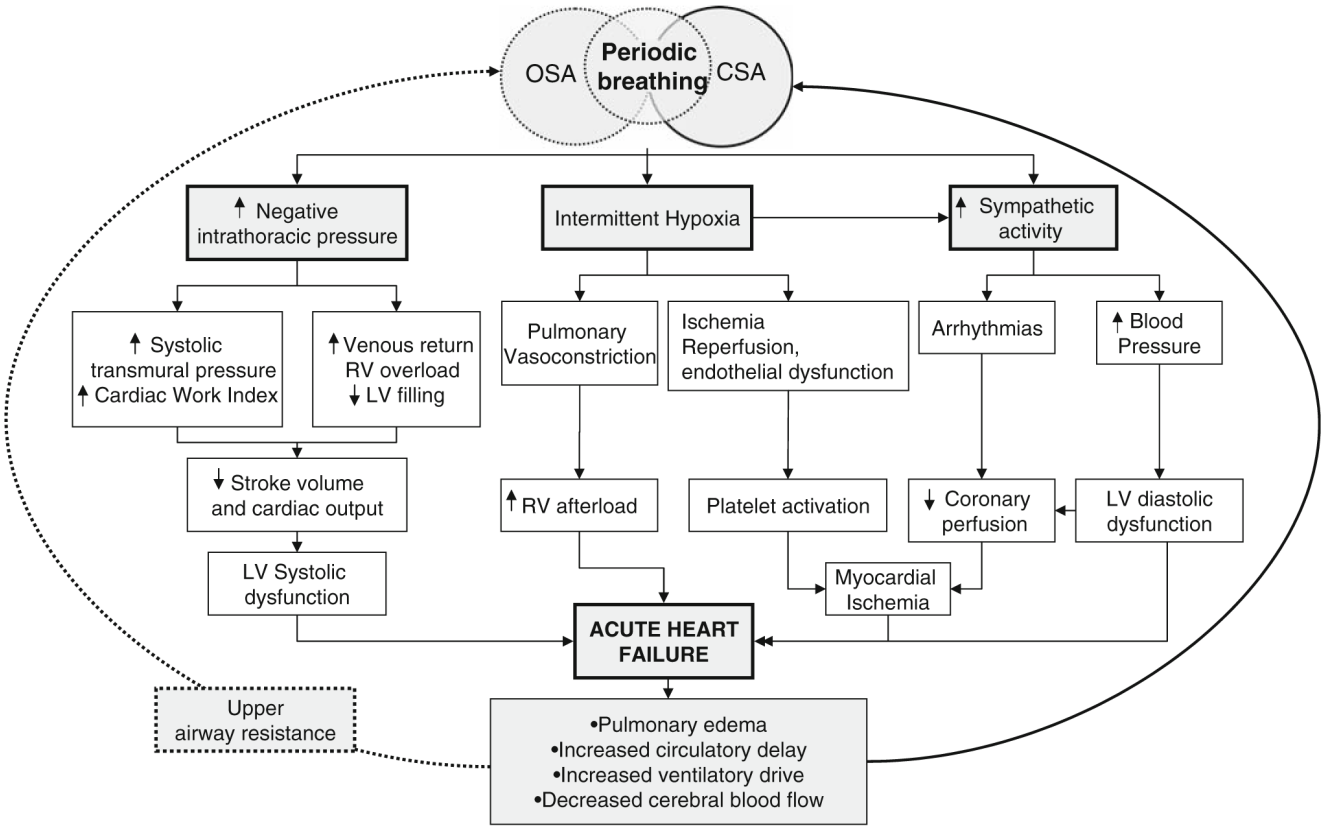

Fig. 1.

The reciprocal relationship between SDB and acute heart failure syndrome. OSA: obstructive sleep apnea; CSA: central sleep apnea; RV: right ventricle; LV: left ventricle; ↑: increased; ↓: decreased

Table 1

Clinical features of SDB syndromes in patients with heart failure

SDB syndrome	Symptoms in the general population	Additional symptoms in cardiac patients	Risk factors in patients with heart failure
Obstructive sleep apnea	Excessive daytime sleepiness	Angina	Obesity
	Snoring	Stroke	Male gender
	Choking or gasping during sleep	Poorly controlled hypertension	Craniofacial abnormalities
	Fatigue	Pulmonary edema	Hypothyroidism
	Impaired concentration		Postmenopausal women
			Family history
			Diastolic dysfunction
Central sleep apnea	Excessive daytime sleepiness	Ventricular tachyarrhythmia	Systolic dysfunction
	Insomnia	Atrial fibrillation	Lower awake PCO_2
		Exacerbation of heart failure	Age greater than 65
		Poor rehabilitation performance or deteriorating functional status	Male sex
			Atrial fibrillation
		Cognitive impairment	Non-obese patients