Impact of Ventricular assist Device on Daily Monitored Sleep Apnea in a Patient with Refractory Congestive Heart Failure

Nicola Vitulano*, Gianluigi Bencardino, Massimo Massetti, and Filippo Crea
Department of Cardiovascular Sciences, Catholic University of the Sacred Heart, Italy

Abstract
The knowledge of the impact of sleep apnea syndrome (SAS) in cardiovascular patients is growing. The prevalence of SAS is high in patients affected by heart failure (HF). This condition is an important comorbidity leading to an increased incidence of major adverse cardiac events. Diagnosis and treatment of this relevant disorder play a pivotal role in the setting of HF, but regarding recent findings physiopathological mechanisms are not still really understood in the complex pathways between the underlying cardiac condition, central and respiratory system. Even if it is a case report, the description of this clinical case offers causes for reflection for further aspects in the complex scenario of HF. New therapeutic approach for advance HF, such as ventricular assist-device (VAD), could have an interesting physiopathological impact on the cardiorespiratory system as focusing on in the following case.

INTRODUCTION
The sleep related physiological changes in the human body, conducing to a cardiovascular state of quiescence, explain the importance of sleep in the human life and the rising interest about sleep disordered breathing (SDB) syndromes. Recent findings and a meta-analysis showed increased cardiovascular risk in patients with SAS [1]. The close link between SAS and cardiovascular disease is now universally recognized. SAS may also worsen preexisting cardiac conditions. The efficacy of new treatment and therapeutic advances have improved the prognosis of cardiovascular diseases, with a relative reduction in hospitalizations and a smaller but significant decrease in mortality in the recent years. At the same time it is important to recognize conditions that might influence clinical course of cardiovascular disease, in order to improve its treatment, above all in patient suffering from HF. SDB is common in patients with chronic heart failure (CHF) and may cause a further deterioration in heart function [2,3]. Despite of advances in pharmacological therapies or in the field of electrical devices (ICD or CRT-D) increasing amount of patients reaches end stage of HF and, awaiting transplantation or as a destination therapy, mechanical assist devices may represent an effective option in these patients [4]. They restore normal haemodynamic parameters improving end-organ function. Few informations about the impact of mechanical assist device treatment on SDB are available and they are often controversial [5,6]. In this article we report, for the first time, the impact of implantation of mechanical VAD on the Apnea-Hypopnea index (AHI), daily monitored by the use of an algorithm (Apnea Scan) available in a recently introduced model of ICD (Incepta, Boston Scientific). In this patient with a previously implanted ICD, AHI analysis marked improvements after VAD implantation compared to the recordings obtained before.

CASE DESCRIPTION
A 62-year-old woman was referred to our department for chronic heart failure not responsive to conventional therapies. She had history of extensive anterior-lateral myocardial infarction, which led to CHF due a severe dilated ischaemic cardiomyopathy, despite of revascularization by PCI with stenting of proximal and medium descending anterior coronary artery as well as circum flex and right coronary artery. At the time of the evaluation, despite optimal medical therapy tolerated (bisoprolol, losartan, furosemide, and spironolactone), echocardiography findings consisted of left ventricular dilatation (end-diastolic diameter: 68mm), severe left ventricular dysfunction (ejection...
the hypothesis of a link between severity of apnea-hypopnea is not completely understood and this clinical case corroborates poor prognosis in patients with advanced heart failure. Moreover, after VAD implantation, of sleep apnea, a well-defined marker of DISCUSSION before VAD implantation (Figure 1).

Recorded by memory of ICD as compared to that one documented trend and an almost complete disappearance apnoic event were improvement of symptoms and overall clinical status. By waiting heart transplantation [9-11].

With regard to the respiratory trend we can notice that there is an disturbance events observed during the programmed sleep period by the number of hours in the sleep period [7,8]. During next clinical assessments, HF scenario was confirmed: the patient reported dyspnea for minimal efforts; at examination heart rate was 90 bpm, respiratory rate was 20; arterial oxygen saturation measured by pulse oximetry while she breathed ambient air was 95%; crackles were heard on auscultation over both low lung fields; third heart sound and an heart murmur consistent with mitral regurgitation were audible at chest auscultation; a chest radiograph showed interstitial edema, an electrocardiogram displayed sinus rhythm.

Interrogation of ICD documented severe SBD (Figure 1). After discussion of the clinical case in a multidisciplinary staff, the patient received VAD (Heart Ware) for treatment of refractory HF (Figure 2). Briefly, Heart ware consists in an inflow cannula designed to allow for pericardial placement, dual motor stators (pump normally runs on both but is capable of running on either stator for added reliability), passive magnetic bearing components housed within impeller and center-post. A percutaneous activation cable links the pump to external controller. The controller, powered by two batteries or electricity socket allows to control the pump function and to monitor the system. Mechanical circulatory support has been developed regarding to shortages in donor organ availability causing decompensation in advanced heart-failure patients or death while awaiting transplantation. Despite of evidence-based guidelines for management of end-stage heart failure are still currently no well-defined and in progress, VAD represents a considerable option in the therapeutic tools of advanced HF for survival to discharge, relief to symptoms and prolonged time to wait heart transplantation [9-11].

After hospital discharge the patient showed a marked improvement of symptoms and overall clinical status. By interrogation of ICD an important improvement of respiratory trend and an almost complete disappearance apnoic event were recorded by memory of ICD as compared to that one documented before VAD implantation (Figure 1).

DISCUSSION

This clinical case demonstrates an impressive improvement, after VAD implantation, of sleep apnea, a well-defined marker of poor prognosis in patients with advanced heart failure. Moreover, the pathogenesis of sleep apnea syndrome in patients with HF is not completely understood and this clinical case corroborates the hypothesis of a link between severity of apnea-hypopnea episodes and neuro-hormonal imbalance typical of advanced heart failure. Approximately 1–2% of the adult population in developed countries suffer by HF, and its prevalence rises up to ≥10% among persons ≥70 years old [12]. In younger age groups, HF is more common among men because its most common cause, coronary heart disease, occurs earlier in men than in women [13,14]. Effective treatment and new therapeutic advances have improved the prognosis of HF, with a relative reduction in hospitalizations and a smaller but significant decrease in mortality in the recent years. At the same time the management of co-morbidities plays a key role in this field for several reasons: the impact of comorbidities on the natural history of HF, the effects of drugs used to treat co-morbidities on the clinical course.
of HF, the interactions between drugs used to treat both HF and associate conditions, reduced patients’ adherence to multiple medical treatments. Sleep apnea is associated with a worse clinical status and is a predictor of poor prognosis among HF patients linking with its in a bidirectional relationship involving mechanical features and central nervous system functions. In HF patients fluid distribution during the night due to a rostral shift from the legs provokes its accumulation in the neck soft tissues. These conditions can cause obstructive apneas through increasing upper airway tissues collapsibility during the sleep by a worsening of the constriction of the pharynx, increasing the loss of tone of the pharyngeal dilator muscle already present during sleeping [15]. In the other way cardiac pump failure provokes increased filling pressures leading to pulmonary congestion, stretching of pulmonary J receptors and an imbalance in gas exchange; these conditions cause the beginning of hyperventilation and a subsequent drop of PaCO2 below the so-called apnea threshold. The consequent hypocapnia causes suppression of the respiratory muscles by the central nervous system, thus causing phases of apnea. During apneic events, due both obstructive and central mechanisms, hypoxia and hypercapnia trigger the respiratory drive with subsequent arousals from sleep in order to terminate the apneic events. Further pathophysiologic mechanisms linking SAS and HF include exaggerated negative intrathoracic pressure that creates an increase in left ventricular transmural pressure, chemoreflex receptors activity and autonomic imbalance with increased sympathetic tone [16]. The above mentioned mechanisms provoke the progression of the underlying cardiovascular disease. Such detrimental triggers on the cardiovascular system can also contribute to the development or worsening of the clinical setting of HF. In additional to several drugs, in elective cases therapy with mechanical assist device improves heart function and haemodynamic parameters: the ventricle overload decreases and there is a reduction of pulmonary filling pressure. The improvement of circulation time prevents fluid stasis above all in the lung and at the same time warrants a better blood oxygenation due to improved gas exchange. An adequate cardiac output reduced nocturnal fluid displacement in the rostral part of body creates the prerequisites to reduce the physiopathological features of sleep apnea. This case demonstrates, by the use of ICD technology, the potential impact of VAD and consequently of a restored cardiac pump function on sleep disordered breathing.

REFERENCES


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