Clinical and polysomnographic characteristics in patients with morning headache

ABSTRACT

Background: The relation between headaches and sleep disorders are complex and heavily questioned. However, there is still controversy about this interrelationship. Objective: To describe the clinical and polysomnographic characteristics of patients with morning headache, and to compare them with patients without morning headaches. Methods: Prospective study between April and August 2009. One hundred and eight patients were included consecutively and by convenience. All patients were submitted to polysomnography and were distributed in the group with headache (group 1) or the group without headache (group 2). Results: Morning headache was reported by 33 (30.6%) patients, 17 (51.5%, p = 0.02) women. The clinical characteristics in the group of morning headache were 42.4% with disease in upper respiratory system, 72.7% with anxiety, 45% with headache in general, 54% with neurocognitive symptoms, 81.2% reported non restorative sleep and 60.6% had insomnia (all p< 0.05). Among the polysomnographic features surveyed, the only variable that showed statistical significance was wake after sleep onset. Almost 43% (vs. 20%) of patients with morning headaches were in normal range. Conclusions: It was not possible to conclude that the presence of the increase apnea/hypopnea indices, desaturation relevant and intermittent and disruption of sleep patterns are sufficient to modulate, by itself, the occurrence of morning headaches. Sleep disorders can act as a trigger for morning headaches in susceptible individuals with specific clinical profile.

Keywords: Polysomnography, headache, sleep disorders

RESUMO

Introdução: As relações entre cefaleia e distúrbios do sono são complexas e muito questionadas. No entanto, ainda existe muita controvérsia a respeito dessa inter-relação. Objetivo: Descrever as características clínicas e polissonográficas apresentadas por pacientes com queixa de cefaleia matinal, comparando-as com os resultados dos pacientes sem cefaleia matinal. Métodos: Estudo prospectivo realizado entre abril e agosto de 2009. Foram inclusos 108 pacientes com encaixeamento para realizarem polissonografia, de modo consecutivo e por conveniência. Os pacientes eram distribuídos no grupo com cefaleia (grupo 1) ou no grupo sem cefaleia (grupo 2). Resultados: Cefaleia matinal foi relatada por 33 (30,6%) pacientes, sendo 17 mulheres (51,5%; p=0,02). As características clínicas do grupo com cefaleia matinal foram: 42,4% com doenças em vias aéreas superiores, 72,7% com ansiedade, 45% com queixa de cefaleia em geral, 54% com queixas neurocognitivas, 81,2% relatavam sono não reparador e 60,6% tinham insônia (todas com p<0,05). Entre as características polissonográficas pesquisadas, a única variável que mostrou significância estatística foi tempo acordado após início do sono. Quase 43% (vs 20%) dos pacientes com cefaleia matinal estavam na faixa de normalidade. Conclusão: Não foi possível concluir que a elevação do índice de apneia/hipopneia do sono, desaturações relevantes intermitentes e a desorganização da arquitetura do sono sejam suficientes para modular, de forma isolada, a ocorrência da cefaleia matinal. Os distúrbios do sono podem funcionar como um gatilho para a cefaleia matinal em indivíduos predispontos que se apresentam com determinado perfil clínico.

Palavras-chave: Polissonografia; Cefaleia; Distúrbios do sono
INTRODUCTION

Headaches and sleep disorders are much studied morbidity high prevalence in the general population\(^{(1-4)}\) and which entail great damage to the quality of life of patients.\(^{(5-7)}\)

The relationship between sleep and headaches is complex, multifaceted and is questioned about the intercausality between both.\(^{(1,8)}\) In an attempt to facilitate the determination of the relationship between headaches and sleep disorders, ratings were created.\(^{(1,8)}\) Among them, the classification of Paia and Hering,\(^{(1)}\) which determines the following points: a) sleep disorders causing headache; b) headache causing sleep disturbances and c) headaches and sleep disorders triggered by secondary diseases.

Physiopathological indications for the relationship between headache and sleep indicate that the neuroanatomic base for both disorders may be in the brainstem. Periarqueudctal gray matter (PAG) and nucleus raphe magnus (NRM) lesions may induce symptoms of migraine.\(^{(9)}\) In addition, regional blood flow studies show that regions of the brainstem that match the area of neurotransmission and NRM noradrenergic locus ceruleus (LC) are activated during acute attacks of migraine.\(^{(10)}\) Cells in this region play a crucial role in REM sleep (Rapid Eye Movement)\(^{(11)}\) and it is believed that a disturbance in the regulation of the cells can form the basis of narcolepsy/catalepsy.\(^{(9)}\) Recently, functional imaging data reinforced the crucial role of the hypothalamus in trigemino-autonomic headache,\(^{(12)}\) not forgetting the role of the hypothalamus in sleep-vigil cycle.\(^{(13)}\)

Until the second edition of the International Classification of Headache,\(^{(1,8)}\) in 2004, the morning headache was not considered a nosological entity. But in the third edition it was included\(^{(15)}\) as sleep apnea headache (ICHD-3b 10.1.4) in the topic ‘Headache attributed to disruption of homeostasis’. It is known that the headache upon awakening can be part of the clinical picture of patients with obstructive sleep apnea syndrome\(^{(14)}\) (OSA), as well as other respiratory disorders, and headache (primary or secondary).\(^{(15,14)}\)

Thus, one can see that there is a relationship between headaches and sleep disorders. However, in spite of the contributions given by ratings and studies, the only consensus among the various authors is that this relationship is not clear and further studies are needed for better clarification.

MATERIAL AND METHOD

Patients

Between April and August of 2009 were included 108 patients in consecutive mode and by convenience. All patients had to undergo polysomnography (PSG). Patients were oriented on researching and all who agreed to participate signed an informed consent, in accordance with the standards of the Declaration of Helsinki. After that, they filled a questionnaire of pre-polysomnography, in which the presence or absence of morning headache allocated them in distinct groups. They were distributed in Group 1 (with morning headache) or Group 2 (no morning headache). In addition to the analysis of questionnaire data were also considered the polysomnography. Patients younger than 18 years were excluded from the sample. The study was approved by the ethics and Research Committee of the Federal University of Sergipe, process # 0097.0.107.000-09.

Polysomnographic assessment

Polysomnographic assessment was made using the Brain Net System 36 of EMSA. The following variables were monitored: 4 EEG (C3/C4/A2-A1-A2-O1/O2/A1) according to the international system,\(^{(10,20)}\) eletrooculography to the right and left, submentonian electromyogram and electrocardiography. The air flow was monitored by a pressure nasal cannula and a thermistor. Respiratory movements were assessed by thoracic and abdominal straps. Snoring has been evaluated by a microphone around the neck and oxygen saturation during sleep was continuously measured using a pulse oximeter.

Leg movements were recorded with electromyography on the right and the left tibia. PSG data were evaluated according to the criteria of Rechtschaffen & Kales and the new criteria of the American Academy of Sleep Medicine (AASM, 2007). Sleep-disordered breathing was diagnosed following the criteria of the AASM Sleep Scoring Manual for 2007.

Apnea is defined as ≥90% the reduction of the amplitude of the air flow for 10 seconds detected by thermistor. Hypopnea is a reduction in the amplitude of a valid measurement of breath ≥30% of the normal and associated with oxygen desaturation ≥4%. Obstructive sleep apnea and hypopnea are typically distinguished of the central event by detection of inspiratory efforts during the event. The apnea-hypopnea index (AHI) was calculated by the number of apneas and hypopneas per hour of sleep. Sleep apnea syndrome was determined using the criteria: AHI < 5, AHI 5 - <15, AHI 15 - <30 and AHI >30. The patients were classified with mild OSA (AHI 5 - <15), moderate (AHI 15 - <30) and severe (AHI >30). The other parameters assessed by PSG were total time of sleep, sleep efficiency, awake time after sleep onset, duration of REM sleep (NREM 1, 2 and 3), REM latency, REM episodes, total duration of REM, episodes of micro-awakenings
and awakenings, average length of apneas/hypopneas, basal pulse oximetry, desaturation \( \geq \) under 4\%, major desaturation, period of time with desaturation <90\%, number of periodic movements members.

**Statistical analysis**

Variables were summarized by category as simple and relative frequency, and with respective confidence intervals when needed. Comparisons between the groups with and without morning headache were conducted using chi-square test and Fisher exact test. For evaluations of variables related to a given proportion was used a binomial test. Analysis was performed using SPSS version 15. The tests were considered as two-tailed with significance level of \( p < 0.05 \).

**RESULTS**

The sample consisted of 108 patients, with a significantly higher incidence of males (64.8\%). In relation to the age, a predominance of 31-60 years was found (63.9\%; \( p < 0.0001 \)), with ages up 30.9 and \( \geq \)60 years with 15.7\% values and 20.4\%, respectively. Patients with normal body mass index (BMI) represented 25.2\% and 29.9\% was overweighted. However, the obese group surpassed (44.9\%; \( p > 0.05 \)). Sample diagnostic findings are shown in Table 1. In Group 1, almost 88\% of the patients showed respiratory sleep alterations against 81.3\% in Group 2 (\( p > 0.05 \)).

The Group 1 (with morning headache) had 51.5\% (\( p = 0.02 \)) of female patients, with age predominance of 31-60 years (72.7\%). Almost 47\% of the patients had BMI \( \geq \)30 Kg/m\( ^2 \). The Group 2 (no morning headache) had 72\% of men, with 60\% of the patients aging between 31-60 years, and 44\% was obese. The pathological background and clinical picture of the sample are shown in Tables 2 and 3. In Group 1, around 42\% had upper airway diseases, and almost 73\% had complaints of anxiety, and headache (45\%), neurocognitive complaints (54\%), non-restorative sleep (82\%) and insomnia (60.6\%).

Related to the presence of morning headache, 33 patients (30.6\%) reported to be used suffering with it (CI 95\%, 22.1-40.2). Of this total, 51.5\% were female (\( p = 0.02 \)). Of the 33 patients with morning headache, four did not know or did not want to report the characteristics of their headache upon awakening. Of the 29 patients who responded appropriately when asked about the duration of pain, a homogeneous distribution among the options was found. Around 52\% of the patients reported 3-7 points in the visual analogue scale (VAS) and 58.6\% had morning headache 3-4 times a week (Table 4).

Table 1 - Total of patients distributed by diagnosis and the distinction in groups with and without morning headache

|                      | morning headache | no morning headache | \( p \) |
|----------------------|------------------|---------------------|--------|
| Mild OSA\(^a\)       | 9                | 27.3                | 18     | 24.0 |
| Moderated OSA        | 10               | 30.3                | 17     | 22.7 |
| Severe OSA           | 6                | 18.2                | 24     | 32.0 |
| Increased upper airway resistance\(^b\) | 0 | 0.0 | 01 | 1.3 |
| Primary snoring      | 2                | 6.1                 | 11     | 14.7 |
| Other Sleep-disordered breathing\(^c\) | 4 | 12.1 | 01 | 1.3 |
| Without sleep changes| 2                | 6.1                 | 03     | 4.0  |
| Total                | 33               | 100.0               | 75     | 100.0 |

\(^a\) Obstructive sleep apnea syndrome
\(^b\) Increased upper airway resistance
\(^c\) Other Sleep-disordered breathing = patients with relevant desaturation in PSG with no criteria to OSA (AHI elevation).

Table 2 - Pathological background of patients with and without morning headache

|                       | headache | no headache | \( p \) |
|-----------------------|----------|-------------|--------|
| SHT\(^d\)             | 11       | 33.3        | 39     | 52.0 | 0.07 |
| Other heart diseases\(^b\) | 5     | 15.2        | 15     | 20.0 | 0.55 |
| Lung Diseases         | 3        | 9.1         | 8      | 10.7 | 1.0  |
| Upper airways diseases\(^d\) | 14    | 42.4       | 18     | 24.0 | 0.053|
| Anxiety               | 24       | 72.7        | 34     | 45.3 | 0.009|
| Depression            | 7        | 21.2        | 14     | 18.7 | 0.76 |
| Headache              | 15       | 48.5        | 7      | 9.3  | 0.002|
| Hypothyroidism        | 2        | 6.1         | 5      | 6.7  | 1.0  |
| Drug                  | 2        | 6.1         | 4      | 9.1  | 0.53 |
| BZD\(^e\)             | 3        | 9.1         | 2      | 2.7  | |
| non BZD               | 2        | 6.1         | 4      | 9.1  | |
| None                  | 27       | 81.8        | 66     | 88.0 | |
| other                 | 1        | 3.0         | 3      | 4.0  | |

\(^d\) Systemic hypertension
\(^e\) Atrial fibrillation, congestive heart failure, coronary artery disease
\(^e\) Asthma, emphysema, bronchitis
\(^d\) Diseases of upper airways (rhinitis, sinusitis)
\(^e\) Benzodiazepines

Table 3 - Clinical exam of patients with and without morning headache

|                     | headache | No headache | \( p \) |
|---------------------|----------|-------------|--------|
| Excessive sleepiness| 23       | 69.7        | 44     | 58.7 | 0.27 |
| Difficulty to sleep  | 11       | 33.3        | 18     | 24.0 | 0.31 |
| Wake up feeling unrested | 27 | 81.8 | 37 | 49.3 | 0.002|
| Insomnia             | 20       | 60.6        | 18     | 24.0 | 0.002|
| Neurocognitive complaints | 18 | 54.5 | 24 | 32.0 | 0.027|

\(^a\) memory loss, difficulties to think

In Table 5 the polysomnography variables are listed in more details. Among the characteristics surveyed, the only variable with statistical significance was WASO (wake up time after onset of sleep). Analyzing the WASO, 42.4\% of patients from Group 1 were in the range of normality, while 20\% of patients from Group 2 were in that range (\( p = 0.01 \)).
### Table 4 - Morning headache features

| Morning headache features | Total | n (29\(^b\)) | \% |
|--------------------------|-------|--------------|----|
| Duration                 |       |              |    |
| < 30 min                 | 5     | 17.2         |
| 30-60 min                | 7     | 24.1         |
| 1 - 2h                   | 7     | 24.1         |
| 2 - 3h                   | 3     | 10.3         |
| >3h                      | 7     | 24.1         |
| Grade                    |       |              |    |
| mild (1-2)               | 13    | 44.8         |
| moderate (3-7)           | 15    | 51.7         |
| severe (8-10)            | 1     | 3.4          |
| Frequency (week)         |       |              |    |
| 1 - 2                    | 5     | 17.2         |
| 3 - 4                    | 17    | 58.6         |
| >5                       | 7     | 24.1         |

\(^b\) 4 patients did not fill the questionnaire

### Table 5 - Polysomnographic features in the groups with and without morning headache (Segunda parte)

| Group                      | Headache | No Headache | \(P\) |
|----------------------------|----------|-------------|-------|
| Sleep efficiency           |          |             |       |
| Normal                     | 87.9 (29) | 78.7 (59)   | 0.26  |
| Altered                    | 12.1 (4)  | 21.3 (16)   |       |
| WASO\(^a\)                 |          |             |       |
| < 10%                      | 42.4 (14) | 20.0 (15)   | 0.01  |
| 10 - 20%                   | 24.2 (8)  | 50.7 (38)   |       |
| ≥ 20%                      | 33.3 (11) | 29.3 (22)   |       |
| NREM 1\(^b\)               |          |             |       |
| ≤4.9                       | 9.1 (3)   | 4.0 (3)     | 0.21  |
| 5 - 10                     | 30.3 (10) | 20.0 (15)   |       |
| >10                        | 60.6 (20) | 76.0 (57)   |       |
| NREM 2\(^c\)               |          |             |       |
| ≤44.9                      | 33.3 (11) | 41.3 (31)   | 0.17  |
| 45-55                      | 54.5 (18) | 36.0 (27)   |       |
| mais de 55                 | 12.1 (4)  | 22.7 (17)   |       |
| NREM 3\(^d\)               |          |             |       |
| No phase 3                 | 3.0 (1)   | 6.7 (5)     | 0.82  |
| Up to 14.9                 | 36.4 (12) | 38.7 (29)   |       |
| 15 - 20                    | 33.3 (11) | 34.7 (26)   |       |
| >20                        | 27.3 (9)  | 20.0 (15)   |       |
| REM latency\(^e\)          |          |             |       |
| Up to 59min59s             | 21.2 (7)  | 13.3 (10)   | 0.71  |
| 60 - 89min59s              | 39.4 (13) | 38.7 (29)   |       |
| 90 - 119min59s             | 15.2 (5)  | 16.0 (12)   |       |
| >120min                    | 24.2 (8)  | 32.0 (24)   |       |
| REM episodes\(^e\)         |          |             |       |
| Up to 3                    | 21.2 (7)  | 37.0 (27)   | 0.17  |
| 4 - 6                      | 72.7 (24)| 53.4 (39)   |       |
| >6                         | 6.1 (2)   | 9.6 (7)     |       |
| REM Total time\(^e\)       |          |             |       |
| Less than 20               | 45.5 (15)| 57.3 (43)   | 0.38  |
| 20 - 25                    | 33.3 (11)| 21.3 (16)   |       |
| ≥26                       | 21.2 (7)  | 21.3 (16)   |       |

\(^a\) time awake after sleep onset; \(^b\) non-REM sleep phase 1; \(^c\) non-REM sleep phase 2; \(^d\) non-REM sleep phase 3; \(^e\) rapid eye movement

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\(^f\) ventricular extrasystoles; \(^g\) supraventricular extrasystoles; \(^h\) heart rate; \(^i\) electroencephalography; \(^j\) apnea/hypopnea index; \(^k\) apnea/hypopnea

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Headache Medicine, v.9, n.2, p.42-48, Apr./May/Jun. 2018
**DISCUSSION**

The prevalence of morning headache in the average population, according to Ohayon et al. (17, 26) and Ulfberg et al. (18), is 7.6% and 5%, respectively. In our study, the headache upon awakening was reported by 30.6% of the sample. This is according to the expectations, since the sample of patients had various indications for polysomnography and most of the patients had sleep-disordered breathing. In patients with obstructive sleep apnea syndrome the morning headache the prevalence rates ranged from 18 (17) to 74% (19).

According to the American Academy of Sleep Medicine, the obstructive sleep apnea syndrome is a sleep respiratory disorder characterized by recurrent episodes of partial or total obstruction of the upper airway during sleep, which lead to intermittent hypoxia, transient hypercapnia and frequent awakenings, associated with clinical signs or symptoms. Among these, headache, especially the morning headache, has been suggested as a clinical sign found in OSA. However, many authors are questioning this hypothesis.

The morning headache has been linked with other sleep disorders, besides the OSA. Poceta et al. (22) reported similar results in a retrospective study in which the incidence of morning headache in patients with sleep apnea (24%) was not significantly different from those with periodic limb movements disorder (PLMD) and psychophysiological insomnia. Göder et al. (23) found that a higher frequency of morning headache in the sleep lab, not only in patients with OSA, but also in patients with other sleep disorders, compared to healthy subjects (25% versus 3%). In our study we cannot conclude if there is difference in the prevalence of morning headache complaint in the diagnosed sleep disorders, because most of our patients had previous diagnosis of some sleep breathing disorder.

Among patients with morning headache, there was a predominance of females (p<0.05). This is not surprising, since there are several reports in the literature of higher prevalence of headache in women. Stovner et al. reported that 58% of women versus 41% of men in the world present complaint of headaches.

In relation to clinical aspects, more than 43% of the patients of our sample reported upper airways diseases. This figure is high above the one of Alberti et al. (19) (26.3%; p<0.05). In 2004, Ohayon et al. (17) concluded that morning headache was a good indicator for mood disorders and insomnia. Our study found that 60.6% (p<0.05) of the patients complaining of morning headache presented insomnia, corroborating with Ohayon et al. (17). However, a little more than 21% (p>0.05) reported depression. Göder et al. (23) described 32% of patients with morning headache presenting mood disorders. Almost 73% of our patients presented anxiety disorder. Ohayon et al. (17) described that anxiety and depression were significantly more prevalent (28.5%) in patients with morning headache when compared to the control group (5.5%).

According to Idiman et al. (25) 60% of patients with OSA had headache. Alberti et al. (19) described 48.2% in their sample, while in our study a rate of 45.5% was found. Comparing the characteristics of morning headache, a prevalence of moderate pain (51.7%) was seen related to the data described by Goksan et al. (26) The latter reported a moderate severity in 49.7% and light headache in 28.3% of patients with OSA. Unlike Alberti et al. (19) who reported that patients with OSA had morning headache of mild intensity (>47%). When correlating duration and frequency factors of Goksan et al. (26) headache lasted 1-4 hours (37.5%) and >4 hours (35.8%), with month frequency of 9 to 15 times (34.9%) and >15 times (26.3%). Alberti et al. (19) reported that in 47.4% pain lasted 2 hours and none reported more than 5 hours of pain. Ten patients (52.6%) had morning headache attacks 1-5 times a month.

Our patients presented homogeneous distribution concerning to the duration and 58.6% reported frequency of 3-4 episodes per week. It is important to note that our sample was composed of patients with various sleep disorders, however, 84 of 108 patients were diagnosed with obstructive sleep apnea syndrome.

Göder et al. (23) studied patients with various sleep disorders, and noted that the patients presenting morning headaches showed decreased sleep efficiency. In our sample no statistically significant difference between the two groups was found.

Göder et al. (23) also described that patients with morning headaches showed decrease in the proportion of REM sleep. The authors suggest that this change in sleep architecture, with reduction and fragmentation of sleep, can play a role in the morning headache presented by patients with sleep disorders. Aldrich and Chauncey (27) demonstrated that patients with apnea/hypopnea index higher than 30 and awakening headaches spent a significantly smaller percentage of the total sleeping time in REM phase, when compared with patients without morning headache. In our study, both groups showed decreased REM sleep duration (p > 0.05): with Group 1 (45.5%) and Group 2 (57.3%) presenting less than 20% of total sleep time in REM. An association between REM and the onset of chronic paroxysmal hemicrania in headache and cluster headache.
was also described. (28) Many possibilities can raise reasons supporting the changes in REM as related to the symptoms of headache. The decrease in REM phase may be compensatory for the migraine onset in that same phase of sleep. Alternatively, the REM fragmentation can play a role by itself in the generation of migraine. (29)

It is believed that the morning headache could be related to a combination of mechanisms. (30) Among them, a lower oxygen saturation and hypercapnia caused by apnea episodes could be triggering factors. Patients with OSA often suffer desaturations in their sleep, and the decrease of saturation may contribute to the complaints of morning headache in this population of patients. (19,30)

Our study did not find a relationship between complaints of headache and blood oxygenation. Accordingly, Idiman et al. (25) found no statistical significance between headache and apnea/hypopnea or maximum desaturation in patients with OSA. Aldrich and Chauncey (27) described patients with OSA and morning headache comparing with those without morning headache, and found no difference on minimum oxygen saturation during the night. These studies can raise significant doubts on the hypothesis that the desaturation could play a pathophysiological role in relationship between morning headache and OSA.

According to the theories that support the relationship between the commitment of the nocturnal oximetry and/or sleep architecture and the presence of morning headache, in theory our groups of patients should present a higher incidence of morning headache. Whether considering the possibility of a correlation between variables and the presence of headache upon awakening the patients from Group 2 had a tendency to larger impairment of oxygen saturation and sleep structure.

Therefore, in this study, we cannot conclude that only the presence of the elevation of the apnea/hypopnea, a relevant intermittent desaturation and the disorganization of the structure of sleep were enough to modulate the presence of morning headache. The difference of the greater impairment of the architecture of sleep and nocturnal oximetry, evidenced in Group 2, could be explained by the higher incidence of patients with IAH > 30 in this sample (33.3% vs. 18.2%). However, this difference was not statistically significant.

CONCLUSION

The tendency to greater impairment of nocturnal oximetry and the sleep architecture that occurred in Group 2 can arise some questionings: what are the factors that truly determine the emergence of morning headache in patients with sleep disorders? Would the clinical characteristics help to identify the groups predisposed to the manifestation of the headache? Analyzing our results, one would see that the profile of previous pathological conditions and complaints related to the sleep disorder are different between the groups. Group 1 (morning headache) was mainly composed by women with history of upper airways diseases, anxiety, headache, complaints of insomnia, and neurocognitive and non-restorative sleep difficulties. This profile seems to expose a group particularly more vulnerable to disturbances of sleep architecture and desaturations at night. Therefore, it is possible that sleep disorders are triggering factors in patients who have a predisposition to the morning headache.

REFERENCES

1. Alberti A. Headache and sleep. Sleep Med Rev. 2006;10 (6): 431-7.
2. Jennum P, Jensen R. Sleep and headache. Sleep Med Rev. 2002; 6(6):471-9.
3. Sahota P. Morning headaches in patients with sleep disorders. Sleep Med. 2003;4(5):377.
4. Brennan KC, Charles A. Sleep and headache. Semin Neurol. 2009;29(4):406-18.
5. Stovner Lj, Hagen K, Jensen R, et al. The global burden of headache: a documentation of headache prevalence and disability worldwide. Cephalalgia. 2007;27(3):193-210.
6. Galego JC, Moraes AM, Cordeiro JA, Tognola WA. Chronic daily headache: stress and impact on the quality of life. Arq Neuropsiquiatr. 2007;65(4B):1126-9.
7. Müller MR, Guimarães SS. Impacto dos transtornos do sono sobre o funcionamento do dia e a qualidade de vida. Estudos de Psicologia (Campinas). 2007;24(4):519-28.
8. Sahota PK, Dexter JD. Sleep and headache syndromes: a clinical review. Headache. 1990;30(2):80-4.
9. Dahmen N, Kasten M, Wieczorek S, Gencik M, Epplen JT, Ulrich B. Increased frequency of migraine in narcoleptic patients: a confirmatory study. Cephalalgia. 2003;23(1):14-9.
10. Weiller C, May A, Limmroth V, et al. Brain stem activation in spontaneous human migraine attacks. Nat Med. 1995;1(7): 658-60.
11. Datta S. Celllular basis of pontine-geniculo-occipital wave generation and modulation. Cell Mol Neurobiol. 1997;17(3): 341-65.
12. May A, Bahra A, Büchel C, Frackowiak RS, Goadsby PJ. Hypothalamic activation in cluster headache attacks. Lancet. 1998;352(9124):275-8.
13. Alstadhaug KB. Migraine and the hypothalamus. Cephalalgia. 2009;29(8):809-17.
14. Headache Classification Subcommittee of the International Headache Society. The International Classification of Headache Disorders. 2nd edition. Cephalalgia. 2004;24:1-160.
15. Headache Classification Committee of the International Headache Society (IHS). The International Classification of Headache Disorders, 3rd edition (beta version). Cephalalgia. 2013;33(9):629-808.

16. Dodick DW, Eross EJ, Panish JM, Silber M. Clinical, anatomical, and physiologic relationship between sleep and headache. Headache. 2003;43(3):282-92.

17. Ohayon MM. Prevalence and risk factors of morning headaches in the general population. Arch Intern Med. 2004;164(1):97-102.

18. Ulfberg J, Carter N, Talbäck M, Edling C. Headache, snoring and sleep apnoea. J Neurol. 1996;243(9):621-5.

19. Alberti A, Mazzotta G, Gallinella E, Sarchielli P. Headache characteristics in obstructive sleep apnoea syndrome and insomnia. Acta Neurol Scand. 2005;111(5):309-16.

20. Iber C, Ancoli-Israel S, Cherson A, et al. The AASM Manual for the Scoring of Sleep and Associated Events: Rules, Terminology and Technical Specifications. 1st ed. Westchester, IL: American Academy of Sleep Medicine, 2007.

21. Jensen R, Olsborg C, Salvesen R, Torbergsen T, Bekkelund S. Is obstructive sleep apnoea syndrome associated with headache? Acta Neurol Scand. 2004;109(3):180-4.

22. Poceta JS, Dalessio DJ. Identification and treatment of sleep apnea in patients with chronic headache. Headache. 1995;35(10):586-9.

23. Göder R, Friege L, Fritzer G, Stengel H, Aldenhoff JB, Hinze-Selch D. Morning headaches in patients with sleep disorders: a systematic polysomnographic study. Sleep Med. 2003;4(5):385-91.

24. Morillo LE, Alarcon F, Aranaga N, et al. Prevalence of migraine in Latin America. Headache. 2005;45(2):106-17.

25. Idiman F, Ozturk I, Baklan B, Ozturk V, Kursad F, Pakoz B. Headache in sleep apnoea syndrome. Headache. 2004;44(6):603-6.

26. Goksan B, Gunduz A, Karadeniz D, et al. Morning headache in sleep apnoea: clinical and polysomnographic evaluation and response to nasal continuous positive airway pressure. Cephalalgia. 2009;29(6):635-41.

27. Aldrich MS, Chauncey JB. Are morning headaches part of obstructive sleep apnoea syndrome? Arch Intern Med. 1990;150(6):1265-7.

28. Montagna P. Hypothalamus, sleep and headaches. Neurol Sci. 2006;27 Suppl 2:S138-43.

29. Greenough GP, Nowell PD, Sateia MJ. Headache complaints in relation to nocturnal oxygen saturation among patients with sleep apnoea syndrome. Sleep Med. 2002;3(4):361-4.

30. Provini F, Vetrugno R, Lugaresi E, Montagna P. Sleep-related breathing disorders and headache. Neurol Sci. 2006;27 Suppl 2:S149-52.

Correspondence
Paulo Sergio Faro Santos
Departamento de Neurologia
Instituto de Neurologia de Curitiba
dr.paulo.faro@gmail.com

Received: June 22, 2018
Accepted: June 24, 2018