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Research Article

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Sleep Misperception and First Night Effect in Laboratory

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Abstract

Study Objectives: *Cortical hyperarousal, observed in insomnia individuals, could be influenced by sleep quality and objective sleep misperception. Usually, to be considered as a first night effect, a decrease in the quality of sleep during the first recording night in the laboratory is observed. The objectives of this study are to examine cortical arousal differences according to sleeper types between nights and to assess if cortical arousal varies according to degree of misperception.*

Methods: *Power spectral analysis has been performed on the EEG of three consecutive nights for 61 adults; 25 good sleepers, 18 participants with psychophysiological insomnia and 18 participants with paradoxical insomnia.*

Results: *The results suggest a first night effect for all sleepers, irrespective of type. Despite this exacerbation of sleep difficulties, cortical arousal is similar between the different sleeper types. Finally, results suggest that a cortical hypoarousal, expressed by an increase in Delta power, would promote an overestimation of sleep quantity compared to what is observed with polysomnography.*

Conclusions: *The study of this relationship could be a promising avenue for the treatment of insomnia, this one being foremost a complaint of sleep difficulties.*

Keywords: Insomnia, Hyperarousal, Hypoarousal, Cortical arousal, First night effect, Misperception, Overestimation.

Abbreviation List

EEG = Electroencephalography
GS = Good sleepers
INS = Insomnia
PARA-I = Paradoxical insomnia
PSA = Power spectral analysis
PSG = Polysomnography
PSY-I = Psychophysiological insomnia
SE = Sleep efficiency
SOL = Sleep onset latency
TST = Total sleep time
WASO = Wake after sleep onset

Introduction

Insomnia is the most frequent reported sleep disorders, with about 30 to 48% of adults presenting symptoms and 10% having symptoms severe enough to meet diagnostic criteria of chronic insomnia [1, 2]. While studies are increasingly focusing on the cortical mechanisms associated with insomnia, most of them concentrate only on different static characteristics, such as cortical hyperarousal, sleep quantity or misperception. The present study is innovative since it is one of the few which actually observes the interaction of all those concepts of interest, that is, the influence of both quantity of sleep and sleep misperception on cortical hyperarousal, mainly found in insomnia individuals (INS).

Chronic insomnia is defined as a subjective complaint of difficulty initiating, maintaining or early morning awakening for at least three months which causes clinically significant distress or impairment in areas of functioning [3]. The International Classification of Sleep Disorders, second edition distinguished several types of insomnia subgroups, the most frequent being psychophysiological insomnia (PSY-I) and paradoxical insomnia (PARA-I) [4]. Although suffering individuals both have sleep complaints and daytime consequences, PARA-I differ from PSY-I based on the significant discrepancy observed between sleep perception and polysomnography (PSG) recording. PARA-I misperceive their sleep quality, defined as perception of sleep difficulties which are a longer sleep onset latency (SOL) and wake after sleep onset (WASO) and a shorter total sleep time (TST), while PSG seems normal and comparable to the one of good sleepers (GS). In contrast, the perception of sleep quality in PSY-I is corroborated by PSG. The third version of The International Classification of Sleep Disorders [5] has removed these subgroups for lack of clarity in clinical settings, but encourages further research work to provide empirical support for classifying these subgroups.

At the core of insomnia resides hyperarousal or increased cortical activity as suggested by the neurocognitive model [6]. Power spectral analysis (PSA), which represents the activity in different frequency bands, is an objective measure of cortical activity. PSA results usually show that in GS, the transition between wakefulness and sleep is characterized by a decrease in the EEG activity in high frequency bands and an increase in low frequency ones, thereby promoting sleep [7]. INS presents a power distribution pattern different from that of GS, both at sleep-onset and during sleep. In fact, decreased power in low frequency bands (Theta and Delta) and increased power in high frequency bands (Alpha, Beta and Gamma) are very often observed in INS before and during sleep onset, as well as during the night [8-13]. Increased power in high frequency bands has been associated with waking cognitive processes such as attention and perception [14, 15]. These results are consistent with higher cortical arousal in INS and enhanced information processing, interfering with mechanisms allowing the adequate transition from wakefulness to sleep. Interestingly, a relationship between increased cortical activity and sleep quantity underestimation exists. Some researchers have observed that higher Beta activity or a lower Delta/Beta ratio is associated with higher degrees of difference between subjective and objective TST and SOL, while another study reports a negative correlation between Beta activity and WASO misperception [13, 16, 17].

Altogether, these results provide a complicated picture of the link between cortical arousal and misperception. In short, few studies suggest that the degree of misperception is related to cortical arousal. Moreover, some show that PARA-I display a cortical arousal pattern even more pronounced than PSY-I [18]. Nevertheless, the study of the relationship between cortical arousal and misperception remains very exploratory and several limits are to be considered. In fact, most results derive from a single night of PSG recordings (night 1 or 2). This is problematic as sleep patterns can differ across nights, both for INS and GS [19]. In fact, a first-night effect, which refers to the tendency to sleep less during the first laboratory night than on subsequent ones, is often observed in GS. This effect can be observed through the objective measurement of PSG, which typically shows an increase in SOL, REM sleep and deep sleep stages (stages 3 and 4) latency, a decrease in TST, less REM sleep and lower sleep efficiency (SE) [20-22]. While those studies have observed this phenomenon in GS and in INS, others do not observe any differences whatsoever between the first night and consecutive nights in the sleep laboratory [23, 24].

First night effects of REM sleep increased power in Delta, Theta and Beta1 bands and decreased mean power frequency of the second night compared to the first one have been observed in GS [25]. In fact, a slow wave rebound on night 2 can also be observed [26]. As regards to cortical activity distribution over the scalp, an increase in Delta power in the central region during the first night and an increase in Beta power at posterior sites have been observed [27]. Finally, greater activity in Alpha and Gamma bands and lower activity in Theta and Sigma bands was observed during awakenings and while falling asleep on the first night compared to the two subsequent ones [28]. On the other hand, reverse first night effect was even observed in some INS thus reporting a better sleep quality night (greater sleep quantity) on the first night than subsequent ones [29, 30]. These results altogether, seem most consistent with a transient hyperarousal in GS during the first night compared to subsequent ones [25-28]. This could be partly responsible for the typical sleep difficulties defined as a first night effect. Strangely, no study has yet used spectral analysis to examine the relationship between cortical arousal and sleep quantity among INS. Would hyperarousal also be transient in these individuals? In addition, could cortical hyperarousal, as measured by an increase in activity of high power frequency bands and/or decrease in activity of low power frequency bands, be linked to the degree of misperception on different nights spent in the laboratory in INS? The present research aims to further the understanding of the link existing between cortical arousal and sleep misperception, which is assessed through sleep quantity fluctuations.

First, we will investigate if a first night effect is present amongst types of sleepers. It is expected that there will be a first night effect for GS, PSY-I and for objective data in PARA-I. The PARA-I subjective sleep quantity will be similar on consecutive nights since they always misperceive sleep [31].

Secondly, we will investigate arousal differences among groups and between nights (nights 1, 2 and 3). It is expected that GS and PSY-I will display higher cortical arousal during the first night compared to subsequent nights because of a first night effect. On the other hand, for PARA-I, cortical arousal will be similar between nights since they are constantly aroused. There will be no arousal difference between the second and third night as all the participants will return to their usual sleep pattern.

Finally, assessing whether cortical arousal varies according to the degree of misperception is a third objective of the present research. It is expected that arousal will vary according to the degree of misperception: lower cortical arousal will be associated with less discrepancy between objective and subjective data and vice versa.

Methods

Participants

Participants originate from a database of studies conducted between 2004 and 2011, as part of research projects in the Sleep and Cognitive Evoked Potentials Laboratory of Centre de recherche de l'Institut universitaire en santé mentale de Québec (CERVO), from which the project was approved for ethics (#303-2012).

Good sleepers

Participants report being satisfied with their sleep. Participants do not report any subjective complaints of sleep difficulties or daytime sleep-related consequences do not meet insomnia's diagnostic criteria and do not use sleep-promoting medication. They have a mean SE of 85% or more on two weeks of sleep diary and a score lower than 8 on the Insomnia Severity Index [32].

Insomnia participants

The INS meet the following inclusion criteria: (a) presence of a subjective complaint of insomnia, defined as difficulty initiating (SOL > 30 minutes) and/or maintaining sleep (WASO > 30 minutes) and/or early-morning awakening for a minimum of three nights per week; (b) insomnia duration of at least 6 months; (c) insomnia and/or its perceived consequences is responsible of significant distress and/or alteration of functioning and (d) the presence of a subjective complaint of at least one negative consequence due to lack of sleep (e.g. fatigue, irritability). Inclusion criteria match those of the *International Classification of Sleep Disorders* [5] and the *Diagnostic and Statistical Manual of Mental Disorder* [3] for insomnia [33].

Insomnia subgroups: psychophysiological and paradoxical

To ensure that the two subgroups of INS are distinct, the inclusion criteria for the PARA-I and PSY-I match those defined by St-Jean and Bastien [34]. These criteria, which are based on those of Edinger et al., are preferred to the latter as they propose a better discrimination between PARA-I and PSY-I, by putting more operational criteria on the difference between objective and subjective data [35]. To be part of the PARA-I group, individuals must present on two consecutive PSG recording nights: 1) a TST ≥ 380 minutes and a SE ≥ 85%; 2) an overestimation of ≥ 60 minutes of their SOL, underestimation of ≥ 60 minutes of their TST or ≥ 15% of their SE based on the difference between objective (PSG recording) and subjective (sleep diary filled in laboratory) sleep measures. If INS do not meet these criteria, they are then included in the subgroup of PSY-I.

Exclusion criteria for all participants are: (a) Current presence of a medical condition (e.g. cancer, diabetes) or neurological disorder (e.g. dementia, Parkinson's disease) that can significantly disrupt sleep; (b) presence of a major psychopathology (e.g. anxiety disorder, mood disorder); (c) alcohol or drugs abuse during the past year; (d) evidence of another sleep disorder (e.g. sleep apnea index ≥ 10 or periodic limb movement index ≥ 10 during sleep); (e) a score of 23 or higher on the Beck Depression Inventory; (f) use of psychotropic or other medications known to impair sleep (e.g. bronchodilators); and (g) use of a sleep-promoting agent (e.g. benzodiazepines). Participants using medication to facilitate sleep, twice a week or less, had to follow a two-week withdrawal period before entering the study [36].

Research protocol

Procedure

All participants were recruited via newspaper advertisements. Participants were asked to complete the Beck Depression Inventory and Beck Anxiety Inventory, sleep; sleep diary for two weeks and the Insomnia Severity Index [37]. These questionnaires provide all good psychometric validity and reliability [36-38]. Those individuals corresponding to our research criteria were invited for a clinical interview. The Structured Clinical Interview for DSM-IV Axis I Disorders and the Insomnia Diagnostic Interview were administered respectively by a doctoral student in clinical psychology and a sleep specialist [32, 39]. Potential participants were then invited to undergo four consecutive nights of PSG recording at the sleep laboratory. Participants received a thorough evaluation of their sleep and an honorarium for their participation.

Materials

The Insomnia Diagnostic Interview is designed in a semi-structured format and evaluates the presence of insomnia and the factors that can potentially contribute [32].

The sleep diary is a daily diary assessing the subjective quality of sleep [32]. The different sleep-wake parameters measured are: SOL, WASO, morning awakening, awakening frequency during the night, total time spent in bed, TST and SE. The sleep diary is usually completed upon arising each morning for a 2-week baseline period preceding the recording nights. Thereafter, participants complete the sleep diary each morning in the sleep laboratory. A mean value was calculated for each variable of the sleep diary.

The Insomnia Severity Index is an instrument reliable and valid for determining quantitatively the insomnia severity index as perceived by the participant and his entourage [32, 40]. To assess the severity of insomnia symptoms, a severity score of 12 points from the first three questions of the ISI was used rather than the total score. Each item is rated on a 5-point Likert scale. Higher scores reflect higher insomnia severity.

Polysomnographic recordings

Participants slept four consecutive nights in the sleep laboratory. They arrived at the sleep laboratory at around 8:00 p.m. each night to complete electrodes-montage and preparation. They were instructed to refrain from alcohol, drugs, caffeine and nicotine before coming to the laboratory. Bedtime and the time spent in bed are determined by the bedtime and time in bed usually reported in the sleep diary, with a minimum of 8 hours of PSG recording. Lights-out was initiated after bio-calibration and was similar for the three nights of each participant (between 10:30 pm and 7:30 am).

Standard PSG montages (10-20 system) were used for all nights and included electroencephalography (EEG; Fp1, Fp2, F3, F4, Fz, C3, C4, Cz, P3, P4, Pz, O1 and O2), electromyography (chin), electrocardiography (heart) and electrooculography (left and right supra-orbital ridge of one eye and the infra-orbital ridge of the other) recordings. The electrooculography especially allows rejection of EEG artifacts caused by EOG movement or blinking. Moreover, breathing and electromyography (electrodes on the anterior tibialis) are monitored during the first PSG recording night in order to document the presence of other sleep disorders, such as sleep apnea or periodic limb movements. Electrodes were referred to linked mastoids with a forehead ground and interelectrode impedance was maintained below 5 kOhms. A Grass model 15A54 amplifier system (Astro-Med, Inc., West Warwick, USA; gain 10,000; band pass 0.3-100 Hz) was used. PSG signals were digitized at a sampling rate of 512 Hz using a commercial software product (Harmonie, Stellate System, Montréal, Canada). Sleep recordings were scored visually (Luna, Stellate System, Montréal, Canada) by qualified technicians according to standard criteria using 20-seconds epochs. Sleep staging was carried out using central and occipital EEG leads [41].

The four consecutive nights were part of a protocol aimed at studying cortical arousal measured with cognitive evoked potentials before and during sleep onset [42]. The first night was an evaluation and adaptation night. The fourth night is not considered in this study since it was disturbed by the presentation of auditory stimuli. Clinical data derive from nights 1, 2 and 3 rather than a single night, thus increasing data accuracy.

Objective measures of sleep included SOL (defined as lights off with the intention to sleep to the first consecutive minute of stage 2), WASO, TST and SE (as a percentage of TST/total recording time). It is based upon these sleep parameters that the presence of a first night effect and/or reverse first night effect is quantified between the first night and subsequent ones.

Power spectral analysis

The EEG spectral analysis was conducted at C4 by computing fast Fourier transforms (FFT). Manual selection of portions of the nights for the PSA included parts of each sleep stage (stage 1 to 4 and REM) of each sleep cycle, excluding epochs with mini-arousals (0.1-7 seconds), micro-arousals (7.1-14.9 seconds) and arousals (15 seconds and longer), movement time, movements or artefacts and the 5 minutes before and after a stage shift. During waking, artefacts especially due to eye and body movements were manually removed. Within a cycle,

if no uninterrupted period of a specific sleep stage lasted longer than 10 minutes; a portion of this sleep stage was selected while excluding the first and last 40 seconds (two epochs).

PSA was computed on consecutive 4-second epoch, with a resolution of 0.25 Hz and EEG segment length of 20 seconds. PSA is performed only on the frequencies of interest; that are the low (0.25-4 Hz) and high (14-30 Hz) frequency bands, in accordance with the literature suggesting differences mostly in these frequency ranges. Absolute PSA, measurement of the actual power in the designated frequency band, was used to measure cortical arousal.

Misperception

Misperception is defined as a discrepancy between the objective duration of sleep or of awakening and that reported by the participant. A misperception value is calculated with the objective data (PSG recording) and subjective data (sleep diary of each laboratory night using [TST objective-TST subjective], [WASO objective - WASO subjective] and [SOL objective -SOL subjective]. A score of 0 is a perfect estimate of the PSG while a score lower than 0 indicates an overestimation and a score higher than 0 indicates an underestimation of the PSG. Subsequently, the misperception values were transformed into categorical variables, corresponding to discrepancy scores in minutes: 0 to30, 30 to 60, 60 to 90 and over 90 minutes for the underestimation and -30 to 0, -60 to -30, -90 to-60 and -90 or less for the overestimation. These categorical variables were chosen according to insomnia criteria defined as SOL or WASO greater than 30 minutes.

Statical Analyses

The collected data were analyzed using IBM SPSS Statistics version 20 software. One-way ANOVA, Kruskal-Wallis tests, and Chi-square analyses were performed to compare groups on socio-demographic variables and clinical data period Two-way mixed ANOVAs were performed to compare objective and subjective measures, according to nights and sleeper's groups. Given the presence of a similar pattern of strong positive asymmetry and the nature of the objective, which is to verify the effect of the night and diagnosis as well as their interaction, a logarithmic transformation was performed to meet normality assumptions. This transformation has been preferred to the use of nonparametric tests since no equivalence could take into account the interaction and to maintain a reasonable number of statistical tests, which would have significantly decreased statistical power. When the Mauchly's sphericity test indicated that the assumption of sphericity was not reached for the interaction between factors, a Greenhouse-Geisser correction was applied. When the Levene's test for equality of variances was not respected, the nonparametric test of Friedman was used for night effect, while the Kruskal-Wallis test was used per night, to assess differences between groups for each night. Both tests were performed on the original data. Holm-Bonferroni correction was applied for Post hoc multiple comparisons [43].

Thereafter, a linear mixed model including three within-subject factors (night, three levels, night 1, 2 and 3; sleep parameters, three levels, SOL, WASO, TST; frequency band, two levels, low and high) and two between-subject independent factors (misperception, eight levels, -90 and less, -90 to -60, -60 to -30, -30 to 0, 0 to 30, 30 to 60, 60 to 90 and over 90; group, three levels, GS, PARA-I, PSY-I) was computed based on the PSA data of electrode C4. Post hoc pairwise comparisons were performed with a Bonferroni correction. Significant level was set at 0.05.

For all statistical analyses, necessary premises were verified: normality was assessed by visual inspection of the histogram and residual QQ-plots, extreme data corresponding to more than 3.29 standard deviations of a Z distribution were replaced by the score of the last outlier participant within this limit, homogeneity of variance and covariance was assessed respectively by Levene's test and Box's test for equivalence of covariance matrices. When the assumptions were not met, nonparametric alternatives were preferred for non-normal data [44].

Results

Socio-demographic and psychological data

The sample was composed of 61 participants for whom data were available for three consecutive nights of PSG. Statistical analyses revealed that groups were equivalent on age, $K^2(2) = 3.08$, gender, $\chi2(2) = 4.97$, and education, $K^2(2) = 1.131$. Concerning the severity of insomnia, there is a significant difference between groups, $K^2(2) = 42.94$. In agreement with their condition, PARA-I and PSY-I reported greater insomnia symptoms than GS, as shown by a higher score on the Insomnia Severity Index. Regarding psychological symptoms, significant differences between groups were found on depressive and anxiety symptoms $K^2(2) = 12.65$ and $K^2(2) = 17.72$, respectively. Both subgroups of insomnia presented higher scores on the Beck Depression and Anxiety Inventories) than GS. However, all participants remained under the clinical threshold for psychiatric disorders. Table 1 presents the means and standard deviations of sociodemographic and psychological data.

Table 1: Means (SD) of socio-demographic and psychological data of good sleepers (GS), psychophysiological insomnia sufferers (PSY-I) and paradoxical insomnia sufferers (PARA-I).

	GS = 25	PSY-I = 18	PARA-I = 18
Age (years)	36.04 (9.95)	41.33 (8.51)	40.28 (10.13)
Gender (female/male)	14/11	9/9	15/3
Education	3.12 (1.30)	3.17 (1.43)	3.56 (1.10)
Questionnaires			
ISI	1.16 (1.25)[ab]	6.28 (2)	7.06 (1.29)
BDI	3.20 (3.33)[ab]	6.44 (4.38)	6.70 (2.53)
BAI	1.96 (2.22)[ab]	6.53 (5.94)	6.70 (4.82)

ISI = Insomnia Severity Index, 12-point version; BDI = Beck Depression Inventory; BAI = Beck Anxiety Inventory; a Significant difference with PSY-I; b Significant difference with PARA-I. Education was categorized as follow: 1 = Primary, 2 = Secondary, 3 = Collegiate, 4 = Undergraduate, 5 = Graduate, 6 = Postgraduate

Objective and subjective sleep measures

Means and standard deviations of objective and subjective measures for nights 1, 2 and 3 can be found in Table 2. Untransformed data are presented rather than logarithmic, given the greater relevance of these descriptive data. Therefore, it is important to be cautious with the interpretation of data presented in **Table 2**.

Table 2: Means (SD) of subjective and objective sleep parameters of good sleepers (GS), psychophysiological (PSY-I) and paradoxical (PARA-I) insomnia sufferers for each night.

		GS			PSY-I			PARA-I	
	N1	N2	N3	N1	N2	N3	N1	N2	N3
Subjective									
SOL[a]	28.48 (27.80)[b]	18.96 (18.32)[b]	15.71 (19.87)[b]	43.89 (44.18)	29.89 (26.85)	26.39 (21.95)	60.67 (55.64)	53.33 (36.43)	39.33 (44.27)
TST	431.00 (54.41)[b]	446.80 (53.07)[b]	444.20 (53.38)[b]	356.79 (79.41)	382.50 (81.26)	406.79 (54.76)	269.55 (108.87)	305.91 (79.02)	295.91 (81.39)
WASO[c]	19.50 (26.34)[bd]	19.56 (52.67)[bd]	10.08 (17.68)[bd]	48.09 (41.31)	50.35 (42.22)	19.47 (16.75)	47.31 (32.19)	73.85 (41.04)	51.92 (46.48)
SE (%)[c]	89.26 (7.64)[b]	93.50 (7.20)[bd]	92.18 (6.17)[bd]	78.53 (12.39)	87.75 (7.31)	83.24 (10.68)	60.95 (22.47)	62.79 (15.79)	83.61 (10.68)
Objective									
SOL[ae]	18.99 (20.90)	13.63 (17.98)	9.05 (8.04)	21.30 (23.63)	12.98 (9.81)	11.17 (13.35)	21.15 (28.13)	12.02 (10.63)	14.13 (26.48)
TST[ae]	402.89 (45.72)	418.32 (55.99)	417.85 (49.20)	348.19 (68.26)	406.35 (53.68)	416.80 (40.24)	359.89 (59.60)	410.85 (26.38)	391.91 (52.55)
WASO[af]	34.40 (28.95)	27.83 (21.90)	26.33 (25.08)	58.07 (46.34)	53.89 (50.82)	35.52 (32.63)	50.63 (30.66)	48.54 (29.54)	42.80 (45.75)
SE (%)[a]	87.68 (12.76)[b]	90.08 (6.72)[c]	91.96 (4.98)	81.39 (13.21)	82.67 (13.33)	89.11 (7.67)	64.76 (18.00)	83.94 (7.30)	88.00 (9.17)

SOL = Sleep onset latency. TST = Total sleep time. WASO = Wake after sleep onset. SE = Sleep efficiency. [a] Significant difference between night 1 and 3. [b] Significant difference with PARA-I. [c] Significant overall difference between nights. [d] Significant difference with PSY-I. [e] Significant difference between night 1 and 2. [f] Significant overall difference between groups.

Subjective measures

The two-way mixed ANOVAs showed a significant effect of night on SOL, $F(2,104) = 10.85$, participants reporting taking longer to fall asleep during night 1 compared to night 3. There was a significant difference between nights for WASO, $F(2,70) = 4.19$ and SE, $\chi2(2) = 11.33$. However, Post hoc comparisons failed to find more specific differences between nights, given the statistical correction applied. There was no significant difference between nights for TST, $\chi2(2) = 5.46$.

Subsequently, a between groups significant difference was observed for SOL, $F(2,52) = 6.47$, PARA-I reported taking significantly longer to fall asleep than GS. A significant difference for WASO, $F(2,35) = 12.25$ showed that GS reported spending less time awake after the sleep onset than PSY-I and PARA-I. Regarding TST, a difference between groups is present on the three nights (night 1: $K^2(2) = 21.12$; night 2: $K^2(2) = 24.64$; night 3: $K^2(2) = 25.21$. GS reported sleeping longer than PARA-I on all three nights. Concerning SE, a significant between groups difference can be found for the three nights (night 1: $K^2(2) = 22.87$; night 2: $K^2(2) = 39.12$; night 3: $K^2(2) = 16.58$. A better SE was observed in GS than in PARA-I on all nights. Comparisons showed that PSY-I, for their part, had lower SE than GS on nights 2 and 3. No significant interaction Group X Night was found, suggesting a similar subjective estimate of nights 1, 2 and 3 in each group.

Objective measures

Statistical analyses on objective measures showed a night effect for SOL, $F(2, 114) = 15.31$, with a higher sleep onset latency on night 1 compared to night 2 and 3. Thereafter, TST was different between nights, $F(2, 116) = 9.23$. Specifically, all participants slept less during night 1 compared to night 2 and night 3. There was a difference between nights for WASO, $F(2,116) = 7.09$, participants spending more time awake after sleep onset during night 1 compared to night 3. Finally, there was a difference between nights for SE, $\chi2(2) = 17.44$, night 1 displaying a lower SE than night 3.

Regarding differences between groups on objective data, analyses showed a significant difference for WASO, $F(2,58) = 3.39$. As for SE, there was a significant difference between groups for night 1, $K^2(2) = 19.51$, and night 2, $K^2(2) = 7.62$. Comparisons showed a lower SE among PARA-I than among GS for night 1 only. No significant difference was present for the other measures (TST: $F(2, 58) = 2.02$, $p = 0.14$ and SOL: $F(2,57) = 0.14$, $p = 0.87$). No significant Group X Night interaction was found, suggesting a similar sleep pattern between nights 1, 2 and 3 in each group.

Power spectral analysis measures

Cortical activity model

A linear mixed model analysis was performed on PSA data at C4. A linear regression was ran in order to verify premises regarding the linear mixed model. The analysis revealed they were all met: VIF data indicated no collinearity problem, residuals were normally distributed as assessed by histogram and QQ-plot and there was no reason to doubt the independence of residuals. **Table 3** presents the detailed results of the linear mixed model.

Table 3: Linear mixed model analysis results.

Variable(s)	Numerator df	Denominator df	F	Sig.
Intercept	1	116.601	218.36	.00
Group	2	127.732	1.23	.30
Night	2	738.245	.83	.44
Sleep parameter	**2**	**750.993**	**91.71**	**01***
Frequency band	**1**	**700.646**	**337.98**	**01***
Misperception	**7**	**754.478**	**2.20**	**.03***
Group * night	4	739.992	.19	.94
Group * sleep parameter	4	744.396	.87	.48
Group * frequency band	2	700.646	1.42	.24
Night * frequency band	2	700.646	.35	.71
Night * misperception	14	747.849	.53	.92
Sleep parameter * frequency band	**2**	**700.646**	**108.00**	**.01***
Sleep parameter * misperception	12	755.122	1.23	.26
Frequency band * misperception	**7**	**700.646**	**3.35**	**.01***
Group * night * sleep parameter	8	737.523	.32	.96
Group * night * frequency band	4	700.646	.25	.91
Group * night * misperception	17	746.393	.28	.99
Group * sleep parameter * frequency band	4	700.646	.91	.46
Group * sleep parameter * misperception	11	754.329	.87	.57
Group * frequency band * misperception	14	700.646	1.17	.29
Night * sleep parameter * frequency band	4	700.646	.72	.58
Night * sleep parameter * misperception	15	749.102	.56	.91
Night * frequency band * misperception	14	700.646	.62	.85
Sleep parameter * frequency band * misperception	**12**	**700.646**	**2.03**	**.02***
Group * night * sleep parameter * frequency band	8	700.646	.75	.65
Group * night * sleep parameter * misperception	9	748.412	.50	.88
Group * night * frequency band * misperception	17	700.646	.49	.96
Group * sleep parameter * frequency band * misperception	11	700.646	1.17	.31
Night * sleep parameter * frequency band * misperception	15	700.646	.73	.75
Group * night * sleep parameter * frequency band * misperception	9	700.646	.62	.78

Dependent variable = Cortical activity; * = significant difference

Cortical activity and misperception

The sleep parameter X frequency band interaction showed significant differences. However, since these two variables alone are unspecific, interpretation would be inaccurate.

Thereafter, frequency band X misperception interaction was statistically significant. As sleep parameters are not separated from this interaction, it offers little interpretation possibilities.

Finally, the three-way sleep parameter X frequency band X misperception significant interaction provided a clearer interpretation of the variables associated with cortical activity. Post hoc comparisons revealed that differences lied exclusively in the Delta frequency band during TST. **Figure 1** present data interactions for misperception and Delta frequency during TST. Those who underestimated their TST between 0 and 30 minutes displayed greater power in the Delta band than those who underestimated between 30 and 60 minutes. Those overestimating between 0 and 30 minutes their TST displayed greater power in the Delta band compared to all participants underestimating it. Participants who overestimated over 90 minutes appear to be the ones displaying the greatest power within the Delta band compared to all misperception groups. More generally, all of those overestimating their TST had a larger Delta mean power than those who underestimated it.

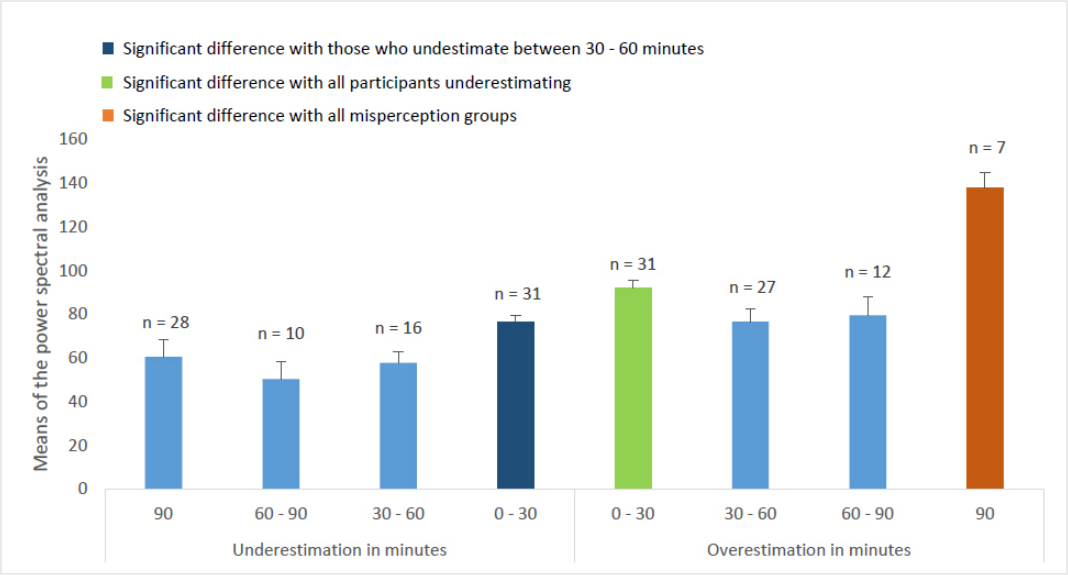

Figure 1: Means of the Delta power according to total sleep time and misperception categories.

Cortical activity, night and sleeper type

There was no significant interaction between spectral activity, sleeper type and night, thus suggesting no effect of these variables on cortical activity.

Discussion

Group's classification

Results of the present study indicate that PARA-I differ on several subjective sleep measures compared to GS. PARA-I perceive significantly longer sleep onset latency and wake after sleep onset while reporting a shorter total sleep time which provide them lower sleep efficiency than GS. These data corroborate those in the literature suggesting that PARA-I have a greater tendency to underestimate their sleep quantity compared to GS [45]. As expected for PSY-I, they report having longer awakening periods during the night compared to GS and despite that they do not report sleeping less than GS, they indicate spending a greater proportion of the night lying in bed without sleep, as shown by the sleep efficiency results. As for the lack of difference between PSY-I and PARA-I, this is explained by the fact that they both have complaints of sleep difficulties, reducing the differences between these groups.

Concerning objective measures, they do not always seem to corroborate the subjective experience of the participants. This surprising similarity between sleeper groups on objective measures, although in agreement with some studies, can first be explained by the variability of the INS sleep pattern. In fact, chronic insomnia is characterized by an important inter-night variability, alternating between good and bad nights in a typical week [46, 47]. This variability can be observed during the laboratory nights, thus reducing the representativeness of the sleep pattern and reducing the differences between sleeper groups normally recorded. In addition, an epidemiological study revealed that the most common type of insomnia is sleep maintenance insomnia [48]. Therefore, it is possible that the objective sleep problems of INS are overrepresented by difficulties maintaining sleep during the recording nights in the laboratory, rather than difficulties equally distributed on all the variables, as suggested by a significant difference in wake after sleep onset.

Night effect

The results of this study confirm the first hypothesis about the first night effect. It appears that the first laboratory night is objectively different from the second and third ones. In fact, the first night exacerbates sleep difficulties of all sleepers. Even PARA-I have the same objectively more pronounced sleep difficulties as others. In fact, longer sleep onset latency, longer awakenings during the night, a shorter total sleep time and more time spent in bed without sleeping, as indicated by lower sleep efficiency, are observed. The first laboratory night also differs from other nights on the sleep onset latency perception. As multiple comparisons are not significant for wake after sleep onset and sleep efficiency, it does not allow for any firm conclusion of the origin of the difference. However, descriptive data suggest that night 1 differs from other nights on these two variables also. Contrary to what we expected, PARA-I appear to have the same subjective alterations during the first night than other participants.

Overall, this study confirms the presence of a first night effect, observable by a decrease in objective and subjective sleep quantity, and therefore the quality, regardless of sleeper type. These results corroborate previous studies [20-23] supporting research protocols using more than one recording night in the sleep laboratory. It would be on the second laboratory night only that sleep would return to a normal laboratory sleep pattern, as indicated by the similarity of the sleep measures of nights 2 and 3.

Cortical arousal depending on the night and the insomnia type

The results of the linear mixed model invalidate the second hypothesis that cortical arousal is different depending of sleeper's groups (GS, PARA-I and PSY-I) and nights (nights 1, 2 and 3). Cortical arousal is similar between GS and both types of INS despite the exacerbation of sleep difficulties during the first recording night. Cortical arousal, as measured with the power spectral analysis, would not be linked to the subjective or objective sleep quantity of the night unlike results observed with cognitive evoked-related potentials [49, 50]. For data suggesting an arousal similarity between groups, they do not seem to be in agreement with the Neurocognitive model which

states that cortical hyperarousal is a static characteristic of INS [6]. However, results on misperception can add valuable information to this model, clarifying the role of cortical arousal on misperception. One hypothesis is raised as cortical arousal is not a feature allowing group differentiation or does not fluctuate with night quality, but as it will be discussed in the next section, rather depends on the degree of misperception. Since INS tend to further underestimate their sleep compared to GS, this could explain why the literature sometimes observed arousal differences between groups, [8,12,13] while results are less obvious in other studies [17,18,51].

Arousal and misperception

The final objective of this study was to assess whether cortical arousal was associated with the degree of misperception. Our results partially support the hypothesis that higher cortical arousal is linked with a greater difference between subjective and objective data and vice versa. This association between cortical arousal and misperception is observed exclusively during sleep, as reflected through total sleep time.

Overestimation of total sleep time

It is light sleep overestimation (between 0 and 30 minutes) and very large overestimation (over 90 minutes) which present greater power activity in Delta bands. Although results may suggest the presence of two subgroups of total sleep time overestimation, an alternative interpretation can be offered. The power high number of participants in this category might increase the representativeness of this group and a greater statistical power. For the group at the extreme end of the overestimation continuum, it is possible that Delta power is so elevated compared to others, that it would explain the observed difference despite the smaller number of participants in this category. Since groups were divided a priori according to sleeper types (GS, PARA-I and PSY-I) and not by the degree of misperception, the distribution of participants is uneven and can explain why differences are not observed for all the overestimation groups. Moreover, those who overestimated the most their total sleep time are also those with more Delta power compared to all other categories of misperceptions. Therefore, it seems justified to say that the overestimation of the total sleep time in general is associated with greater Delta activity.

It would seem that a cortical arousal decrease, represented by an increase in Delta waves, raises the tendency of sleepers to overestimate the time spent asleep. This cortical hypoarousal found among overestimations of total sleep time, or positive misperceptions, is complementary to observations in the literature [13, 16, 18]. While cortical hyperarousal, designated by a greater power in high frequency bands, increases the propensity of total sleep time underestimation, or negative misperception, an increase in Delta waves decreases this hyperarousal and promotes a better sleep estimate [13, 18]. The results from our study even suggest that after a certain decrease in cortical arousal, hypoarousal occurs up to the point where the sleeper would overestimate the time spent asleep. In other words, there would be a cortical hypoarousal, which, rather than generating complaints of sleep, would be linked to a perception biased in favor of night sleep compared to what is observed with PSG. Sleep stages 3 and 4, mainly represented by delta waves, are characterized by a deep sleep, a decrease in response to external stimuli and a higher awakening threshold. Furthermore, the effect of sleep inertia, defined as a transitional state of alteration of the cognitive performance due to a reduced arousability following the awakening, is more pronounced when awakened from deep sleep compared to other sleep stages [52]. Therefore, it is possible that cortical hypoarousal is responsible for the overestimation of the total sleep time via some information processing looseness, cognitive alterations and a decrease in vigilance during awakening. Thus, it increases the difficulty of distinguishing between waking and sleep with a propensity to perceive continuous sleep, without disturbance throughout the night. However, one should be careful while interpreting these results as they suggest an association and by no means causality between variables.

Underestimation of total sleep time

No significant effect in the Beta band was found in association with sleep underestimation. This is surprising as this frequency band has been associated with cortical hyperarousal seemingly responsible for the underestimation of sleep. However, our results suggest that cortical hyperarousal, as defined by an increase in high frequency bands, would have no influence on the perception of a lower quality of sleep than what is objectively observed with PSG for the sleep onset latency, wake after sleep onset and total sleep time. On the other hand, the absence of relationship may partly be explained by the inclusion criteria of high frequency bands as was done in previous research. Subsequently, studies examining the link between cortical arousal and sleep misperception have only taken into account participants overestimating their sleep difficulties or have considered the concept of misperception in its whole, without distinguishing between misperception types. This study is one of the few to have made this distinction, allowing the observation of an additional mechanism between positive and negative misperception.

Misperception in general

Based on our data and those of the literature, we suggest that it is the balance between the proportion of fast and slow waves during sleep which would allow a fair estimation of sleep. Hyperarousal as well as hypoarousal appear both to contribute to sleep misperception and vice versa. While negative misperception is associated with complaints of sleep difficulties despite a normal objective sleep, positive misperception includes individuals who tend not to complain generally about the quality of their sleep. Still, a few individuals report daytime alterations as sleepiness, fatigue and tiredness, given a lower objective sleep [53]. The present study shows the importance of considering the whole continuum of misperception, taking into consideration those who overestimate their sleep difficulties as well as those who underestimate it, this latter group being often neglected.

It would therefore be relevant if other studies could focus on positive misperception, namely if this erroneous perception is the reality of some sleepers or rather a transitional state. These studies could use total sleep deprivation or SWS sleep deprivation in order to observe the misperception degree according to cortical arousal during the recovery night. Since there is a rebound of slow waves sleep during the recovery night, it would be informative for the study of cortical hypoarousal in sleep. The study of this relationship between slow waves sleep and positive misperception could be promising for the treatment of insomnia, which is foremost, a complaint of subjective sleep difficulties. This could further explain the sleep restriction component of cognitive behavioral therapy for insomnia which creates a slight sleep deprivation in order to increase the sleep pressure and an increase in slow wave sleep, which in turn would be responsible for an attenuation of the negative misperception and perhaps even a more favorable perception of sleep than reality being.

Limits

First of all, the power spectral analysis has been carried out exclusively at one central derivation (C4). Although the central electrode is the one that is used in most of the studies since it is the most representative of the brain activity, the study of other sites could have provided more information on the 'distribution' of cortical arousal. In that regards, a study suggested higher activity in Beta band among INS compared to GS in frontal regions of the scalp [54].

Another limitation is the use of a logarithmic transformation for the objective and subjective sleep measures. This transformation might modify the nature of the original data, thus slightly reducing their representativeness and limits interpretation. However, this was the best statistical alternative given the nature of the mixed-design analysis of variance. There is no nonparametric equivalence which allows to take into account the interaction of variables and to limit to a reasonable number of tests.

A third limit is the restricted number of variables used to compare the objective data according to groups and nights. In addition to those used (SOL, WASO, TST and ES), latency and proportion of time spent in each of the sleep stages would have given a broader picture concerning the first night effect and could have contributed to the presence of a significant interaction with cortical arousal. That being said, the choice of variables is consistent with what is most often found within the insomnia literature and allows to limit the number of statistical tests performed, thus increasing statistical power.

Finally, the frequency bands used are limited to waves between 0.25 and 4 Hz and 14 and 30 Hz. It is therefore possible that results would be different with a broader frequency range. Still, existing data in the literature suggest differences more particularly in our chosen frequency bands.

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References

1. Ohayon MM (2002) Epidemiology of insomnia: What we know and what we still need to learn. Sleep Med. Rev 6: 97-111.
2. Morin CM, LeBlanc M, Daley M, Gregoire JP, Mérette C (2006) Epidemiology of insomnia: Prevalence, self-help treatments, consultations, and determinants of help-seeking behaviors. Sleep Med 7: 123-130.
3. American Psychiatric Association (2013) Diagnostic and Statistical Manual of Mental Disorders 5th ed. Washington, USA: American Psychiatric Publishing.
4. American Academy of Sleep Medicine (2005) International Classification of Sleep Disorders: Diagnostic and Coding Manual, 2nd ed. Westchester, USA: American Academy of Sleep Medicine.
5. American Academy of Sleep Medicine (2014) International Classification of Sleep Disorders: Diagnostic and Coding Manual, 3nd ed. Darien, USA: American Academy of Sleep Medicine.
6. Perlis ML, Giles DE, Mendelson WB, Bootzin RR, Wyatt JK (1997) Psychophysiological insomnia: the behavioural model and a neurocognitive perspective. J. Sleep Res 6: 179-188.
7. De Gennaro L, Ferrara M, Bertini M (2001) The boundary between wakefulness and sleep: Quantitative electroencephalographic changes during the sleep onset period. Neuroscience 107: 1-11.
8. Freedman RR (1986) EEG power spectra in sleep-onset insomnia. Electroencephalogr. Clin. Neurophysiol 63: 408-413.
9. Lamarche CH, Ogilvie RD (1997) Electrophysiological Changes During the Sleep Onset Period of Psychophysiological Insomniacs, Psychiatric Insomniacs, and Normal Sleepers. Sleep 20: 724-733.
10. Merica H, Gaillard JM (1992) The EEG of the sleep onset period in insomnia: A discriminant analysis. Physiol. Behav 52: 199-204.
11. Luc Staner, Franc Oise Cornette, Damein Maurice, GeoffreyViardot, Olivier Le Bon, et al., (2003) Sleep microstructure around sleep onset differentiates major depressive insomnia from primary insomnia. J. Sleep Res 12: 319-330.
12. Merica H, Blois R, Gaillard J (1998) Spectral characteristics of sleep EEG in chronic insomnia. Eur. J. of neuroscience 10: 1826-1834.
13. Perlis ML, Smith MT, Andrews PJ, Orff H, Giles DE, et al., (2001) Beta/Gamma EEG Activity in Patients with Primary and Secondary Insomnia and Good Sleeper Controls. Sleep 24: 26-28.
14. Pulvermüller F, Birbaumer N, Lutzenberger W, Mohr B (1997) High-frequency brain activity: Its possible role in attention, perception and language processing. Prog. Neurobiol 52: 427-445.
15. Başar-Eroglu C, Strüber D, Schürmann M, Stadler M, Başar E, et al., (1996) Gamma-band responses in the brain: A short review of psychophysiological correlates and functional significance. Int. J. Psychophysiol 24: 101-112.
16. Maes J, Verbraecken J, Willemen M, De Volder, van Gastel A, et al., (2014) Sleep misperception, EEG characteristics and Autonomic Nervous System activity in primary insomnia: A retrospective study on polysomnographic data. Int. J. Psychophysiol 91: 163-171.
17. Buysse DJ, Germain A, Hall M, Moul DE, Nofzinger EA, et al., (2008) EEG spectral analysis in primary insomnia: NREM period effects and sex differences. Sleep 31: 1673-1682.
18. Krystal AD, Edinger JD, Wohlgemuth WK, Marsh GR (2002) NREM sleep EEG frequency spectral correlates of sleep complaints in primary insomnia subtypes. Sleep 25: 630-640.
19. Vallières A, Ivers H, Bastien CH, Beaulieu-Bonneau S, Morin CM (2005) Variability and predictability in sleep patterns of chronic insomniacs. J. Sleep Res 14: 447-453.
20. Agnew HW, Webb WB, Williams RL (1966) The first night effect: an EEG study of sleep. Psychophysiology 2: 263-266.
21. Mendels J, Hawkins DR (1967) Sleep laboratory adaptation in normal subjects and depressed patients ('first night effect'). Electroencephalogr. Clin. Neurophysiol 22: 556-558.
22. Toussaint M, Luthringer R, Schaltenbrand N, Carelli G, Lainey E, et al., (1995) First-night effect in normal subjects and psychiatric inpatients. Sleep 18: 463-469.
23. Kupfer DJ, Weiss BL, Detre TP, Foster FG (1974) First night effect revisited: a clinical note. J Nerv Ment Dis 159: 205-209.

24. Kader GA, Griffin PT (1983) Reevaluation of the phenomena of the first night effect. Sleep 6: 67-71.
25. Toussaint M, Luthringer R, Schaltenbrand N, Nicolas A, Jacqmin A, et al., (1997) Changes in EEG power density during sleep laboratory adaptation. Sleep 20: 1201-1207.
26. Le Bon O, Staner L, Hoffman G, Dramaix M, San Sebastian I, et al., (2001) The first-night effect may last more than one night. J. Psychiatr. Res 35: 165-172.
27. Curcio G, Ferrara M, Piergianni A, Fratello F, De Gennaro L, et al., (2004) Paradoxes of the first-night effect: A quantitative analysis of antero-posterior EEG topography. Clin. Neurophysiol 115: 1178-1188.
28. Tamaki M, Nittono H, Hayashi M, Hori T (2005) Spectral analysis of the first-night effect on the sleep-onset period. Sleep Biol. Rhythms 3: 122-129.
29. Hauri PJ, Olmstead EM (1989) Reverse first night effect in insomnia. Sleep 12: 97-105.
30. Riedel BW, Winfield CF, Lichstein KL (2001) First night effect and reverse first night effect in older adults with primary insomnia: Does anxiety play a role? Sleep Med 2: 125-133.
31. Salin-Pascual RJ, Roehrs TA, Merlotti LA, Zorick F, Roth T, et al., (1992) Long-term study of the sleep of insomnia patients with sleep state misperception and other insomnia patients. Am. J. Psychiatry 149: 904-908.
32. Morin CM (1993) Insomnia: Psychological assessment and management. New York, USA: Guilford Press.
33. Lacks P, Morin CM (1992) Recent advances in the assessment and treatment of insomnia. J. Consult. Clin. Psychol 60: 586-594.
34. St-Jean G, Bastien CH (2009) Classification of insomnia sufferers based on laboratory PSG recordings and subjective sleep reports. Sleep 32: abstract supplement.
35. Edinger JD, Bonnet MH, Bootzin RR, Doghramji K, Dorsey CM, et al., (2004) Derivation of research diagnostic criteria for insomnia: report of an American Academy of Sleep Medicine Work Group. Sleep 27: 1567-96.
36. Beck AT, Steer RA, Carbin MG (1988) Psychometric properties of the Beck Depression Inventory: Twenty-five years of evaluation. Clin. Psychol. Rev 8: 77-100.
37. Beck AT, Epstein N, Brown G, Steer RA (1988) An inventory for measuring clinical anxiety: Psychometric properties. J. Consult. Clin. Psychol 56: 893-897.
38. Bastien CH, Vallières A, Morin CM (2001) Validation of the insomnia severity index as an outcome measure for insomnia research. Sleep Med 2: 297-307.
39. Spitzer RL, Williams JBW, Gibbon M, First M (1996) Structued Clinical Interview for DSM-IV (SCID). Washington, USA: American Psychiatric Association.
40. Blais FC, Gendron L, Mimeault V, Morin CM (1997) Assessment of insomnia: Validation of three questionnaires. L'Encéphale Rev. Psychiatr. Clin. Biol. Thérapeutique 23: 447-453.
41. Rechtschaffen A, Kales AA (1968) Manual of standardized techniques and scoring system for sleep stages of human subjects. Washington, USA: Brain Information/Brain Research Institute.
42. Bastien CH, St-Jean G, Morin CM, Turcotte I, Carrier J, et al., (2008) Chronic psychophysiological insomnia: hyperarousal and/or inhibition deficits? An ERPs investigation. Sleep 31: 887-898.
43. Holm S (1979) A Simple Sequentially Rejective Multiple Test Procedure. Scand. J. Stat 65-70.
44. Field A (2009) Discovering statistics using SPSS (Introducing Statistical Methods); London, England: SAGE publications.
45. Fernandez-Mendoza J, Calhoun SL, Bixler EO, Pejovic S, Karataraki M, et al., (2001) Sleep misperception and chronic insomnia in the general population: role of objective sleep duration and psychological profiles. Psychosom. Med 73: 88-97.
46. Chambers MJ, Keller B (1993) Alert insomniacs: Are they really sleep deprived? Clin. Psychol. Rev 13: 649-666.
47. Vallières A, Ivers H, Bastien CH, Beaulieu-Bonneau S, Morin CM, et al., (2005) Variability and predictability in sleep patterns of chronic insomniacs. J. Sleep Res 14: 447-453.
48. Taylor DJ, Lichstein KL, Durrence HH, Reidel BW, Bush AJ, et al., (2005) Epidemiology of Insomnia, Depression, and Anxiety. Sleep 28: 1457-1464.
49. Devoto A, Manganelli S, Lucidi F, Lombardo C, Russo PM, et al., (2005) Quality of sleep and P300 amplitude in primary insomnia: a preliminary study. Sleep 28: 859-863.
50. Turcotte I, Bastien CH (2009) Is quality of sleep related to the N1 and P2 ERPs in chronic psychophysiological insomnia sufferers? Int. J. Psychophysiol 72: 314-322.
51. Reynolds CF, Kupfer DJ, Buysse DJ, Coble PA, Yeager A, et al., (1991) Subtyping DSM-III-R primary insomnia: A literature review by the DSM-IV Work Group on Sleep Disorders. Am. J. Psychiatry 148: 432-438.
52. Tassi P, Muzet A (2000) Sleep inertia. Sleep Med. Rev 4: 341-353.
53. Trajanovic NN, Radivojevic V, Kaushansky Y, Shapiro CM (2007) Positive sleep state misperception - A new concept of sleep misperception. Sleep Med 8: 111-118.
54. Corsi-Cabrera M, Figueredo-Rodríguez P, del Río-Portilla Y, Sánchez-Romero J, Galán L (2012) Enhanced frontoparietal synchronized activation during the wake-sleep transition in patients with primary insomnia. Sleep 35: 501-11.

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