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Marked improvement of heart failure upon adequate titration of continuous positive airway pressure in a patient with obstructive sleep apnea

Znaczna poprawa kliniczna u chorego z niewydolnością serca i zespołem obturacyjnego bezdechu sennego po właściwym ustaleniu stałego, dodatniego ciśnienia w drogach oddechowych

Abstract

Sleep disordered breathing (SDB) is frequently present in heart failure (HF), and it may take the form of obstructive (OSA) and central (CSA) sleep apnea. The use of continuous positive airway pressure (CPAP) in patients with OSA and HF is associated with an improved neuroendocrine profile and cardiac function. The degree of upper airway obstruction and the airway closing pressure (and the PAP pressure used to relieve it) may all be highly variable in a setting of uncontrolled HF, mostly due to variable airway oedema. We present a case of a man with HF whose cardiac symptoms radically improved after adequate treatment of his OSA with an auto-adjusting PAP device.

Key words: continuous positive airway pressure, heart failure, obstructive sleep apnea

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Streszczenie

Zaburzenia oddychania w czasie snu często towarzyszą niewydolności krążenia i mogą przybierać postać zespołu obturacyjnego i ośrodkowego bezdechu sennego. Użycie aparatów wytwarzających stałe dodatnie ciśnienie w drogach oddechowych (CPAP, *continuous positive airway pressure*) u chorych na niewydolność krążenia ze współistniejącym zespołem obturacyjnego bezdechu sennego prowadzi do poprawy profilu neuroendokrynnego i polepszenia funkcji serca. Obturacja dróg oddechowych (jak również ciśnienie CPAP konieczne do jej pokonania) u chorego na niewydolność krążenia może podlegać dużym zmianom spowodowanym zmiennym obrzękiem dróg oddechowych. W niniejszej pracy przedstawiono opis pacjenta z niewydolnością krążenia, którego objawy sercowo-naczyniowe znacząco poprawiły się w czasie leczenia współistniejacego zespołu obturacyjnego bezdechu sennego po właściwym ustaleniu ciśnienia przy użyciu aparatu automatycznie dostosowującego dodatnie ciśnienie w drogach oddechowych (AutoPAP).

Słowa kluczowe: stałe dodatnie ciśnienie w drogach oddechowych, niewydolność serca
zespół obturacyjnego bezdechu sennego

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Introduction

Sleep disordered breathing (SDB) is present in 45–76% of patients with heart failure (HF) [1, 2]. It may take the form of obstructive sleep apnea (OSA), related to dynamic airway narrowing during sleep, and central sleep apnea (CSA), caused by an exaggerated ventilatory response, prolonged circulation time, and pulmonary and airway congestion [3]. The use of continuous positive airway pressure (CPAP) in patients with OSA and HF is associated with an improved neuroendocrine profile and cardiac function [4]. The degree of upper airway obstruction and the airway closing pressure (and the PAP pressure used to relieve it) may all be highly variable in a setting of uncontrolled HF, mostly due to variable airway oedema. We present a case of a man with HF whose cardiac symptoms radically improved after adequate treatment of his OSA with an auto-adjusting PAP device.

Case report

A 71-year-old man with coronary artery disease with prior coronary artery bypass graft surgery and heart failure with mildly decreased left ventricular systolic function (ejection fraction of 55%) presented for repeated sleep evaluation. One year earlier he was diagnosed with severe obstructive sleep apnea (OSA) with an apnea-hypopnea index (AHI) of 55/h^{-1}; during the PAP titration study, he responded to bi-level positive airway pressure therapy in the spontaneous mode (BPAP-S) set at inspiratory pressure of 11 cm H_2O (providing maintenance of inspiratory airflow) and expiratory pressure (controlling airway obstruction of OSA) of 7 cm H_2O. He reported improvement of daytime sleepiness. His subjective compliance with BPAP treatment was very good, with reported nightly use of at least 6 hours. His PAP machine did not have a compliance card.

In spite of continued therapy with spironolactone 50 mg twice daily, furosemide 40 mg once daily and carvedilol 6.25 mg twice daily his HF remained poorly controlled. He continued to have exertional dyspnea and dyspnea at rest, and experienced worsening peripheral oedema with an increase in body weight from 236 lbs (107.3 kg) at the time of the sleep study (body mass index, BMI = 33.9 kg/m^2) to 247 lbs (112.3 kg). No orthopnea or paroxysmal nocturnal dyspnea was present. No renal insufficiency, uncontrolled hypertension or cardiac ischaemia, dietary indiscretion, heart arrhythmias or medication non-compliance was present. His echocardiogram showed a moderately enlarged left atrium, no mitral regurgitation, grossly normal left ventricular size and function, and diastolic dysfunction. He was admitted to the hospital for a trial of intravenous diuretic therapy.

Physical examination on admission showed minimal bibasilar crackles and 2+ peripheral oedema; laboratory investigations demonstrated normal basic metabolic profile and complete blood count with mild normocytic anaemia with haemoglobin concentration of 11.6 g/dl. Chest radiograph showed cardiomegaly (Fig. 1) and no vascular congestion. He was treated with furosemide 80 mg twi-

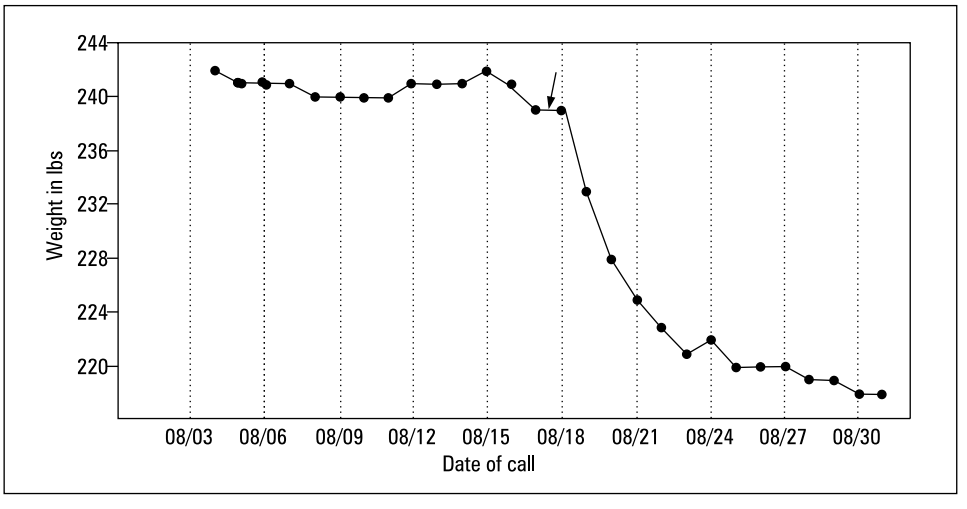

Figure 1. Changes in weight (Y axis, in lbs) in time (X axis, based on daily phone report) before and after institution of AutoPAP therapy (arrow)

Rycina 1. Zmiany masy ciała (oś Y, w funtach) w czasie (oś X, na podstawie raportów telefonicznych) przed i po zastosowaniu terapii AutoPAP (strzałka)

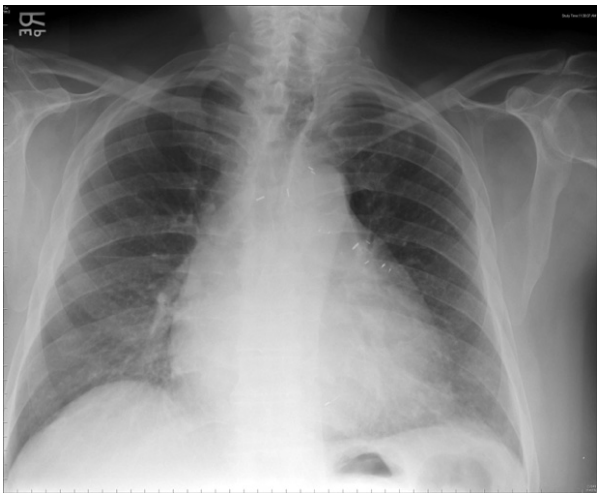

Figure 2. Postero-anterior chest radiograph, showing cardiomegaly and no lung parenchymal changes

Rycina 2. RTG klatki piersiowej w projekcji przednio-tylnej, widoczne powiększenie sylwetki serca, brak zmian w miąższu płuc

ce daily; his other medications were continued at previous doses. This therapy led to no change in weight and minimal improvement in symptoms, but resulted in prerenal azotaemia (blood urea nitrogen of 70 mg/dl and blood creatinine of 2.0 mg/dl). Polysomnography with PAP titration was performed and the optimal pressure was determined to be 12 cm H_2O. No central sleep apnea activity was present. Given the increase in the expiratory PAP requirement from the previous study from 7 to 12 cm H_2O, an auto-adjusting PAP (AutoPAP) set at 5-15 cm H_2O was introduced and the patient was discharged home with no change in medication profile. Over the following two weeks the patient experienced marked diuresis with improvement in symptoms and decrease in weight from 247 lbs (112.3 kg) to 216 lbs (98.2 kg), documented by daily phone weight reporting system (Fig. 2). The patient noted less exertional dyspnea. The download of the compliance card on his device showed that the 90^{th} percentile pressure generated by the machine was 11 cm H_2O. The patient continues to do well after six years and continues to be compliant with his PAP treatment.

Discussion

Sleep-disordered breathing (SDB) in patients with congestive heart failure may take the form of obstructive and or central (CSA) sleep apnea; application of continuous positive airway pressure in HF patients is associated with an improved neuroendocrine profile and cardiac function [4]. The beneficial effects of positive airway pressure therapy in HF patients with SDB are twofold. First, PAP treats the obstructive component of sleep-disordered breathing — it acutely splints the airway, thereby restoring airflow. This has the mechanical effect of preventing wide intrathoracic pressure swings and by decreasing arousals, prevents neurohormonal activation. Second, by introducing positive intrathoracic pressure, PAP decreases cardiac preload, transmural left ventricular pressure, and afterload. The net result of these pneumatic and haemodynamic effects is typically, a decrease in cardiac output [5]. These changes lead to improvements in left ventricular ejection fraction, neurohormonal profile and quality of life, and also result in reduced airway oedema and airway resistance [6, 7].

In our patient, introduction of positive airway pressure therapy initially led to improvement in his daytime sleepiness although symptoms of HF continued despite apparently good compliance. A repeat PAP titration polysomnogram identified that the expiratory pressure set on his BPAP-S device was insufficient to completely control his airway obstruction. Importantly, there was no tendency towards central sleep apnea in the patient during that study. An AutoPAP, a device that can adjust the pressure within the set range in response to the variable obstruction, was then employed to assure airway patency in the face of any changes in airway resistance related to HF-related oedema. Subsequent to the application of the AutoPAP at the pressure that controlled upper airway obstruction, a marked diuresis ensued with concomitant resolution of HF symptoms.

Airway oedema varies with changes in the circulating volume status and the PAP pressure required to keep the airway open (the 'closing pressure') may also vary. Heart failure is a prime example of such changes. Using an AutoPAP device in our patient allowed a dynamic response to these changes and full control of airway obstruction. Complete, rather than incomplete treatment of upper airway obstruction may result in reduction of intrathoracic pressure swings and reduction in preload and afterload swings. In our opinion, this full restoration of airflow was responsible for the patient's clinical improvement.

AutoPAP devices are capable of adjusting the pressure to maintain airway patency [8]. Patients with HF, whose sleep disordered breathing may take the form of both obstructive and central sleep apnea, are not proven candidates for AutoPAP devices and are infrequently treated with this modality. The concern is that in some HF patients, AutoPAP may treat the decrease in airflow due to central sleep apnea as an airway obstruction and may inappropriately respond to it by raising the pres-

sure [9]. Since there are no reliable predictors of central apneas in HF patients, polysomnography is needed to rule out central apnea activity prior to the introduction of an AutoPAP in this patient population. A new bi-level positive airway pressure device that uses adaptive servo technology with the ability to adjust expiratory pressure promises to resolve both obstructive and central apnea activity [10].

In conclusion, providers caring for patients with refractory heart failure should consider performing a sleep study in patients at risk for, or with symptoms of sleep apnea. Additionally, in patients with known sleep apnea, incomplete treatment should be considered. We postulate that in the absence of central or complex sleep apnea, automatically adjusting PAP devices may be useful in managing SDB of HF patients with dynamic, volume status-dependent airway size.

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