

## Cheyne-Stokes Respiration, Obstructive Sleep Apnea and Chronic Heart Failure. Is Auto Servo-Ventilation the Best Choice? A Case Report and Literature Review

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### Abstract

**Background:** Sleep-disordered breathing (SDB) describes a group of disorders in which partial or complete cessation of breathing occurs many times throughout the night, resulting in daytime sleepiness or fatigue that interferes with quality of life of patient. Collectively, obstructive sleep apnea (OSA) and central sleep apnea (CSA)/Cheyne-Stokes respiration (CSR) constitute SDB. Evidence suggests that both forms of SDB and often their combination of both are highly prevalent in patients with cardiovascular disease, including chronic heart failure (CHF).

A 70-year-old male (nonsmoking, with normal weight,) visited to our office with symptoms of excessive daytime sleepiness and a history of snoring.

The principle comorbidities were ischemic cardiomyopathy, chronic atrial fibrillation with left ventricular ejection fraction (LVEF) of 45%. He was screened for SDB and subsequently underwent an ambulatory home sleep apnea study (HSAT), which demonstrated a severe OSA with CSR.

He was then subjected to auto servo-ventilation (ASV), which had benefit; treatment with automatic or fixed continuous positive airway Pressure (APAP/CPAP) was not effective.

His quality of life improved (with reduction of daytime sleepiness and fatigue) and his LVEF increased.

This case report, demonstrates the beneficial effects of ASV for a patient with OSA and CSR, improving his symptoms, representative clinical parameters with regard to heart failure and cardiac function.

**Keywords:** *sleep-disordered breathing; obstructive sleep apnea; Cheyne-Stokes respiration; chronic heart failure; adaptive servo ventilation*

### Introduction

Sleep-disordered breathing (SDB) is one of the common complications of cardiovascular diseases, such as chronic heart failure (CHF) [1]. Two different types can coexist in patients with CHF: Obstructive Sleep Apnea (OSA) and Cheyne-Stokes respirations (CSR) [2,3].

OSA is a breathing disorder during sleep that affects 10% of the population [4]. It is characterized by recurring episodes of upper respiratory tract obstruction episodes, during sleep; these episodes can be complete (apnea) or partial (hypopnea) and are frequently accompanied by a decrease in blood oxygen saturation. OSA patients present to the outpatient clinics with complaints of snoring at night, sleepiness in the daytime and witnessed apnea [2,4]. The clinical goal for this condition is the therapy with positive airway pressure (PAP), optimizing the adherence with education, clinical support and behavioral interventions [5].

CSR is a special form of central sleep apnea (CSA), that is seen in roughly one-third of those with CHF. Coexisting CSA and OSA is a growing concern, because most patients who are referred for sleep studies suffer from comorbidities, such as cardiovascular and neurological disorders, which increases the likelihood of both central and obstructive events [6-8]. The goal of treatment for CSR-CSA is adaptive servo-ventilation (ASV), a ventilator support system that is designed specifically to normalize ventilation in these patients [9]. It effectively suppresses CSA/CSR in heart failure (with left ventricular ejection fraction (LVEF > 45%) ; in several studies this treatment improves functional class, cardiac functions, exercise capacity and brain natriuretic peptide (BNP) levels [10]. Also several small studies suggest that ASV is an effective treatment option for patients with treatment-emergent CSA, previously referred to as complex sleep apnea [11-13]. The standard method for diagnosing of the SDB is the home sleep test (polysomnography or cardiorespiratory monitoring) [2].

We present a case study with a recent concomitant OSA and CSR with CHF. The initiation of CPAP failed to resolve of both conditions. The patient was then treated with ASV.

**Case Report**

We report a clinical case of 70-year-old male, with CHF, hypertensive-ischemic cardiomyopathy and chronic fibrillation. He presented fatigue to the least efforts.

He had developed loud snoring, witnessed apneas, excessive daytime sleepiness with an Epworth Sleepiness Score (ESS) of 14/24 and night time awakenings. Current medications included diuretics, ramipril, amlodipine, beta-blockers and losartan, anticoagulant therapy. The treatment was optimized and the patient had good hemodynamic compensation.

The physical examination revealed a body mass index of 27.7 kg/m2, a neck circumference of 42 cm, Mallampati III and a micrognathia with a second class malocclusion. Echocardiography showed LVEF of 43%. The arterial blood gas analysis showed pH: 7.48, PaO2: 101 mmHg; PaCO2: 28.7 mmHg; HCO3 21.7 mmoli/L and SatO2 98%.

To rule out OSA, a Home Sleep Test (HST) was administered on room air, overnight.

Apneas, hypopneas, and apnea-hypopnea index (AHI) were defined according to current criteria[14]. Other parameters that were analyzed were: respiratory disturbance index (RDI), events and number of events of obstructive apneas (OA) and central apnea (CA), number and events of hypopnea (H), mixed (M), oxygen desaturation index (ODI) and average of arterial saturation (SpO2 average%) with time of desaturation (T<90%).

The exam revealed an AHI of 42.3 events/h, with several prolonged episodes of obstructive sleep apnea and central apneas (Table 1 and Figure 1).

	<b>Diagnostic</b>	<b>CPAP therapy</b>	<b>ASV therapy</b>
AHI (Events/h-N° events)	42.3-227	21.3-130	0.4
RDI Events/h	42.3	21.3	0
OA Events/h-N° events	13.6-93	0	0
CA Events/h-N° events	15.8-108	21.3-130	0
H Events/h-N° events	9.3-64	0	0.1
M (mixed) Events/h-N° events	3.8 -26	0	0
SpO2 average (%)	96.3	96.5	97.8
T<90%	0	0	0
ODI Events/h-N° events	35.6-256	19.8	0.7

**Table 1:** Parameters analyzed in Home Sleep Tests.

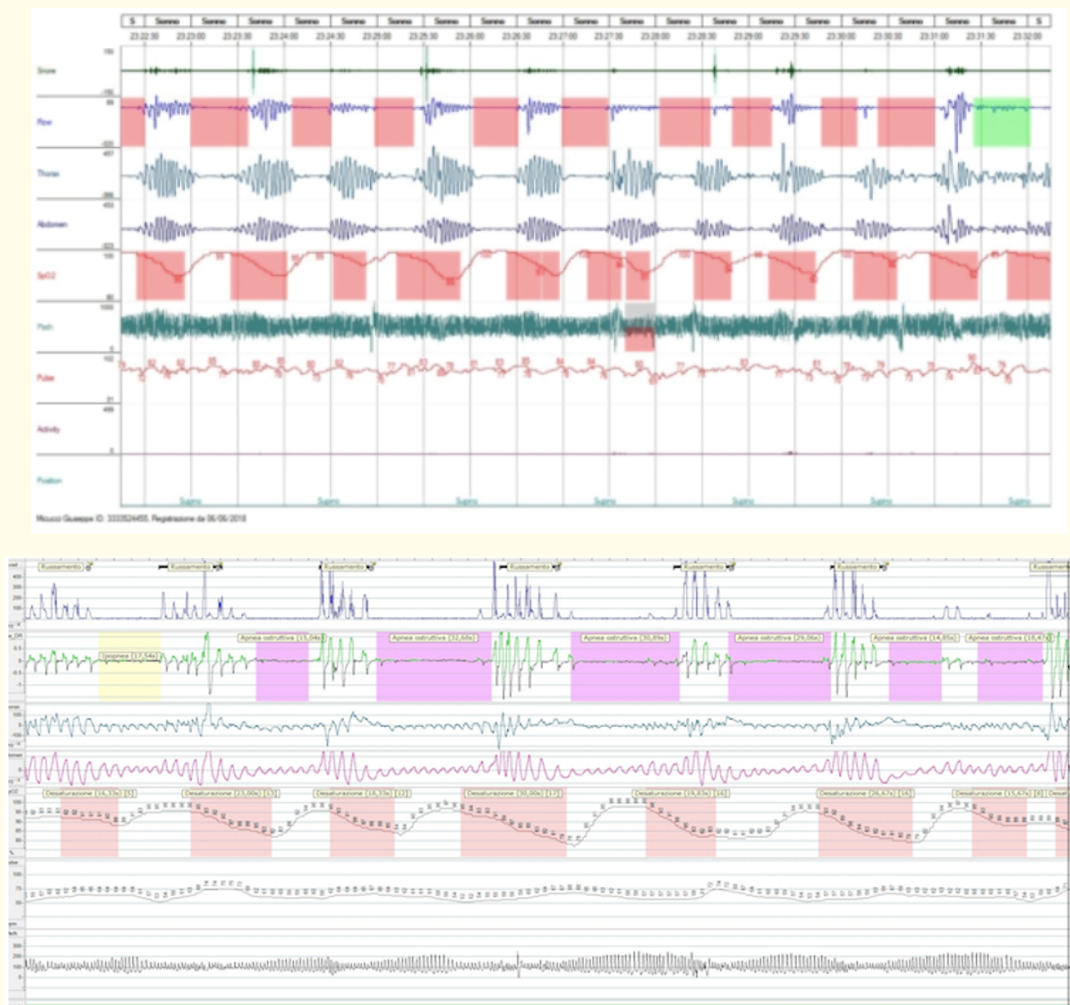


Figure 1: Overnight HSAT at basal time.

The diagnostic study concluded severe OSA with CSR.

Due to the patient’s AHI and our laboratory protocol, we decided perform a split-night polysomnograph and to adapted to CPAP therapy Titration with CPAP was performed demonstrating resolution of obstructive respiratory events but exacerbation of central apneas, (Table 1 and Figure 2) Due to persistent severe CSA, CPAP was replaced with ASV. We using the default settings (EPAP max 9 cmH2O; EPAP min 4.0 cmH2O; PS max 7.0 cmH2O; PS min 2.0 cmH2O) for the patient’s care.

He was administered to ASV therapy, which had a benefit (Table 1). The arterial blood gas analysis at control showed pH: 7,44, PaO2: 94 mmHg; PaCO2: 34 mmHg; HCO3 24.1 mmoli/L; SatO2 98%.

At the 3 months follow-up the patient’s quality of life had improved and his ESS declined to 9/24 and after 6 months from the outset of treatment his LVEF increased to 60%.

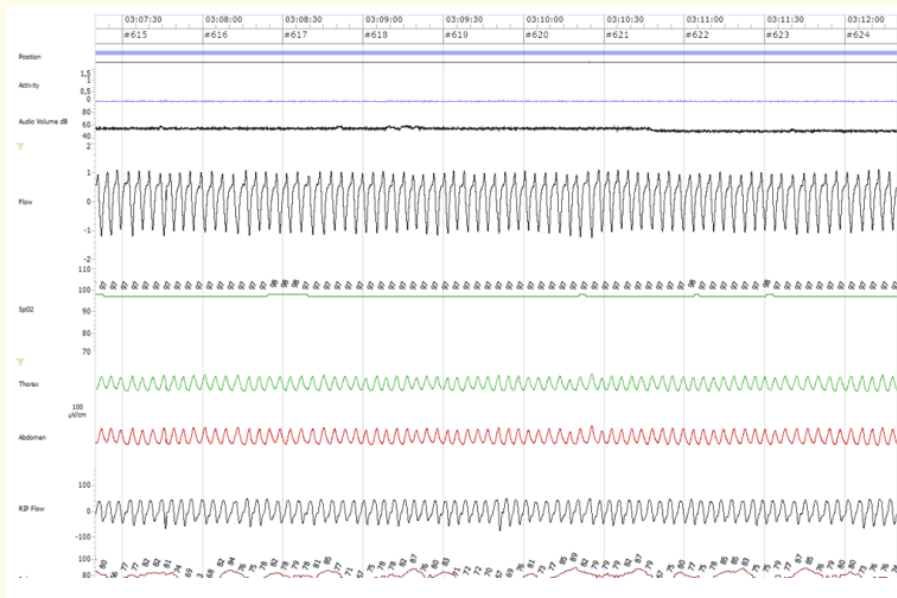


Figure 2: Overnight HSAT during ASV treatment.

## Discussion

OSA, which arises from collapse of the upper airway and CSA, which results from a reduction in central respiratory drive are 2 conditions of SDB [1]. CSA appears to be secondary to HF and occurs when the partial arterial pressure of carbon dioxide falls below the apnea threshold in association with hyperventilation, which occurs chronically in association with pulmonary congestion and increased chemosensitivity in certain CHF patients. Further, delayed transmission in arterial blood gas alterations due to impaired cardiac output causes ventilatory overshoot/undershoot, which typically manifests as CSR [1,2].

The treatment of this combination of aspects (CSA and OSA) remains controversial. Therapy of OSA centered on CPAP has just been established [2], but a large scale clinical trial showed that CPAP does not improve prognosis of such patients with CHF, due to insufficient suppression of SDB [16].

There are a variety of therapeutic approaches for CSR-CSA including night oxygen therapy, non invasive ventilation and optimization of heart failure are described [15]. ASV was designed to stabilize ventilation in patients with CSA and CSR and suppresses central apnea and hypopnea more efficiently than oxygen, CPAP and other types of ventilation (BPAP ST) [17-19]. In observational studies, CPAP or ASV treatment target to suppresses CSA in patient with stable congestive heart failure with reduced ejection fraction with severe CSA is associated with significant improvement in survival [2-21].

The SERVE HF study concluded that patients with CHF and CSR and LVEF <45%, who are treated with ASV have increased mortality, although potential confounding factors (like such as low patient compliance and high cross-over between comparator arms) might have accounted for this risk [22].

In contrast other recent studies reported that ASV improves LVEF and 6-min walk distance, improving cardiac systolic and diastolic function, endothelial function, arterial stiffness and long-term prognosis in CHF patients [23-24].

In our case ASV was efficacious in a patient with coexisting OSA, CSR and CHF, as documented in recent studies: ASV is superior to CPAP in reducing total AHI and obstructive and central apneas [16,25]. After 3 and 6 months of therapy, in the patient's quality of life and left ventricular ejection improved.

Further studies that compare the long-term clinical efficacy of this device against, CPAP are warranted as is an examination of the management of symptoms in OSA-CSR in HF, including objective and subjective outcomes.

### **Acknowledgments**

None.

### **Conflict of Interest**

The authors declare no potential conflicts of interest.

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