



Case Report

# Central Sleep Apnea Syndrome Resolution After Mitraclip®

Mara Volpentesta, Vittoria Monaco, Velia Cassano, Giuseppe Armentaro, Sofia Miceli, Benedetto Caroleo, Raffaele Maio, Maria Perticone, Angela Sciacqua\*

Department of Medical and Surgical Sciences, University Magna Græcia of Catanzaro, Catanzaro, Italy

\*Corresponding author: Angela Sciacqua MD, Department of Medical and Surgical Sciences, Campus Universitario di Germaneto, V.le Europa 88100-Catanzaro, Italy

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## Abstract

Sleep Breathing Disorders (SBD) are alterations of normal breathing occurring during sleep. Their association with Heart Failure (HF) is widely reported in literature, with a prevalence that reaches 75% in reduced ejection fraction Heart Failure (HFrEF). In particular, Central Sleep Apnea syndrome (CSA) prevalence ranges between 25 and 40% in HFrEF and it's related to increased mortality and bad prognosis through to different mechanisms not all understood. In literature, data about prevalence and association of SBD with HF and valvulopathies, frequently associated each other, are lacking nonetheless those about the impact of surgical correction of mitral valve disease on SBD, in particular on CSA, are the most encouraging. Hence, patients with HF and valvulopathies, should be screened for SBD.

**Keywords:** Sleep apnea syndrome; Heart failure; Valvulopathy; MitraClip®

**Abbreviations:** SBD: Sleep Breathing Disorders; HF: Heart Failure; HFrEF: Reduced Ejection Fraction Heart Failure; CSA: Central Sleep Apnea Syndrome; DCM: Dilated Cardiomyopathy; AMI: Acute Myocardial Infarction; PTCA: Percutaneous Transluminal Coronary Angioplasty; COPD: Chronic Obstructive Pulmonary Disease; CPAP: Continuous Positive Airway Pressure; PM-ICD: Pacemaker-Implantable Cardioverter-Defibrillator; EF: Ejection Fraction; PAPS: Indirect Pulmonary Systolic Pressure; PG: Polygraphy Exam; TC90: Time below 90% O<sub>2</sub> Saturation; ODI: Oxygen Desaturation Index; AHI: Number of Apnoic/Hypopnoic Events; NIV: Non Invasive Ventilation

## Introduction

Sleep Breathing Disorders (SBD) include Central Sleep Apnea Syndrome (CSA), Cheyne-Stokes respiration (CSR) and

Obstructive Sleep Apnoea Syndrome (OSAS) and are alterations of normal breathing occurring during sleep. The association between SBD and Heart Failure (HF) is widely reported in literature [1], with a prevalence reaching 75% in Heart Failure with Reduced Ejection Fraction (HFrEF). In particular, CSA prevalence ranges between 25% and 40% in HFrEF and it's related to increased mortality and adverse prognosis through different mechanisms still not well understood [2]. First of all, contrasting apnoic and hypopnoic events generates an inspiratory effort, which modifies intrathoracic pressure and haemodynamics, resulting in an augmented myocardial oxygen request, alterations of cardiac chambers volumes and a reduced cardiac output. Concurrently intermittent hypoxia combined with repeated arousals, causes sympathetic nervous system hyperactivation further worsening haemodynamics. Even without sufficient evidences on prevalence and association of SBD with HF and valvulopathies, data about the impact of surgical correction of mitral valve disease on SBD, in particular on CSA are encouraging [3] and corroborating our suggestion to screen SBD patients with HF and valvulopathies.

## Case Report

We present the case of a 75 years-old man who was admitted to our department complaining of ingravescens dyspnea (NYHA II-III). He had an history of dilated cardiomyopathy (DCM), pacemaker-implantable cardioverter-defibrillator (PM-ICD) implanted after an acute myocardial infarction (AMI) (treated with PTCA and stents on anterior interventricular artery and left coronary), chronic obstructive pulmonary disease (COPD), nocturnal respiratory failure secondary to OSAS on CPAP and O<sub>2</sub> therapy at 2 L/min. Our first approach evaluating the patient involved: chest X-Rays (negative), EGA (normal blood gas values) and ecocardiography. Ecocardiography highlighted a reduction of EF from 42% to 33% Simpson, worsened mitral valve insufficiency (from moderate to severe), left atrial enlargement and increased PAPS (from 40 to 55 mmHg), when compared to the last one (Figures 1A-1C). So we decided to optimize medical treatments for HFrEF and record a new cardiorespiratory polygraphy exam (PG) in basal condition. Nocturnal respiratory failure was confirmed: TC90 60,1%; ODI >3% 88/h; average SpO<sub>2</sub> 89% (Figure 2). Severe CSA with CSR was diagnosed with an number of apnoic/hypopnoic events (AHI) of 87,8/h. According to AASM guidelines, ventilation mode has been changed to BILEVEL PSV (IPAP min-max 13-18 cmH<sub>2</sub>O, EPAP 7 cmH<sub>2</sub>O) keeping O<sub>2</sub> therapy at 2L/min. After one week there

was a reduction in AHI (AHI 12/h) and resolution of nocturnal respiratory failure (TC90 4.5%). Due to functional mechanism of mitral insufficiency, we requested a cardiological consult to evaluate the need of a percutaneous treatment of the underlying mitral insufficiency (MitraClip®). After one month, the patient underwent MitraClip®, with notable reduction of symptoms (NYHA I-II). Three months later even echocardiographic parameters improved (Figures 3A and 3B). One year later, the patient was hospitalized for COPD exacerbation and treated with antibiotic therapy. After the resolution of the acute state, we recorded a nocturnal oximetry on NIV and O<sub>2</sub> therapy at 2L/min, to test the efficacy of past prescribed therapy demonstrating not only the persistence but a further improvement of previously achieved benefits (TC90 from 60.1% to 1.4%, ODI > 3% from 88/h to 18.6/h, SpO<sub>2</sub> average from 89% to 94%). After three months, the patient (as his symptoms further regressed) autonomously chose to lower O<sub>2</sub> therapy to 1,5 L/min and to interrupt NIV. A new PG in O<sub>2</sub> therapy at 1.5 L/min was recorded, showing a reduction in AHI 9.2/h, stable value for TC90, ODI > 3 and average SpO<sub>2</sub> % (Figure 4). EGA in O<sub>2</sub> therapy at 1,5 L/min: pH 7.42, PCO<sub>2</sub> 38 mmHg, PO<sub>2</sub> 81.5 mmHg, SpO<sub>2</sub> 97%. The clinical and instrumental evolution led us to discharge the patient prescribing only O<sub>2</sub> therapy at 1,5 L/min, deeming NIV no longer necessary.

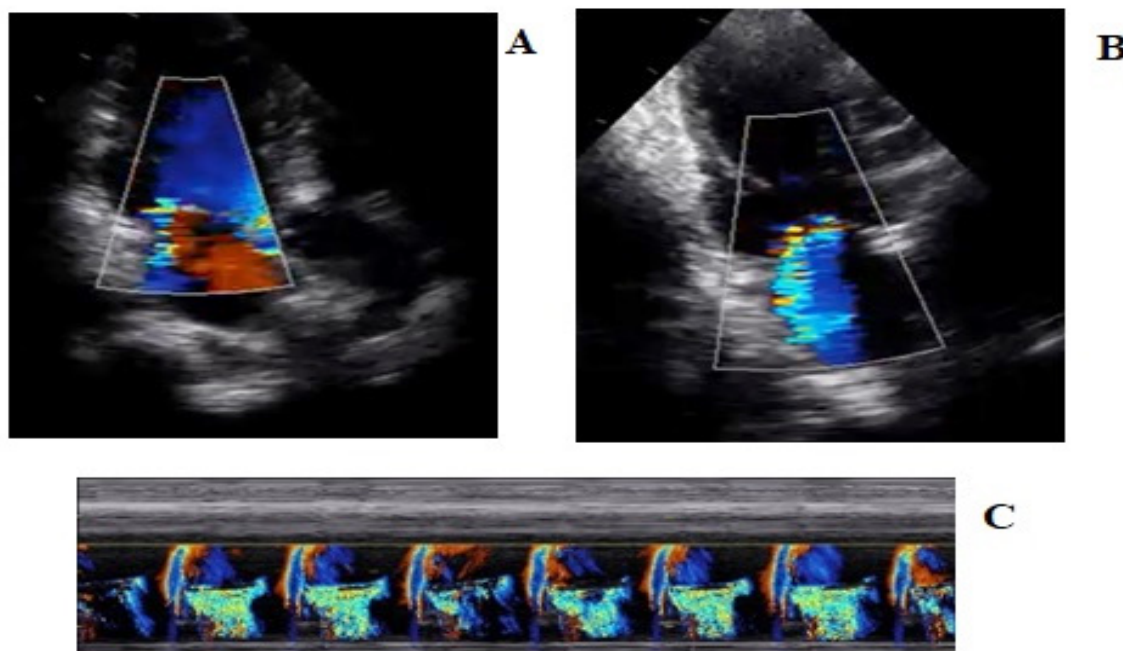
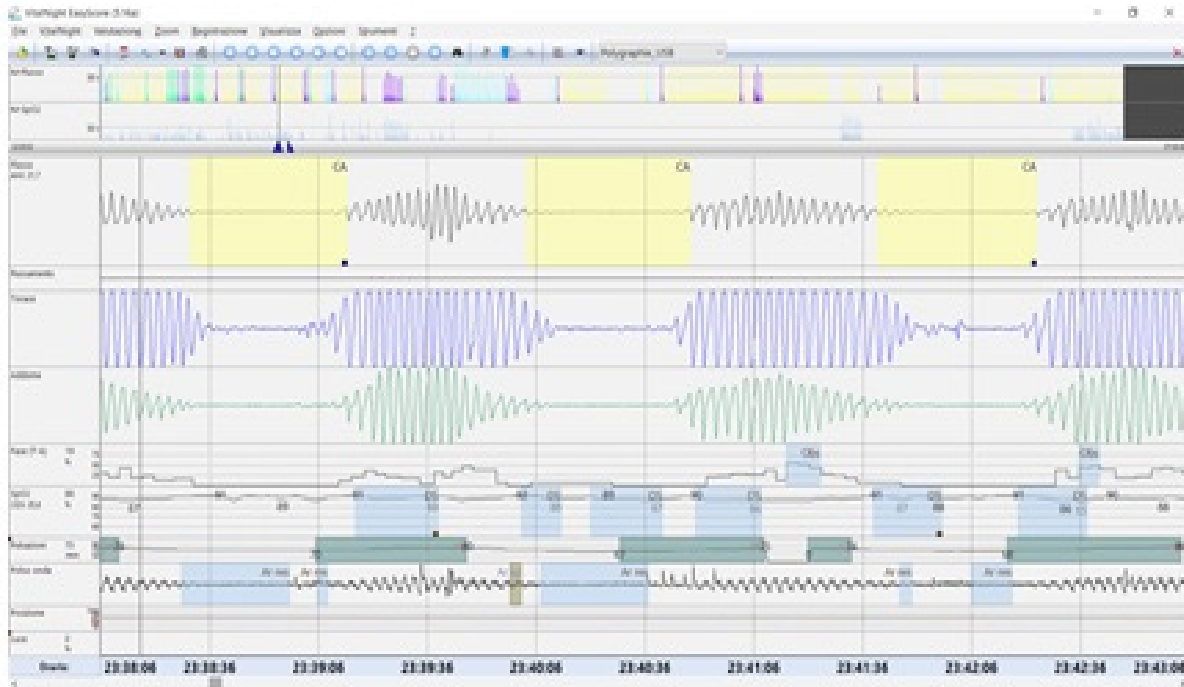
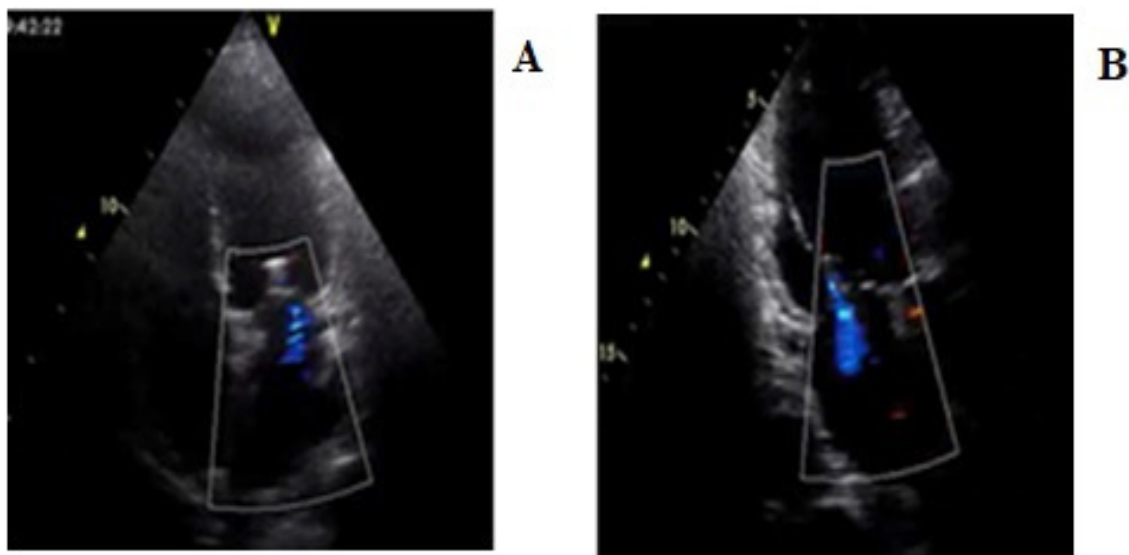


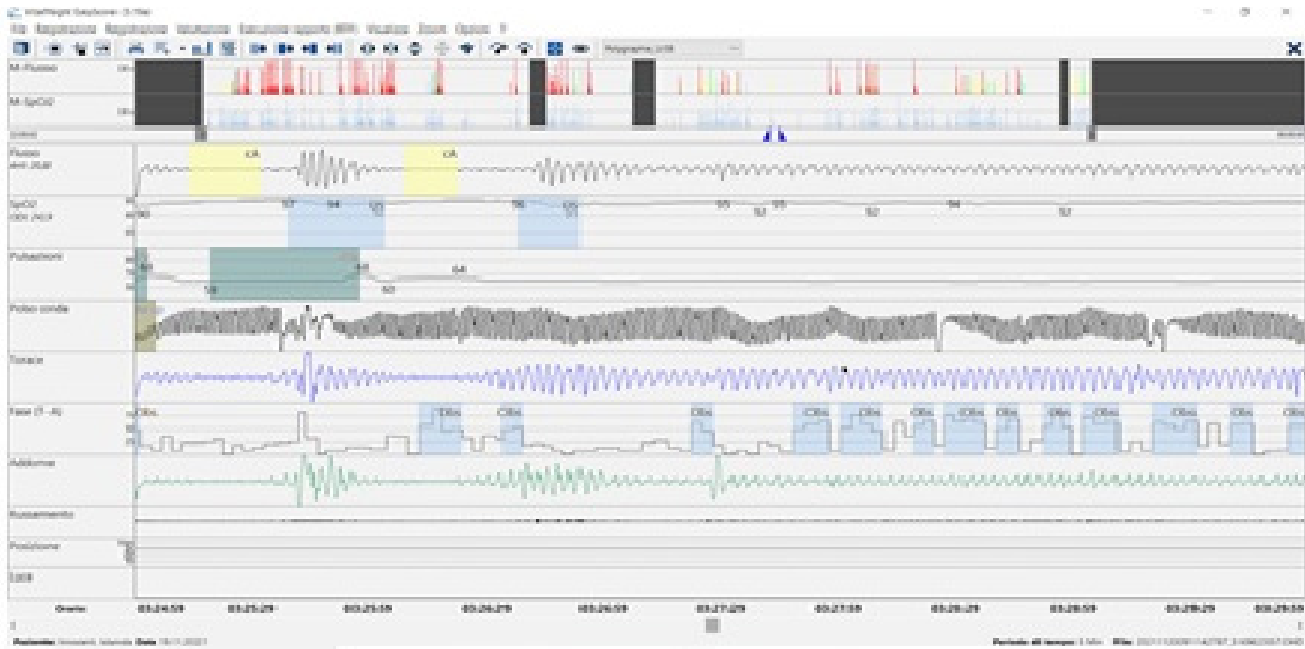
Figure 1A-1C: Echocardiographic images of severe mitral insufficiency before MitraClip®.



**Figure 2:** PG images of CSA before MitraClip®.



**Figures 3A, 3B:** Echocardiographic images after MitraClip®.



**Figure 4:** PG image of CSA resolution after MitraClip®.

## Discussion

CSA can develop through two different mechanisms: an increased sensitivity of the chemoreceptor centers (*controller gain*), showing an hyperventilatory response even for modest increases in nocturnal PaCO<sub>2</sub>, with subsequent hypocapnia and ventilatory drive suppression leading to apnea/hypopnea events; an increased circulatory time (*feedback delay*) in which apnea-induced-hypercapnia stimulates central chemoreceptors at a slower rate, delaying ventilation resumption. This situation can also be exacerbated by overload volume, as in the case of our patient [4]. These two phenomena can occur in HF, stroke, opiates use and brainstem diseases. Finally, in HF and valvulopathies, lung vascular congestion triggers the juxtacapillary receptors (*J receptors*), increasing hyperventilation and worsening apneas. Therefore, to reduce apnoic/hypopnoic episodes, it is crucial to normalize the circulatory time by improving cardiac output and reducing volume overload, through medical and when feasible, percutaneous or surgical therapy. Oxygen therapy, in conditions of normal circulatory time, can contribute to reduce central apneas by decreasing chemoreceptor centers sensitivity.

This clinical case documents the need of a multidisciplinary approach to patients with HF and respiratory diseases and highlights the usefulness of PG in patients with mitral insufficiency eligible for MitraClip®. The presence of CSR and the severity of CSA could suggest the need for early treatment of even moderate and/or paucisymptomatic mitral insufficiency.

## References

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