Annals of Case Reports & Reviews

Case Report

doi: 10.39127/2574-5747/ACRR:1000122.
Vitulano N, et al. Annal Cas Rep Rev: ACRR-122.

Has Regular Heart Rhythm A Positive Influence on Nightly Desaturations? The Possible Answer in A Case Report

Nicola Vitulano MD*, Antonio Di Monaco MD, Federico Quadrini MD, Federica Troisi PhD, Massimo Grimaldi PhD

Cardiology and Arrhythmology Unit-Ente Ecclesiastico Ospedale Regionale "F. Miulli"-SP 127 Acquaviva-Santeramo Km. 4,100, 70021 Acquaviva delle Fonti (BA)- Italy

***Corresponding author:** Dr. Nicola Vitulano, P.co dei Cedri, 46-71043 Manfredonia (FG)-Italy. Tel: 00393923975425; Email: nicola.vitulano@gmail.com

Citation: Vitulano N, Di Monaco A, Quadrini F, Troisi F, Grimaldi M (2020) Has Regular Heart Rhythm A Positive Influence on Nightly Desaturations? The Possible Answer in A Case Report. Annal Cas Rep Rev: ACRR-122.

Received Date: 21 May, 2020; **Accepted Date:** 25 May, 2020; **Published Date:** 31 May, 2020

Abstract

In the last decades several findings have documented the strong association between sleep apnea and cardiovascular disease, above all in the arrhythmologic field. The hypoxic trigger due to repetitive nightly apnoic events may be responsible of the onset and the maintenance of the arrhythymias such as atrial fibrillation or atrial flutter. The evidence supports a close link between these two pathological entities. Even if the perspective was always directed from sleep apnea to cardiovascular disease, the physiopathological pathways that link both are not still completely know. So, it is thinkable that also the cardiovascular condition might influence the respiratory trend as the first input regarding the continuous changes happening during alternance between arrhythymias and restore heart rate stability. This case report could be an opportunity of reflection in this way. Future research is expected to clarify the real link between heart and sleep in the different cardiac condition and vice versa.

Keywords: *Sleep apnea syndrome, nightly desaturation, atrial fibrillation, atrial flutter*

Introduction

In the last decades several findings focused on the close relationship between sleep apnea syndrome (SAS) and cardiovascular disease. Apnoic events are characterized by absence of airflow or respiratory flow reduced by more than 90% for at least 10 seconds with a desaturation > 4% from the basal level [1]. Frequent nightly decreases in oxygen levels are harmful triggers for cardiovascular system. Several studies showed a higher incidence of arrhythmias in patient with sleep apnea SAS [2] and at the same time a reduction of arrhythmic burden in the subjects treated by CPAP application, above all in term of recurrence of atrial fibrillation after electrical cardioversion or transcatheter ablation. The pathophysiological pathways considered move from apnoic desaturation triggers towards arrhythmic instability. Instead this clinical case offers the possibility to analyze the link between heart and nightly respiratory trends through an opposite point of view, testing the influence of the heart rhythm regularization on nightly oxygen trends.

Case report

A 58 years-old man was clinically followed for paroxymal atrial fibrillation. He treated hypertension with sartan with successful control and his BMI was 26kg/m2. Despite of optimal therapy (DOAC and double antiarrhythmic drugs) during his clinical history he presented various episodes of atrial fibrillation. Thus, he was underwent to transcatheter ablation procedure. He was in a stable cardiac clinical setting. No symptoms of heart failure were presented. In the clinical follow-up after about one year, the patient reffered some paroxymal episodes of palpitations, more regular than the previous ones, so he was admitted to hospital for futher evaluations. During the clinical and therapeutic assessment, the patient was underwent to Berlin Questionnaire (high risk of sleep apnea), Epworth Sleep Scale ($\geq$ 9 points, measuring daytime sleepiness suggestive of apnea during the night) and then to a nightly cardiorespiratory monitoring because of referred symptoms compatible with sleep disorder breathing. The nightly monitor of heart rate and spO2 value showed normal aspect during sinusal rhythm (figure 1).

Figure 1

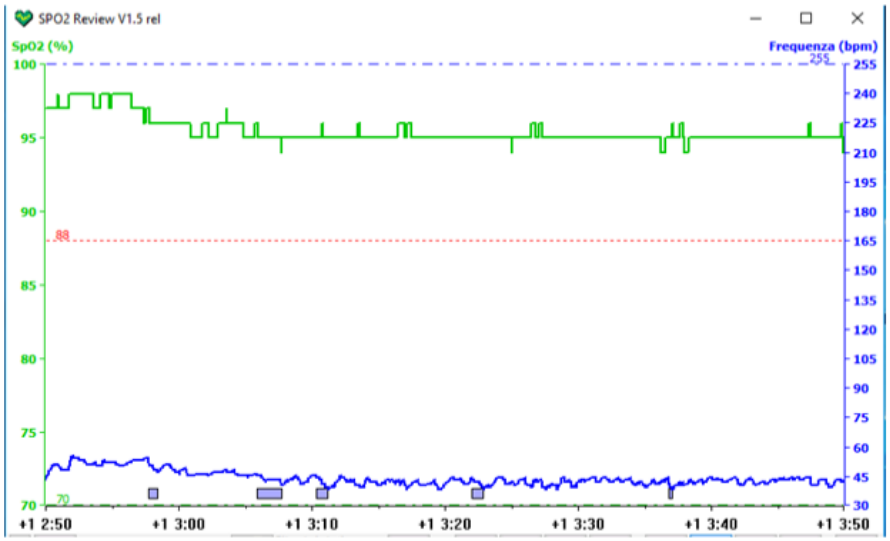

Figure 1: A detail of nightly cardiorespiratory monitor, the upper green line is referred to SPO2 values while the lower blue line shows the heart rate trend. During this part of night, the heart rhythm seems regular with values of heart rate included between 70 and 75 beats/min. At the same time SPO2 values are normal, with minimal oscillations below the physiological range.

Regular heart rate is documented with normal beats/min and at the same time there are no pathological changes in saturation, only minimal physiological fluctuations from the normal threshold. Instead a significative change in the respiratory pattern appeared when atrial flutter succeeded sinusal rhythm (figure 2A): after the onset of atrial flutter with a mean ventricular rate of 120 bpm, the respiratory trend has changed completely; irregular respiratory pattern substituted for previous regular oscillations; frequent drops in saturation from baseline value are documented. Just the heart rate becomes regular the spO2 trend stabilized without significant fluctuations (figure 2B); while other times during the night just atrial flutter (documented also with ecg telemetry) recovers it is possible to highlight the oxygen saturation drops from the normal baseline, with features of accentuated decrease of SO2 values and with recurring episodes moreless cyclical during the night, typical of subjects affected by SAS (with a prevalence obstructive component).

Figure 2

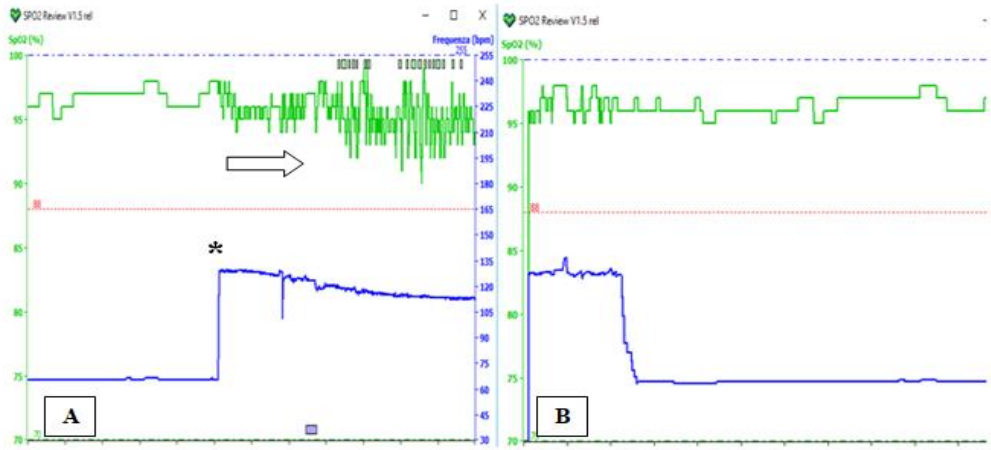

Figure 2: In this picture a significative changes of nightly cardiorespiratory trends are documented. (2A) After the onset of atrial flutter (*) the heart rate goes up to 120 bpm and at the same time the respiratory pattern drastically changes (arrow): irregular respiratory pattern substitutes for previous regular oscillations; frequent drops in saturation from baseline such as appears in patients suffered for SAS are documented. (2B) Just the heart rate becomes regular the spO2 trend stabilized without significant fluctuations.

Discussion

Atrial fibrillation (AF) and atrial flutter (AFlut) are the commonest cardiac arrhythmias with significant related morbidity and mortality and with prevalence rising above all in individuals aged > 75 years [3]. Onset of AF has been explained by several mechanisms, including variations in autonomic nervous system. The heart is richly innervated by the autonomic nerves and a pivotal role in its regulation is played and keeped by maintenance of sympatho-vagal balance. The effects of changes in this system affect also other apparatus and systems, such as the respiratory system, above all with cycling differences while awake and while asleep. An increasing number of researches in recent period have linked SAS to AF/AFlut through, among other co-factors, an altered trigger on sympatho-vagal system. Nightly desaturations due to apnoic events are a harmful trigger on cardiovascular system eliciting arrhythmias by various mechanisms. On the other hand, cardiac stability could influence respiratory activity through physiopathological pathways even now unknown, including for example regular perfusion and chemoreceptor activation, normal fluctuating pulmonary receptors perfusion or physiological neurohumoral activation between heart and lung. Heart rate variability (HRV) may be a useful tool to monitor autonomic nervous system (ANS), and in SAS patients it is found an increased sympathetic dominance when compared with controls. At the same time also in subjects with cardiac disease an altered autonomic activity is documented. When SAS and cardiac disease were contemporanely considered, findings suggest a decreased sympathetic dominance in those subjects suffering from both conditions, as well as in subjects with SAS that will develop cardiovascular disease, which was reflected in a significantly reduced sympathovagal balance [4,5]. Even if it is well known that SAS may trigger AF, the reciprocal relationship is not so well estabilished. Since the beginning of this century some researches have tried to give a different key of lecture moving from the heart rate stability and regular heart activity. In the 2002 Garrigue et al. studied 15 patients with central or obstructive sleep apnea who had received permanent atrial-synchronous ventricular pacemakers for symptomatic sinus bradycardia; analyzing data from sleep time, pacing or spontaneous rhythm time and apnea or hypopnea index, it is found that atrial overdrive pacing significantly reduces the number of episodes of central or obstructive sleep apnea without reducing the total sleep time [6]. But these findings were not confirmed three years later, although both of studies considered a small sample of patients [7]. During the last decades several researches succeded in this opposite view of the matter adding different causes for reflection but without a univocal result. In 16 patients with AF or AFlut and SAS (apnea-hypopnea index >10) receiving cardioversion or ablation of cavotricuspidal isthmus, sleep polygraphy was perfomed before and immediately after restoring sinus rhythm. Despite of maintenance of sinus rhythm no significant changes were found in apnea-hypopnea index, so SAS could not be improved by cardioversion of AF/AFlut [8]. In another study 138 consecutive patients with AF or AFlu were underwent to nightly cardiorespiratory polygraphy before and immediately after electrical cardioversion in absence of ventilation therapy. The results documented a reduction of AHI within the first night after CV due to a significant decrease above all in central respiratory events [9]. As known a patient with cardiovascular disease may be affected by different features of SAS, and different pattern of apnea could be experienced various effects to restore regular sinus rhythm. Sleep time, sleep efficiency, time in different sleep stages, and subjective daytime sleepiness were normal when detected in 23 patients with AF and SAS underwent to cardioversion. Although SAS is highly prevalent in patients with persistent AF, obstructive sleep apneas are not statistically influenced by the cardioversion of AF to sinus rhythm [10]. The real interdependence between AF or AFlu and SAS is still controversial, above all during and after the sudden transition from those ones to sinus rhythm, such as shown in this case report adding new informations moving from another point of view. Based on the previous studies presented and the data from the clinical practice through this case, it is possible to consider others features of this complex physiopathological loop linking atrial arrhythmias and apnoic events. In patients suffering of SAS the cascade of events, leading to the sleep fragmentation, arousal and changes in intrathoracic pressure, provokes a negative impact on the cardiovascular system, also through an hyperactivation or disregulation of nervous system (parasympathetic and ortosympathetic). But at the same time regularitazion of heart rhythm could have a positive influence on nightly desaturation and sleep pattern. Heart rate stability guarantees a costant and more regular blood flow, with resulting in a different receptors activation for the breathing control at various levels. As known the control of respiration is due to a central command, to a chemical check by receptor of glomus cells, and in addtion to a various signals feeding back to pulmonary receptors. Central neuronal net with rhythmic activity and rhythmic oscillations of membrane potential (such as a pacemaker activity like in the heart) and peripheral chemoreceptors located in carotid and aortic glomus cells receive by the vascular capillary net not only nutrition but also useful chemical and physical informations. Moreover, J receptors are sensitive termination in the alveolus activated by stress condition like oedema or congestion, they seem to be responsible to increase of respiratory rate and dyspnea. An arrhythmia (AF or AFlu) results in a period of high heart rate, irregular beat and consequent irregolar blood distibution to periferical and central system of control (both for flow rate and for blood gas levels). Thus, the restore of regular blood circulation due to regular rhythm might be the substrate to a regular perfusion of the pulmonary circulation with conseguent decrease of pulmonary receptors activation. The more regular circulation involves positive changes in PO2 and PCO2 values, their stabilization is monitored by a costant glomus cells surveillance. All of these biochimical data producted probably are sent to central control to stabilize nightly respiratory pattern.

Conclusions

Although most findings could move to a different conclusions, there is undeniable link between atrial arrhyhtmias ad sleep apnea in terms of regularization of heart rate and consequent stabilization of nightly respiratory pattern. This relationship develops more likely through several physiopathological pathway not completely know. The features of this clinical case could offer an alternative key of lecture of these complicated mechanisms, focusing on the importance of heart rate stability in the nightly cardiorespiratory balance.

References

1. Berry RB, Budhiraja R, Gottlieb DJ, Gozal D, Iber C, Kapur VK, Marcus CL, Mehra R, Parthasarathy S, Quan SF, Redline S, Strohl KP, Davidson Ward SL, Tangredi MM; American Academy of Sleep Medicine. Rules for scoring respiratory events in sleep: update of the 2007 AASM Manual for the Scoring of Sleep and Associated Events. Deliberations of the Sleep Apnea Definitions Task Force of the American Academy of Sleep Medicine. J Clin Sleep Med. 2012 Oct 15;8(5):597-619. doi: 10.5664/jcsm.2172.
2. Stevenson IH, Teichtahl H, Cunnington D, Ciavarella S, Gordon I, Kalman JM. Prevalence of sleep disordered breathing in paroxysmal and persistent atrial fibrillation patients with normal left ventricular function. Eur Heart J 2008;29(13):1662–9.
3. Camm AJ, Kirchhof P, Lip GY, Schotten U, et al. European Heart Rhythm Association, European Association for Cardio-Thoracic Surgery. Guidelines for the management of atrial fibrillation: The Task Force for the Management of Atrial Fibrillation of the European Society of Cardiology (ESC). Europace. 2010;12:1360–420.
4. Milagro J, Deviaene M, Gil E, Lázaro J, Buyse B, Testelmans D, Borzée P, Willems R, Van Huffel S, Bailón R, Varon C. Autonomic Dysfunction Increases Cardiovascular Risk in the Presence of Sleep Apnea. Front Physiol. 2019 May 17;10:620. doi: 10.3389/fphys.2019.00620. eCollection 2019.
5. Lombardi C, Pengo MF, Parati G. Obstructive sleep apnea syndrome and autonomic dysfunction. Auton Neurosci. 2019 Nov;221:102563. doi: 10.1016/j.autneu.2019.102563. Epub 2019 Jul 10.
6. Garrigue S1, Bordier P, Jaïs P, Shah DC, Hocini M, Raherison C, Tunon De Lara M, Haïssaguerre M, Clementy J. Benefit of atrial pacing in sleep apnea syndrome. N Engl J Med. 2002 Feb 7;346(6):404-12.
7. Pépin JL1, Defaye P, Garrigue S, Poezevara Y, Lévy P. Overdrive atrial pacing does not improve obstructive sleep apnoea syndrome.Eur Respir J. 2005 Feb;25(2):343-7.
8. Lissel C1, Hennigs S, Hoffmann-Castendiek B, Gardiwal A, Oswald H, Welte T, Klein G. Am J Cardiol. 2008 Sep 15;102(6):709-11. doi: 10.1016/j.amjcard.2008.04.052. Epub 2008 Jul 2. Effect of restoring sinus rhythm on sleep apnea in patients with atrial fibrillation or flutter.
9. Fox H, Bitter T, Horstkotte D, Oldenburg O. Cardioversion of atrial fibrillation or atrial flutter into sinus rhythm reduces nocturnal central respiratory events and unmasks obstructive sleep apnoea. Clin Res Cardiol. 2016 May;105(5):451-9. doi: 10.1007/s00392-015-0940-2. Epub 2015 Nov 9.
10. Höglund N, Sahlin C, Kesek M, Jensen SM, Franklin KA. Cardioversion of atrial fibrillation does not affect obstructive sleep apnea. Ups J Med Sci. 2017 Jun;122(2):114118.doi:10.1080/03009734.2017.1291545.Epub 2017 Mar 14.