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Obstructive sleep apnea: acute effects of CPAP on polyssonographic variables

Apneia obstrutiva do sono: efeitos agudos do CPAP sobre variáveis polissonográficas

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Abstract

Introduction: The use of non-invasive ventilation in the form of continuous positive airway pressure (CPAP) is among the main therapeutic options for patients with obstructive sleep apnea (OSA). Yet the effects of CPAP obtained on the first night of use are underreported. **Objective**: To evaluate the acute effects of CPAP on polysomnographic variables in patients with OSA. **Materials and methods**: This study is a case series with 31 patients (55.8 ± 11.4 years; 22 men) in the initial phase of CPAP treatment. The subjects were evaluated by means of polysomnography with and without CPAP (10.2 ± 3.1 cmH_2O) and without CPAP, on different days, by means of the following variables: sleep stages 1, 2 and 3 (N1, N2 and N3), rapid eye movement (REM) sleep, apnea and hypopnea index (AHI), AHI in REM sleep (AHIREM) and the micro-arousal index (MAI). **Results**: The use of CPAP resulted in a reduction of N2 ($p < 0.001$), AHI ($p < 0.001$), AHIREM ($p < 0.001$) and MAI

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(p = 0.001). There was an increase in N3 (p = 0.006) and REM sleep (p < 0.001) during the night with use of CPAP. **Conclusion**: This study demonstrated that, from the first night of use by patients with OSA, CPAP promotes greater balance between sleep phases, and improves sleep quality. These results should be presented to patients and their families in order to encourage greater adherence in the initial phase of treatment with CPAP.

Keywords: Obstructive sleep apnea. Continuous positive airway pressure. Polysomnography.

Resumo

Introdução*: O uso de ventilação não invasiva sob a forma de Continuous Positive Airway Pressure (CPAP) está entre as principais opções terapêuticas no manejo de pacientes com apneia obstrutiva do sono (AOS). No entanto, os efeitos obtidos logo na primeira noite de uso do CPAP ainda são pouco relatados.* ***Objetivo****: Avaliar os efeitos agudos do CPAP sobre variáveis polissonográficas em pacientes com AOS.* ***Materiais e métodos****: Trata-se de uma série de casos, com um total de 31 pacientes (55,8 ± 11,4 anos; 22 homens) em fase inicial de tratamento com o CPAP no Instituto do Sono de Santa Maria (RS). Os sujeitos foram avaliados pela polissonografia sem e com CPAP (10,2 ± 3,1 cmH_2O), em dias diferentes, por meio das seguintes variáveis: estágios do sono 1, 2 e 3 (N1, N2 e N3), sono REM (*rapid eyes moviment*), índice de apneia e hipopneia (IAH), IAH no sono REM (IAHREM) e índice de microdespertares (IMD).* ***Resultados****: Houve diminuição com o uso do CPAP no N2 (p < 0,001), IAH (p < 0,001), IAHREM (p < 0,001) e IMD (p = 0,001). O N3 (p = 0,006) e o sono REM (p < 0,001) aumentaram durante a noite com o CPAP.* ***Conclusão****: Este estudo demonstrou que o CPAP, logo na primeira noite de sua utilização, promove um maior equilíbrio entre as fases e melhora na qualidade do sono de pacientes com AOS. Esses resultados devem ser apresentados aos pacientes e aos seus familiares, visando estimular maior adesão na fase inicial do tratamento com o CPAP.*

Palavras-chave*: Apneia obstrutiva do sono. Pressão positiva contínua nas vias aéreas. Polissonografia.*

Introduction

Obstructive sleep apnea (OSA) is characterized by recurrent episodes of pharyngeal collapse, causing partial or total obstruction of air flow, and resulting in cardiorespiratory and neurological imbalance during sleep, in addition to promoting changes in behavioral characteristics and daytime neurocognition (1, 2). The sudden changes of lung pressures and volumes cause oxygen desaturation, respiratory acidosis, activation of the sympathetic nervous system and imbalance in sleep stages (3). In OSA, the sleep phases are interrupted by episodes of apneas and hypopneas, which can result in reduced vitality and overall mental and physical health (4). These combined factors predispose patients to higher rates of morbidity and mortality (5, 6).

Among the main therapeutic options in the management of patients with OSA, non-invasive ventilation in the form of continuous positive airway pressure (CPAP) is indicated as the first line of treatment (7), with great results on quality of sleep and life (8), improved functional condition (9) and positive repercussions on cardiovascular risk and mortality rates (5). Widely prescribed and used by physical therapists, the use of CPAP reduces episodes of apneas and hypopneas and levels of inflammatory markers such as C-reactive protein, tumor necrosis factor-α and Interleukin-6 (10).

The main challenges to the implementation of and adherence to CPAP arise from difficult adaptation to airflow, the interface used and noise nuisances reported by some patients (11-13), which result in non-adherence in the first month of treatment by 30% of patients (14). However, given the importance of CPAP for the management of OSA, publication of the immediate effects of this therapy may be a strategy to enhance initial adherence by patients to treatment. Thus, this study aimed to investigate the acute effects of CPAP on the first night of use on polysomnographic variables in subjects with OSA.

Materials and methods

This study is a case series whose participants included 31 patients with clinical diagnosis of OSA being

treated at the Sleep Institute of Santa Maria (SISM), who were randomly selected between December 2012 and March 2013. The project was approved by the Research Ethics Committee of the Federal University of Santa Maria (CAAE: 08798612.0.0000.5346/2012), as per resolution 196/96 of the National Health Council, and all subjects signed a free and informed consent form.

Patients of both sexes were included in the study, with no age limit, and each underwent the basal nocturnal polysomnography (PSG). Patients with cognitive dysfunction that would prevent the understanding of evaluations, with a diagnosis of chronic lung disease, smokers and other sleep-related disorders were excluded from the study.

The subjects were selected based on the analysis of patient records, and evaluated according to the routine schedule of the health care service. The PSG was performed on the first night for diagnosis of OSA and basal data collection, and on the second night the PSG was performed in association with use of CPAP, for titration of pressure to be established, and definition of the optimal parameters for each patient.

Polysomnographic evaluation

The patients filled out a screening document with records of body weight, height and body mass index (weight/height^2). They then received guidelines regarding installation of the electrodes on their head, face and chest, temperature sensors and movement of air in their nostrils and mouth. After all doubts were clarified, the electrodes were installed on the patient, who was requested to lie down, relax and try to sleep (15). The PSG evaluated sleep stages 1, 2 and 3 (N1, N2 and N3), rapid eye movement (REM) sleep, apnea-hypopnea index (AHI), AHI in REM sleep (AHIREM), average peripheral oxygen saturation during sleep (SpO_2med), average heart rate during sleep (HRmed), micro-arousal index (MAI) and total sleep time (TST). The evaluation was overseen by a trained specialist using a digital polygraph (Homed brand Icelera™ Fast-poly model, São Paulo) with six channels for electroencephalography (EEG), two channels for electrooculography, two channels for electrocardiogram, two channels for chin electromyogram, two channels for leg electromyogram, two extra channels for legs, bruxism or EEG, one channel for SpO_2 and one channel for heart rate. Additionally, air flow sensors, thoracic and abdominal straps, a microphone for snoring, position sensor, pressure transducer with flow cannula, pressure transducer with cannula for snoring and an extra channel for the CPAP were used. These were associated with pulse oximetry and a video system integrated with infrared to monitor the polysomnographic variables and vital signs.

Installation and CPAP titration

Initially, the most suitable type of mask for the patient's breathing pattern (oronasal mask for oral respirator or nasal mask for nasal respirator) was defined. The mask was placed on the face of the patient without handles for compression, using the CPAP with minimum pressure of 4 cmH_2O for around five minutes to adapt to the interface and air flow. After this period, the patient lay down and started sleeping under the supervision of a physical therapist with technical training and experience in the management of CPAP during sleep, which remained attentive to physiological changes and responses during the entire period. CPAP pressure was increased by 1 cmH_2O in the following situations: occurrence of two or more obstructive apneas, three or more hypopneas, five or more microarousals, and three minutes or more of loud or unequivocal snoring in a period less than or equal to five minutes. Titration was performed in ascending order according to the respiratory events observed, remaining for a period of ≥ 30 minutes without the occurrence of event, and maintaining a maximum of 20 cmH_2O (16) for approximately 5 hours.

Statistical analysis

The software Statistical Package for the Social Sciences (SPSS) version 13.0 was used. Analysis of data distribution was performed by means of the Kolmogorov-Smirnov normality test. The data are presented as mean, standard deviation and percent variation. Comparison between interventions occurred by paired Student's t-test. A probability of less than 5% was considered to be statistically significant ($p < 0.05$).

Results

A total of 22 men and 9 women participated in the study, aged 55.8 ± 11.4 years, and with a body mass index of 30.9 ± 5.7 kg/m^2. The pressure used in CPAP titration was 10.2 ± 3.1 cmH_2O. During the night on which titration was performed, there was improvement in vital signs, with higher values of SpO_2med and reduction of HRmed. As regards sleep stages, there was reduction in N2 and increase in N3 and REM sleep during the evaluation with use of the CPAP. The results also showed reduction in AHI, AHIREM and MAI values. N1 and TST did not change with the use of CPAP (Table 1).

During the night with the use of CPAP, there was a reduction of HRmed values by 4.3%, of N2 by 19.6%, AHI by 64.2%, AHIREM by 67.1% and MAI by 54.4%. An increase in 1.9% in SpO_2med, 22.8% in N3 and 67.9% in REM sleep was also observed.

Table 1 - Physiological and polysomnography values with and without use of CPAP

	without CPAP	with CPAP	p value
HRmed (bpm)	65.6 ± 9.2	62.8 ± 9.3	0.012
SpO2med (%)	91.5 ± 2.7	93.2 ± 2.3	< 0.001
Stage 1 (%)	3.3 ± 2.0	3.7 ± 2.7	0.387
Stage 2 (%)	64.9 ± 9.6	52.2 ± 9.4	< 0.001
Stage 3 (%)	20.6 ± 9.0	25.3 ± 7.9	0.006
REM sleep (%)	11.2 ± 6.0	18.8 ± 6.8	< 0.001
AHI (episodes/h)	56.2 ± 24.9	20.1 ± 15.8	< 0.001
AHIREM (episodes/h)	59.9 ± 23.3	19.7 ± 18.2	< 0.001
MAI (episodes/h)	23.2 ± 17.2	10.6 ± 8.6	0.001
TST (minutes)	305.3 ± 38.8	314.6 ± 30.5	0.312

Note: HRmed = average heart rate during sleep; SpO_2med = average peripheral oxygen saturation during sleep; AHI = apnea-hypopnea index; AHIREM = apnea-hypopnea index in REM sleep; MAI = micro-arousal index; TST = total sleep time.

Discussion

The main findings of this study demonstrate that on the first night of use, CPAP reduced N2, AHI, AHIREM, MAI and HRmed values, while N3, REM sleep and peripheral oxygen saturation increased.

Among the effects of CPAP, the identification of decrease in N2 stage and increase in N3 stage and REM sleep enabled a broader analysis, which indicated a greater balance between the phases of sleep, with consequent improvement of their quality. The N2 stage is an intermediate stage of non-REM sleep, characterized by low-voltage waves, loss of consciousness, and decreased muscle tone and heart and respiratory rates in relation to awake time and N1 (17). It is the longest stage of sleep, occupying 45% to 55% of TST, and its reduction observed in this study is possibly the result of the decrease in MAI, because the occurrence of microarousals interrupts the N3 stage, with the cyclical return and increase in the N2 stage (18).

The N3 stage is considered the deepest sleep, when the greatest cardiorespiratory balance occurs, and its duration should compose TST by a minimum duration of 15% (17). At this stage, an individual has absence of eye movements, and EEG features present slow waves of low frequency and high amplitude (delta waves). Hormonal release linked to growth also occurs in this stage, as well as recovery from cellular homeostasis and daily fatigue (15). The findings of this study show that on the first night of CPAP usage there was an increase in N3 stage, which can be decisive for improvement in the overall quality of sleep and effective recovery of daytime fatigue.

Another important finding of this investigation was the increased period of REM sleep, which

approached the normal value defined as 20% to 25% of the TST (17). Low-amplitude, mixed electroencephaligraphic frequencies, with rapid eye movement and low muscle tone, occur when an individual is in REM stage. In this phase, oneiric activity is more present, and the processing of day-to-day information, related to learning and memory, occurs (19). Thus, even with only a single night of CPAP use, there may be hope for improvement of cognitive aspects by increasing REM sleep among patients with OSA.

The first night of CPAP usage enabled important reduction in AHI and AHIREM values, with some patients even ceasing to present apnea and hypopnea, which demonstrated the drop in frequency of interruptions from, or reductions in, air flow during sleep. These events increase intermittent hypoxia, which exacerbates some sub-clinical conditions such as endothelial dysfunction and atherosclerosis (20). Considering a frequency of 20 to 30 events per hour as moderate OSA, and more than 30/h as severe (21), the mean AHI value of these patients enabled them to be classified as moderate OSA when treated with CPAP for just one night. However, it should be noted that this is a momentary response, and should not be regarded as a staging parameter of the disease. The decrease in AHI from 56 episodes/h to 20 episodes/h, even in a period of definition of the ideal parameters of the CPAP, may result in improvement in sleep architecture and OSA symptoms. As regards the relevance of AHI records, one recent study suggested the Effectiveness of Treatment Apnea-Hypopnea Index, developed to estimate control of OSA by AHI with different therapies (22).

The significant reduction in MAI value in response to CPAP identified in our study is also of paramount importance to the management of OSA, because these events promote fragmentation of the sleep architecture and induce sympathetic activation. This imbalance in the central nervous system causes elevated systemic blood pressure and heart rate, which has deleterious effects on the cardiovascular system (3). In that respect, MAI emerges as an indirect predictor of cardiovascular risk in patients with OSA (23, 24). The mechanism capable of explaining the reduction in MAI is that CPAP increases the end expiratory lung volume (25), and causes caudal traction of the trachea while maintaining continuous air flow (19).

During the night of CPAP titration there was an increase in peripheral oxygen saturation, which can be expected due to some typical effects of positive pressure ventilation. Among them, the increase in pulmonary compliance, vital capacity, residual functional capacity, and reduction of respiratory work and rate (26). Moreover, the phenomenon of opening the Kohn pores and Lambert canals is well-documented, both responsible for intercommunication between the alveoli (27), which optimizes oxygen diffusion and consequently increases peripheral saturation. Patients also showed a reduction in HRmed, which can be explained by the effects of CPAP on improved thoracoabdominal balance (28), which decreases left ventricular transmural pressure (29), with a consequent reduction in afterload, improvement in ejection fraction, reduction in left ventricle wall thickness and cardiac work (30).

The analysis and description of the acute effects of CPAP are indispensable to offer a feedback proposal to patients. This strategy is being examined by our group, which may be adopted in order to improve the initial management of patients with OSA indicated for CPAP treatment. Adherence to treatment was not evaluated in this study, but may be a subject of future research.

As a routine in specialized services on evaluation and treatment of sleep, the diagnosis of OSA occurs with the first PSG and, based on indication and patient interest, CPAP titration is performed in conjunction with another PSG. This sequence of evaluations may have influenced the results, due to patients being more adapted to the site and monitoring in the second PSG. However, as there was no familiarization with CPAP, the responses may have been compromised during its use.

Conclusion

The results of this study demonstrated that CPAP, from the titration period and in a single night's sleep, reduces fragmentation and promotes improvements in sleep balance and architecture, favoring a greater decrease in nocturnal cardiac work and improved oxygenation. It is recommended that these findings be shared with patients and their families, with the aim of encouraging the use of CPAP and adherence in the initial phase of treatment.

References

1. Patil SP, Schneider H, Schwartz AR, Smith PL. Adult obstructive sleep apnea: pathophysiology and diagnosis. Chest. 2007;132(1):325-37.

2. Sforza E, Roche F, Thomas-Anterion C, Kerleroux J, Beauchet O, Celle S, et al. Cognitive function and sleep related breathing disorders in a healthy elderly population: the synapse study. Sleep. 2010;33(4):515-21.

3. Somers VK, Dyken ME, Clary MP, Abboud FM. Sympathetic neural mechanisms in obstructive sleep apnea. J Clin Invest. 1995;96(4):1897-904.

4. Karkoulias K, Lykouras D, Sampsonas F, Drakatos K, Sargianou M, Drakatos P, et al. The impact of obstructive sleep apnea syndrome severity on physical performance and mental health. The use of SF-36 questionnaire in sleep apnea. Eur Rev Med Pharmacol Sci. 2013;17(4):531-6.

5. Sampaio R, Pereira MG, Winck JC. Psychology morbidity, illness representations, and quality of life in female and male patients with obstructive sleep apnea syndrome. Psychol Health Med. 2012;17(2):136-49.

6. Yaggi HK, Concato J, Kernan WN, Lichtman JH, Brass LM, Mohsenin V. Obstructive sleep apnea as a risk factor for stroke and death. N Engl J Med. 2005; 353(19): 2034-41.

7. Kushida CA, Morgenthaler TI, Littner MR, Alessi CA, Bailey D, Coleman Jr J, et al. Practice parameters for the treatment of snoring and obstructive sleep apnea with oral appliances: an update for 2005. Sleep. 2006;29(2):240-3.

8. Sampaio R, Pereira MG, Winck JC. A new characterization of adherence patterns to auto-adjusting positive airway pressure in severe obstructive sleep apnea syndrome: clinical and psychological determinants. Sleep Breath. 2013;17(4):1145-58.

9. Weaver TE, Mancini C, Maislin G, Cater J, Staley B, Landis JR, et al. Continuous positive airway pressure treatment of sleepy patients with milder obstructive sleep apnea: results of the CPAP Apnea Trial North American Program (CATNAP) randomized clinical trial. Am J Respir Crit Care Med. 2012;186(7):677-83.

10. Baessler A, Nadeem R, Harvey M, Madbouly E, Younus A, Sajid H, et al. Treatment for sleep apnea by continuous positive airway pressure improves levels of inflammatory markers: a meta-analysis. J Inflamm (Lond). 2013;10(1):13.

11. Pires FS, Drummond M, Marinho A, Sampaio R, Gonçalves TPM, Neves I, et al. Effectiveness of a group education session on adherence with APAP in obstructive sleep apnea-a randomized controlled study. Sleep Breath. 2012;17(3):993-1001.

12. Sullivan CE, Issa FG, Berthon-Jones M, Eves L. Reversal of obstructive sleep apnoea by continuous positive airway pressure applied through the nares. Lancet. 1981;1(8225):862-5.

13. Borel JC, Tamisier R, Dias-Domingos S, Sapene M, Martin F, Stach B, et al. Type of mask may impact on continuous positive airway pressure adherence in apneic patients. PLoS One. 2013;8(5):e64382.

14. Poulet C, Veale D, Arnol N, Lévy P, Pepin JL, Tyrrell J. Psychological variables as predictors of adherence to treatment by continuous positive airway pressure. Sleep Med. 2009;10(9):993-9.

15. Epstein LJ, Kristo D, Strollo Jr PJ, Friedman N, Malhotra A, Patil SP, et al. Clinical guidelines for the evaluation, management and long-term care of obstructive sleep apnea in adults. J Clin Sleep Med. 2009;5(3):263-76.

16. Kushida CA, Littner MR, Morgenthaler T, Alessi CA, Bailey D, Coleman Jr J, et al. Practice parameters for the indications for polysomnography and related procedures: an update for 2005. Sleep. 2005;28(4):499-521.

17. Iber C, Ancoli-Israel S, Cheeson Jr A, Quan SF. The AASM Manual for the Scoring of Sleep and Associated Events: Rules, Terminology and Technical Specification. Westchester: American Academy of Sleep Medicine. 2007 [cited 2013, Set 23] Available from: http://www.nswo.nl/userfiles/files/AASM%20-%20Manual%20for%20the%20Scoring%20of Sleep%20and%20Associted%20Events%20-%2005-2007_2.pdf

18. Weaver TE, Sawyer AM. Adherence to continuous positive airway pressure treatment for obstructive sleep apnoea: implications for future interventions. Indian J Med Res. 2010;131:245-58.

19. Ferini-Strambi L, Marelli S, Galbiati A, Castronovo C. Effects of continuous positive airway pressure on cognitition and neuroimaging data in sleep apnea. Int J Psychophysiol. 2013;89(2):203-12.

20. Feng J, Zhang D, Chen B. Endothelial mechanisms of endothelial dysfunction in patients with obstructive sleep apnea. Sleep Breath. 2012;16(2):283-94.

21. Hori T, Sugita Y, Koga E, Shirakawa S, Inoue K, Uchida S, et al. Proposed supplements and amendments to "Manual of Standardized Terminology, Techniques and Scoring System for Sleep Stages of Human Subjects", the Rechtschaffen & Kales (1968) standard. Psychiatry Clin Neurosci. 2001;55(3):305-10.

22. Boyd SB, Walters AS. Effectiveness of treatment apnea--hypopnea index: a mathematical estimate of the true apnea-hypopnea index in the home setting. J Oral Maxillofac Surg. 2013;71(2):351-7.

23. Berry RB, Budhiraja R, Gottlieb DJ, Gozal D, Iber C, Kapur VK, et al. 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;15;8(5):597-619.

24. Basner RC. Continuous positive airway pressure for obstructive sleep apnea. N Engl J Med. 2007;356(17): 1751-8.

25. Wallace A, Bucks RS. Memory and obstructive sleep apnea: a meta-analysis. Sleep. 2013;36(2):203-7.

26. Naughton MT, Rahman MA, Hara K, Floras JS, Bradley TD. Effect of continuous positive airway pressure on intrathoracic and left ventricular transmural pressures in patients with congestive heart failure. Circulation. 1995;91(6):1725-31.

27. Denehy L, Berney S. The use of positive pressure devices by physiotherapists. Eur Respir J. 2001;17(4): 821-9.

28. Cross AM. Review of the role of non-invasive ventilation in the emergency department. J Accid Emerg Med. 2000;17(2):79-85.

29. Meyer E, Lorenzi Filho G, Schettino GPP, Carvalho RR. Ventilação não-invasiva no cardiopata grave. Rev Soc Cardiol Estado de São Paulo. 1998;8(3):420-7.

30. Dursunoglu N, Dursunoglu D, Ozkurt S, Kuru O, Gür S, Kiter G, et al. Effects of CPAP on left ventricular structure and myocardial performance index in male patients with obstructive sleep apnoea. Sleep Med. 2007;8(1):51-9.

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