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Abstracts from the 1st congress of the Canadian Sleep Society: Sleep Odyssey 2001

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The Canadian Sleep Society (CSS) / Société Canadienne du Sommeil (SCS) is a professional association of clinicians, scientists and technologists formed in June 1986 to further the advancement and understanding of sleep and its disorders through scientific study and public awareness.

Sleep Onset: Shutting Down the Activating System and Closing the Afferent Gateway to the Cerebral Cortex

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Cortical activation and waking are actively maintained by neuronal relays distributed through the brainstem, thalamus, hypothalamus and basal forebrain. Glutamatergic neurons of the reticular formation together with cholinergic and noradrenergic neurons of the brainstem project forward along a dorsal pathway into the thalamus and a ventral pathway into and through the hypothalamus to the basal forebrain. From both the thalamus and basal forebrain, widespread projections serve to activate the cerebral cortex. As an important contingent of the basalo-cortical projection, cholinergic neurons are now known from single unit recording as well as neurotransmitter release studies, to discharge maximally in association with cortical activation (Manns et al., 2000a). Together with neurons of the brainstem reticular formation, the cholinergic basal forebrain neurons decrease firing in association with the irregular slow wave activity of sleep. This decrease could be due to active inhibition by GABAergic neurons that are co-distributed with cholinergic neurons in the basal forebrain, and also with the cholinergic and glutamatergic neurons in the brainstem reticular formation (Maloney et al., 2000). By single unit recording as well as neurotransmitter release studies, particular GABAergic neurons are now known to increase their discharge with irregular slow wave activity of sleep(Manns et al., 2000b). Accordingly, such GABAergic cells can shut down the activating system of the brainstem and basal forebrain at sleep onset.

Within the thalamus, the neurons comprising the nonspecific thalamo-cortical projection and relay system reduce their firing as the glutamatergic, cholinergic and noradrenergic input diminishes from the brainstem and basal forebrain with sleep onset. In addition, the GABAergic neurons of the thalamic nucleus reticularis that surround the thalamo-cortical projection neurons become hyperpolarized and change their pattern of firing from a tonic to a bursting mode, a mode which imposes a strong, temporally patterned inhibition upon the non-specific and specific thalamo-cortical projection neurons (Steriade and Llinas, 1988). Evident in human imaging studies (Hofle et al., 1997), this powerful inhibition of the thalamic nuclei results in the closing of the afferent gateway to the cerebral cortex. Yet, regions of the cerebral cortex, particularly the visual and auditory association areas, remain active while cut off from specific afferent input and thus perhaps free to generate the imagery of which dreams are made.

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Different EEG topographies for kinesthetic and visual images reported at sleep onset

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Relationships between EEG changes at sleep onset (SO) and the production of SO imagery require further clarification and study. In this experiment, we compared the EEG topographies of two types of SO images (kinesthetic vs. visual) sampled from sleep onset stage 4 of the Hori, et al., 1994 scoring system. Twenty-four healthy subjects reported SO images while falling asleep in a seated position. Two judges rated the sensory content of these images. EEG samples preceding the images were recorded from a 19-channel EEG montage while EEG samples from the wakefulness stage preceding each image were similarly recorded. Statistical comparisons were made between the EEG topographies of imagery vs. wakefulness samples and between kinesthetic vs. visual imagery samples.

Relative to preceding wakefulness, imagery samples were characterized by significant decreases in all frequency bands except Delta, for which significant increases were observed over several electrode sites. Kinesthetic images were accompanied by prefrontal and frontal Delta activation; this pattern differed from that of visual images, which involved Delta activation in more left-central and temporal regions.

The results suggest that the rapid anterior to posterior spread of Delta power that is known to occur early in SO may be associated with sense-specific mentation processes unfolding over time. The results are also consistent with a novel explanation for the phenomenon of the 'sleep start' that is commonly accompanied by vivid kinesthetic images of falling at the point of sleep onset.

Behavioural and EEG Evidence for a Sleep Onset Period and Process

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Introduction: Sleep onset is considered by many to be a point, not a process. For practical purposes, sleep is said to begin the moment that alpha levels fall below 50 percent of an EEG epoch[1]. The idea that sleep might theoretically commence much more slowly began to re-emerge about twenty years ago when renewed examinations of behavioural and physiological evidence suggested that the "point" of sleep onset varied according to the measure used to define it. When examining the entry into sleep in detail, significant discrepancies appeared among a number of criteria used to study sleepiness and sleep (i.e., behavioural responsiveness[2], digitized EEG frequencies[3], systematic EEG changes within wakefulness and stage 1 "sleep"[4], most ERP components[3], and sleepiness scales[2].) These measures failed to converge and thereby failed to indicate a "point" or moment of sleep onset. Instead, they define a systematic sequence of events which characterize the Sleep Onset Period and Process.

All mammalian systems examined to date (behavioural, EEG, other physiological measures and indices of consciousness) are profoundly influenced by the wakefulness-to-sleep transition process. Detailed descriptions of these systematic changes will be integrated into a three-step electrophysiological model of CNS regulation during the Sleep Onset Period **– the Alpha-Theta-Sigma Model[5]**. Essentially, this model simplifies Hori's[4] nine EEG W/S stages. The **alpha phase** (Hori stages 1-3) continues until there is no trace of alpha and in it important changes in behavioural, EEG, ERP and subjective activity have begun. In addition to the EEG characteristics, behavioural responses become slow and infrequent, P300 amplitude decreases as do most faster ERP components, decreased respiratory ventilation occurs at the alpha-theta transition, subjective sleepiness, when measurable, approaches maximal levels, and thought-like mentation becomes dream-like as alpha vanishes[6]. In the **theta-vertex phase** (Hori stages 4-8), responses virtually cease as theta gives way to vertex waves and later to incomplete spindles. *We still know very little about correlates of theta, vertex and early spindle activity*. P300 is replaced by N350, and dream reports diminish before spindles begin. The termination of wakefulness is marked by **sigma sleep spindle**-related processes (Hori stage 9). Investigations of sleep onset in people with sleep apnea, narcolepsy, insomnia and mild head injury produce relatively unique electrophysiological patterns which may be of clinical significance.

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Convergence Among Physiologic, Behavioural, and Subjective Estimates of Sleep Onset Latency During Prolonged Wakefulness

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Introduction: The Multiple Sleep Latency Test (MSLT) and the Maintenance of Wakefulness Test (MWT) are paradigms in which the dependent measure is sleep onset latency measured polysomnographically. In principle, however, sleep onset latency could equally well be assessed by self-report or behaviourally. In the present study, sleep onset latencies defined physiologically, behaviourally, and subjectively were investigated under conditions of prolonged wakefulness and their sensitivity to MSLT and MWT instructions were compared.

Method: Thirty-two healthy undergraduates (16 male, 16 female; mean age = 18.9) underwent four test sessions at 2230, 0100, 0330, and 0600 during one night of sleep loss. Each test session consisted of two 30-minute sleep latency tests - one under MSLT instructions and one under MWT instructions - separated by one hour of performance testing. Sixteen of the subjects were also monitored behaviourally during the latency tests by way of a wrist-mounted, vibratory stimulus-response device. Following each test, all subjects were asked to give an estimate of their sleep onset latency.

Results: Latencies to Stage 2 (see Figure 1) decreased with prolonged wakefulness ($F(3, 90) = 168.82$, $p < .001$), were longer under the MWT condition ($F(1, 30) = 28.66$, $p < .001$), and demonstrated an interaction between time of night and instruction ($F(3, 90) = 7.48$, $p < .001$). This general pattern of main effects and interaction was replicated for Stage 1, subjective, and behavioural sleep onset latencies, respectively, with only the interaction for the behavioural parameter falling short of significance (See Figure 2). Secondly, Pearson correlations calculated among all sleep onset parameters for the subjects in the behavioural monitoring condition revealed an average shared variance of 46.3% in the MSLT condition and 71.0% in the MWT condition. Finally, between-parameter comparisons revealed that, on average, behavioural and Stage 1 sleep onset latencies did not differ significantly but were shorter than those identified by Stage 2 which were, in turn, shorter than subjective estimations.

Figure 1
Stage 2 Sleep Onset Latencies

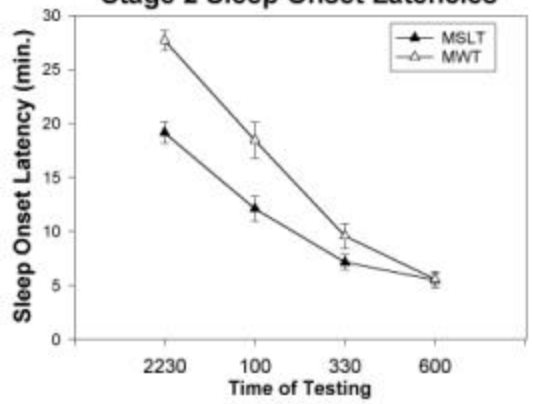
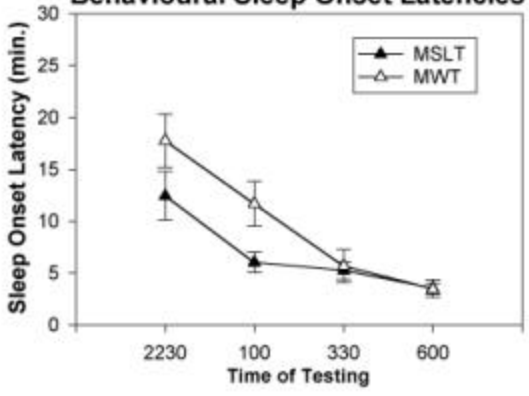


Figure 2
Behavioural Sleep Onset Latencies



Conclusions: Sleep onset latencies measured behaviourally and subjectively displayed a similar pattern of results to those assessed physiologically - they decreased with prolonged wakefulness and varied with test instructions. Furthermore, convergence among the parameters was quite good - particularly under MWT instructions. These results provide additional support for the use of behavioural and subjective indices of sleep onset latency as alternative, or adjunct, measures for assessing sleepiness.

Post-traumatic Delayed Sleep Phase Syndrome and Dim Light Melatonin Onset Test

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Introduction: Circadian rhythm sleep disorders may occur after traumatic brain injury[1]. Here we present a case of a 44-year-old man who developed a prominent delayed sleep phase syndrome (DSPS) consequent on his motor vehicle accident. He suffered what appeared to be only minor injuries, including injuries to a soft tissue of his head, bruises and cuts. He also lost his consciousness for a few minutes and suffered an anterograde amnesia. He did not have any further neurological sequela. However shortly after the accident the patient developed severe sleep onset insomnia (inability to fall asleep before 04:00 h), frequent nightmares, and headaches. The diagnosis of DSPS was established based on several physiological markers of the sleep-wake rhythm: Dim Light Melatonin Onset (DLMO) test[2] using enzyme linked immunosorbent assays (ELISA), wrist activity, sleep log and sleep architecture (EEG).

Method: The patient underwent two overnight sleep studies. On the first night the patient was allowed to go to bed whenever he wanted and to sleep as much as he could. The patient arrived in the sleep clinic at 18:30 h and saliva samples were obtained at hourly intervals from 19:00 to 03:00 h. Ambient light intensity was controlled and did not exceed 15 lx. On the second night, a 24:00 to 08:00 sleep period was imposed.

Results: DLMO test revealed 3 hours delay in endogenous melatonin secretion. DLMO occurred at 23:00 hours. The polysomnographic studies also showed a convincing evidence of delayed sleep phase syndrome. There was normal sleep onset latency (12 minutes), normal sleep duration (7.1 h) and normal sleep efficiency (91.4%) on the night when the patient selected bedtime and rise time. Sleep onset latency was 78 minutes on the second night when conventional bedtime was imposed. Frequent arousals, low sleep efficiency (65.4%) and short sleep duration (5.2 h) were observed on this occasion. Administration of exogenous melatonin 5 mg (Penn Pharmaceutical Ltd) at 18:00 h for 28 days normalised sleep-wake cycle as was evidenced by wrist actigraphy and sleep log. Post-treatment DLMO occurred at 21:00 h.

Conclusion: The learning point of this case is that the DLMO assays can be used as a precise tool to assess the phase of the circadian pacemaker in patients suspected of having a chronobiologic component to their sleep disorder. The abnormal timing of DLMO can also provide an indication for the optimal timing of treatment (medication administration, exposure to light).

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Sleep Disturbances In Patients Affected With Retinitis Pigmentosa

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Introduction: Since light entrains circadian rhythms and thereby influences sleep/wake cycles, we hypothesized that visually impaired patients with RP would have more subjective sleep complaints, sleep disturbances and daytime sleepiness when compared to a control population. We examined for the first time sleep, daytime sleepiness and the ability to stay awake during the day in subjects with Retinitis Pigmentosa (RP), to further delineate photoreceptors' role in the circadian cycle.

Methods: Twelve individuals diagnosed with RP (40±8 yr.) and twelve normally sighted healthy individuals (39±7 yr.) matched for age, body mass index (BMI) and sex were selected for the study. Participants had their sleep recorded on two consecutive nights and were monitored on the two following days. On the first day, their ability to stay awake and on the second, their sleep propensity were assessed using the Maintenance of Wakefulness Test (MWT) and the Multiple Sleep Latency Test (MSLT) respectively. Self-report measures were obtained using the Pittsburgh Sleep Quality Index (PSQI), the Epworth Sleepiness Scale (ESS), and the Toronto Hospital Alertness Test (THAT).

Results: Daytime sleepiness (ESS: 9±5 vs 6±4; $p=0.053$) and sleep propensity (MSLT: 10±5 vs 17±3 min; $p<0.000$) were significantly higher in RP patients than controls, whilst their alertness (THAT: 29± 9 vs 38±7; $p=0.016$) and ability to stay awake (MWT: 21± 9 vs 29± 2 min; $p=0.006$) were reduced. RP participants had more disturbed nighttime sleep, with significantly more awakenings (arousal index: 14±8 vs 8±6/h; $p=0.039$), and tended to have less REM sleep (19±5 vs 22±3 %; $p=0.094$).

Conclusion: Patients with RP have more disturbed nighttime sleep of poorer quality than controls, with increased daytime sleepiness and reduced alertness/wakefulness than their normally sighted counterparts, suggesting an influence of photoreceptor degeneration on the circadian cycle.

Imagery rehearsal for nightmares in children: A nine month follow-up pilot study

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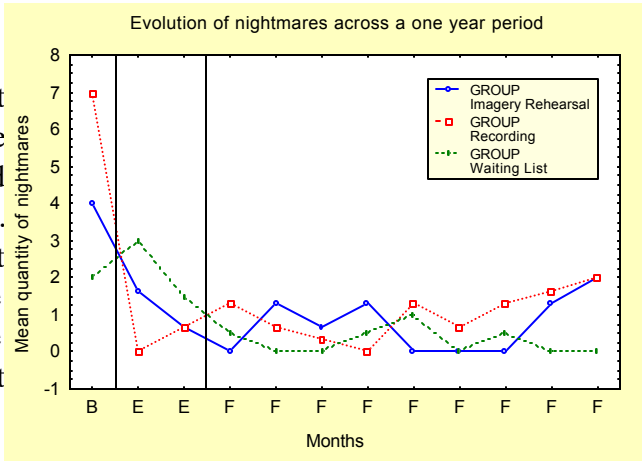
Introduction: Imagery rehearsal for the treatment of nightmares requires the person to modify the nightmare and mentally rehearse the modified version. It has proved to be quite effective in reducing the frequency of nightmares with PTSD victims both at a 3-month period and at a 30-month follow-ups (Krakow et al., 1993). A similar imagery rehearsal approach has been applied successfully with children with nightmares (St-Onge et al., 2000). We are reporting here on assessment of the long-term efficacy of the application of imagery rehearsal in children suffering from nightmares.

Methods: So far, eight children with moderate to severe nightmares (1 or more/week) but without PTSD and aged between 9 and 11 (M=10) have completed the one year study. They attended a first individual meeting to fill in questionnaires. They then kept a daily journal for a 1-month baseline period, recording the quantity of dreams and nightmares and their level of distress. From then on, they were randomly divided into three groups: imagery rehearsal treatment, recording, or waiting list. The treated group (N=3) were asked to record on tape their nightmares and dreams each morning and rate their level of distress in a separate journal for a period of two months. Whenever they recorded a nightmare in the morning, they were trained for and asked to apply the imagery rehearsal treatment (St-Onge et al., 2000) The recording group (N=3) was asked to only record their nightmares and dreams for the same period. The waiting list group (N=2) was asked to note in a journal the number of nightmares and dreams they had during this period. At the end of this two-month experimental period, they all completed the questionnaires again. At this point, the recording and waiting list groups received the treatment. Subsequently, parents and children were contacted by phone each month for a 9-month period for a follow-up concerning the frequency of nightmares.

Results: While there are not enough participants so far to compare the groups statistically, it is clear from Figure 1 that once they all received the treatment, they all maintained a frequency of less than 2 nightmares per month during the follow-up period which is below the criteria to be included in the research.



Conclusions: These preliminary results suggest that the imagery rehearsal treatment is effective to reduce the frequency of nightmares applicable in children and that this efficacy is maintained over a 9-month period. It also seems that the previous grouping did not affect the application of the treatment later on, since the frequency of all three groups remained below the inclusion criteria after they all received the treatment and did not reveal any significant difference.

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Comparing sleepiness and fatigue in a group of shift-workers at an underground mine

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Introduction: Sleepiness is a desire or tendency to fall asleep and fatigue is a sense of physical tiredness with or without physical exertions[1]. Chronic fatigue is more prevalent (20-25%)[1] than excessive sleepiness (5-15%)[2] in primary health care settings and even more in shift-workers[3]. However, fatigue has been poorly understood and under-emphasized compared to sleepiness in clinical practice due to limitations in currently available psychometric tools to measure non-specific fatigue[1,2]. Fatigue and/or sleepiness can be caused by sleep restriction or sleep disruption, either or both can be caused by a broad range of sleep disorders. The goal of this clinical investigation was to compare subjective fatigue and sleepiness to evaluate their possible separation or overlapping in association with underlying sleep pathology.

Methods: This cross-sectional study involved questionnaire based Fatigue Severity Scale (FSS) and Epworth Sleepiness Scale (ESS), which were administered to 196 subjects at an underground mine in Timmins, a Northern Ontario town. The subjects were almost exclusively male (95.7%), 41.9±7.0 years mean age, married (84.2%), working at the mine for 17.6±5.7 years. From the total subject pool, 23 most fatigued and 23 least fatigued miners were selected on the basis of FSS, a 9 item self-report questionnaire providing a subjective measurement of daytime fatigue, independent of daytime sleepiness. The two groups were matched for age, gender and body mass index (BMI). Mean FSS score for the most fatigued subjects was 4.9±5.0 and the least fatigued was 2.0±3.0 ($p< 0.0001$). The subjects from each group had undergone polysomnographic evaluation of sleep to identify abnormality in various sleep parameters on two consecutive occasions at a temporary sleep laboratory in Timmins.

Results: The most fatigued subjects scored 10.0±4.0 on the ESS. There was a weak positive correlation between fatigue and sleepiness in this group (rho=. 504, p= .05). In the group of the least fatigued subjects mean ESS score was 5.5±2.5. No significant correlation between two scores was found (rho= .016, p>.05). The polysomnographic data displayed significant findings of sleep pathology, such as sleep apnea, periodic limb movements and parasomnia in 14 out of 23 (61%) most fatigued subjects. Compared to that, only 4 out of 23 (17%) have displayed similar findings in least fatigued subjects. We also developed a modified Sleep Quality Scale (SQS)[4] to measure severity of abnormal sleep architecture suggestive of non-restorative sleep, which may ultimately produce fatigue or sleepiness. SQS is composed of total sleep time, sleep efficiency, percentage of various sleep stages, arousal index and oxygen desaturation. There was a significant difference between mean SQS scores in most fatigued subjects and in least fatigued subjects (2.9±.3.7 vs. 2.4±.4.3, p=.0005).

Conclusions: It appears from the results of the study that subjective fatigue and sleepiness are not strongly correlated, suggesting their importance as independent phenomena. It also seems plausible that high level of chronic fatigue can be a manifestation of underlying sleep pathology and abnormal sleep architecture, which warrants independent assessment of fatigue and sleepiness severity.

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Mental Activities During SOREMP Within A Multi-Phasic Sleep Wake Schedule

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Introduction: We have found that REM dreams differ qualitatively and quantitatively from NREM dreams when sleep processes before these sleep episodes were controlled by elicitation of sleep onset REM periods (SOREMPs) using the Sleep Interruption Technique (SIT).[1,2] In this study, we modified the SIT into a multi-phasic sleep wake schedule (MPS) to compare mental activities during SOREMPs (REM latencies <25min) and usual REM periods (REM latencies >25 min; REMPs).

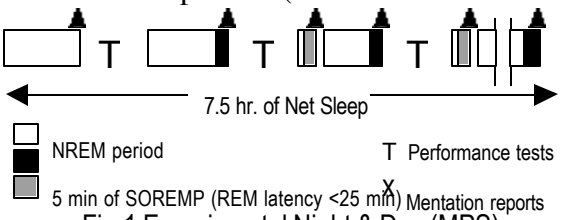

Fig.1 Experimental Night & Day (MPS)

Procedures: Participants were 13 healthy students(10 , 3 , 20.9(1.8)y) without any potential narcolepsy-related symptoms. They spent 3 consecutive (2 adaptation and 1 baseline) nights and 1 night and day (MPS) in the lab. The MPS (Fig. 1) started at midnight on the 4th night and ended when participant's net sleep reached 7.5 hrs (Mean awakening, 19:43 (34min)). During the MPS, following the initial interruption, participants were awakened whenever 5 min of REM sleep appeared and were kept awake for 1 hr with mentation reports and performance tests (grip, flicker, blood pressure, and vigilance test). Participants assessed mental activities using the Dream Property (DP) scales[3,4] for Bizarreness, Evaluation, Impression, and Activity.

Results: A total of 69 SOREMPs appeared during the MPS (Table 1). There were no significant differences of appearance time in SOREMPs and REMPs. Interactions of awakening time and SOREMP/REMP on DP scores were observed (Evaluation; $F(1,24)=4.687$, $p<.05$, Impression; $F(1,24)=3.060$, $p<.1$). Later in the schedule (after 10:00 a.m.), participants reported SOREMP dreams with less impression and increased negative evaluation compared to REMP dreams. Furthermore, sleep paralysis experiences (ISP) were distinct from SOREMP dreams. ISP had more intense Impression ($t(14)=4.652$, $p<.001$) and negative Evaluation ($t(14)=3.991$, $p<.001$; Fig.2).

Table 1 Mentation during SOREMP and REMP

	SOREMP	REMP	Unidentified	Total
Dream	38	8	0	46
Sleep paralysis	5	0	2	7
Nothing	26	11	0	37
Total	69	19	2	90

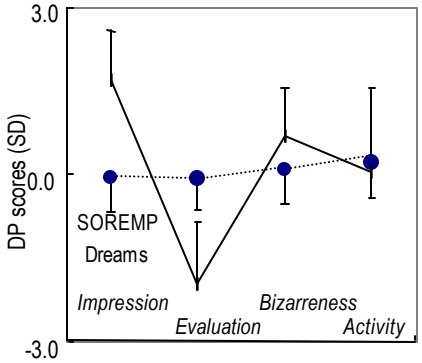

Fig. 2 ISP & SOREMP Dreams

Discussion: Negative evaluation and intense impression in later part of schedule as well as during ISP may suggest that REM mechanisms during SOREMPs reflect increased stress level in participants more than REMP due to their proximity to arousal points.

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Do middle-aged subjects have an earlier circadian phase than young subjects?

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Introduction: Compared to the young, elderly subjects show an earlier habitual sleep timing which is associated with a phase advance of the output of the circadian timing system (1-2). In the middle years of life (20-60y), increasing age is already related to earlier habitual wake time, earlier bedtime and higher morningness-eveningness scores (3). Our aim was to evaluate whether age-related differences in circadian temperature rhythm are associated with changes in habitual sleep habits in the middle years of life.

Methods: Twenty-four healthy subjects matched for gender separated into two groups according to their age: 11 Young (20-39 years; mean: 31.0 ± 4.5 y) and 13 Middle-aged (40-59 years; mean: 49.2 ± 6.4 y) completed a sleep diary for 14 days. Habitual bedtime, wake time, night time in bed and subjective sleep quality were calculated from the sleep diary. Subjects came to the laboratory for consecutive 4 nights and 2 days. Immediately following their habitual wake time of the third night, they entered a mini-constant routine of 25 hours during which rectal temperature was recorded every minute. Subjects were kept awake in bed in constant behavioral and environmental conditions. Estimates of temperature circadian phase were derived from cosinor analysis with a 24h period applied to each subject's temperature rhythm. Habitual phase angle was defined as the time interval between clock time occurrence of the fitted minimum and habitual wake time.

Results: Compared to the young subjects, bedtime and wake time were significantly earlier by one hour in the middle-aged subjects (see Table). No significant difference was found between the groups in habitual night time in bed or in subjective sleep quality or in amplitude of the circadian temperature rhythm or in habitual phase-angle. The Figure illustrates habitual bedtime/wake time and means of body temperature for the two groups. The fitted minimum of the circadian temperature rhythm was significantly earlier by 1:45 hour in the middle-aged compared to the young (see Table). After we controlled for the effect of group, phase was significantly associated with habitual bedtime and wake time. The phase of the circadian temperature rhythm explained 24% of the variance for habitual bedtime and 34% of the variance for habitual wake time.

Table 1: Circadian parameters (Means and SD) for the Young (Y) and Middle-aged (M) Groups

Habitual Sleep timing parameters	**Y (n=11)**	**M (n=13)**	**P**
Wake time	7:51 ± 0:46	6:47 ± 0:56	0.01
Bedtime	23:41 ± 0:48	22:42 ± 0:56	0.01
Night time bed (min)	451.7 ± 44.2	457.5 ± 38.3	n.s
Subjective sleep quality	77.2 ± 9.8	77.4 ± 8.2	n.s
Time of the nadir	6:33 ± 1:14	4:46 ± 1:59	0.02
Amplitude	0.25 ± 0.07	0.28 ± 0.12	n.s
Phase-angle	1:18 ± 1:24	2:01 ± 1:22	n.s
Correlation with phase after controlling for the effects of age			
Habitual Sleep timing parameters	**Beta**	**R^2**	**P**
Wake time	.611	.344	0.001
Bedtime	.520	.249	0.007

Figure 1 :

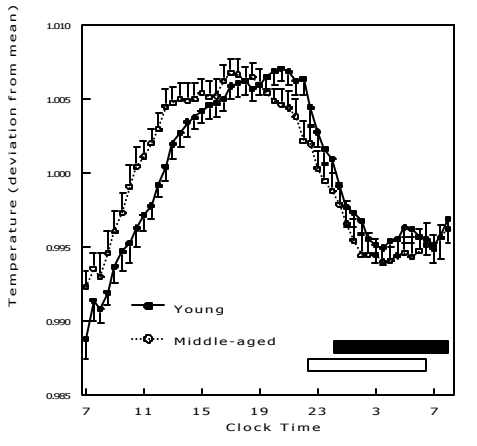

Figure. 30 minutes of body temperature for Y and M.

Conclusions: These results suggest that age-related changes in the timing of the output of the circadian timing system appear as early as the middle years of life.

Reference: 1. Carrier, J. et al. Chronobiology International 1999, 16: 79-91. **2.** Duffy et al. American Journal of Physiology 1998, 275: R1478-R1487. **3.** Carrier J et al. Journal of Sleep Research 1997, 6: 230-237.

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Sleep Quality and Psychological Adjustment in Chronic Fatigue

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Despite their almost universal complaint that they awaken unrefreshed and experience debilitating fatigue, little is known about the sleep characteristics of individuals with chronic fatigue syndrome (CFS). In addition, although psychological maladjustment has generally been assumed, there is little documentation of their psychological functioning. In a pilot study, sleep characteristics and psychological adjustment were measured by questionnaire in 36 individuals with CFS (31 female, 5 male, mean age = 43.4). For 24 participants, polysomnography (PSG) was carried out to screen for physiological sleep disorder (e.g., sleep apnea [SA], restless leg syndrome [RLS], periodic limb movement disorder [PLMD]).

Sleep Characteristics	
Positive finding on PSG (n=24)	62%
Apnea	50%
RLS / PLMD	36%
Mixed	14%
Presence of insomnia (n=36)	88.5%
Mean duration	8 years
Sleep Onset Insomnia	75%
Sleep Maintenance Insomnia	67%
Terminal Insomnia	44%
Unrefreshing Sleep	86%
Sleep Efficiency	74%
Daytime Sleepiness	88%
Mean quality of sleep	3.7 / 10
Mean satisfaction with sleep	3.3 / 10

Psychological Adjustment	
Depression	
Beck Depression - PCI	Low Average
Anxiety	
Spielberger Trait Anxiety	High Average
Rating tension at bedtime	Low Average
Personality (EPQ)	
Neuroticism	Average
Extraversion	Average
Lie (Social Desirability)	Average

- A very high percentage of the CFS participants had physiological sleep disorder on polysomnography assessment. Neither the patients nor their physicians had been aware that they had these disorders.
- Almost all participants reported insomnia of long duration including sleep disruption such as difficulty initiating sleep, difficulty returning to sleep after nocturnal awakening, and terminal insomnia.
- In the daytime, they complained not only of awakening unrefreshed and experiencing extreme fatigue, diagnostic criteria for CFS, but also of feeling sleepy.
- Contrary to popular stereotype, this sample demonstrated good psychological adjustment: Compared to non-clinical samples, depression, anxiety, neuroticism, and extraversion scores were all within normal limits.

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Will late life insomnia improve with the passage of time or does cognitive behavioural treatment help?

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Objective 1: Conduct a comparative evaluation of the nocturnal use of audiobooks and audiotaped relaxation in the management of insomnia and compare these to self-monitoring only. 41 older individuals were randomly assigned to a Self-Monitoring Only control group and to 2 experimental, short-term, non-pharmacological interventions for insomnia: Self-Monitoring plus nocturnal Relaxation or Audiobook. All groups were evaluated in terms of: demographic descriptors; sleep quality; daytime functioning; cognitive-affective variables. Participants were tested pre- and post-intervention and at short (2 week) and long term (approx. 20 month) follow-up. Results showed comparable significant improvement pre- to post-test on sleep parameters and subjective sleep variables in all 3 groups. Gains were generally maintained at long-term follow-up. There was no significant improvement on daytime fatigue or cognitive-affective variables.

Objective 2: In view of the equivalence of the three intervention conditions at post-testing, including the Self-Monitoring Only "control" group, are the "gains that were maintained" really the result of our interventions? We compared pre-test and long-term follow-up scores of two groups of older poor sleepers: those whom we had treated approximately 1-1/2 years earlier and comparable poor sleepers who received no treatment for insomnia between the two testing times. We also examined what happened to older good sleepers during the same time. There were, thus, three groups of participants: Treated Poor Sleepers (n=23), Untreated Poor Sleepers (n=24), and Good Sleepers (n=31). All were tested at the pre and long-term follow-up testing times. Results show that, overall:

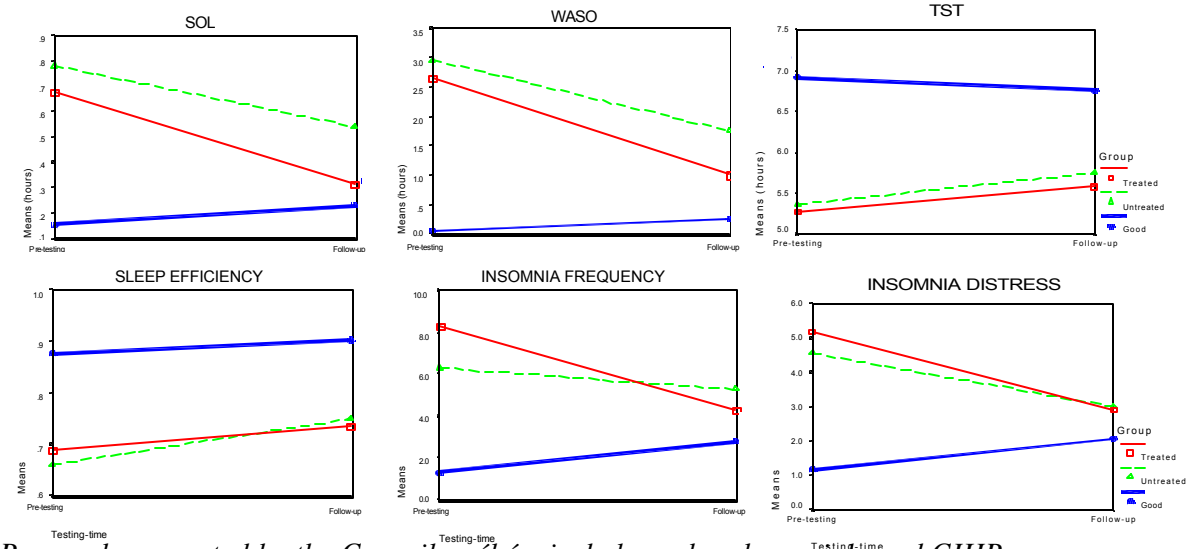
Treated Poor Sleepers improved substantially and significantly on insomnia frequency, insomnia distress and wake after sleep onset (WASO); Untreated Poor Sleepers improved significantly only on WASO.

Good Sleepers had the best scores on all variables at both testing times-Poor Sleepers generally did not come to resemble Good Sleepers over time and they generally remained unchanged or deteriorated very slightly.

Good and Poor Sleepers differed more at pre-test than at the long-term follow-up.

Daytime Fatigue was not consistently related to Good or Poor sleep status and did not change over time.

Conclusions: Are the "gains that were maintained" really the result of our interventions? Yes, cognitive-behavioural therapy, including self-monitoring, affects subjective sleep related variables such as perceptions of insomnia frequency and distress over time. Nocturnal sleep parameters such as WASO may improve spontaneously over time. Good sleep stays that way, generally. Treatment needs to be enhanced to target daytime fatigue and cognitive-affective variables.



Research supported by the Conseil québécois de la recherche sociale and CIHR.

Increased Number and Density of REMs in Individuals of Varying I.Q. Levels Following Acquisition of Two Procedural Tasks

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Introduction: In humans, the changes in REM sleep following successful acquisition of cognitive procedural material have reportedly included increases in REM sleep time as well as increases in number of rapid eye movements (REMs) and REM densities. Animal studies have suggested that faster learning subjects show larger changes in these variables than slow learning subjects. The present study examined changes in REM sleep parameters in college students of varying I.Q. following training in two cognitive procedural tasks. It was predicted that there would be an increase in both number of REMs and REM densities following training. Further, it was predicted that High I.Q. individuals would exhibit more REMs than those in the Medium or Low I.Q. groups.

Method: Participants were 20 introductory college students (male and female) from Trent University. Subjects were screened for sleep problems and administered the MAB-II I.Q. scales. They were then assigned to one of three different I.Q. groups - High, Medium or Low. After an acclimatization night (night 1) in the sleep lab, subjects were given a second night of baseline sleep (night 2). On the evening of sleep night 3, subjects were asked to learn the Mirror Trace and Tower of Hanoi tasks. Subjects were all retested one week later to assess levels of learning. A fourth, non-learning control group was also included. Sleep stage scoring was done on all subjects. As well, all eye movements during REM sleep larger than 10 uv were also counted for these two nights.

Results: Learning. All test groups successfully learned the Mirror Trace task [$F(1,13) = 29.14$, $p < .0001$] and the Tower of Hanoi task [$F(1,13) = 49.58$, $p < .0000$].

Sleep States. The three test and non-learning control groups did not differ in terms of number of minutes of Stage 1, Stage 2, Stage ¾, REM sleep, %REM sleep or Total sleep.

Rapid Eye Movements. The total number of REMs was significantly larger for all groups on post training night 3 compared to baseline night 2 [$F(1,16) = 16.05$, $p < .001$]. There were no differences between test groups on this measure, although all test groups had significantly more REMs on post training night 3 than did the non-learning controls [$F(3,16) = 4.35$, $p < .02$]. The REM density measure also showed an increase from night 2 to night 3 [$F(1,16) = 22.69$, $p < .0002$]. The three test groups were superior to the non-trained control group [$F(3,16) = 4.46$, $p < .02$]. While the High I.Q. group did not differ initially from the other test groups (Low and Medium I.Q.) on this measure, there was a marginal difference ($p < .06$). The High I.Q. group had significantly higher densities than the control group on baseline night 2 ($p < .05$). On post training night 3, the High I.Q. group had higher REMs densities than all other groups ($p < .004$).

Conclusions: As predicted, the total number of REMs was increased in all groups (Low, Medium and High I.Q) following learning. Further, the REM densities were also increased in all test groups following task acquisition. The High I.Q. group had the largest increase in REM densities, with values significantly greater than the Medium and Low I.Q. groups as well as the controls. The Medium and Low I.Q. groups did not differ from each other in terms of REM densities. Results support the hypothesis that REM sleep, particularly the phasic component is involved with consolidation of cognitive procedural material.

Research supported by Canadian Institutes of Health Research (CIHR)

The Relationship Between Stage 2 Sleep Spindles and Intelligence

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Introduction: A number of studies have been done to examine the relationship between Stage 2 sleep and learning. One such study has reported that the number of sleep spindles was related to learning efficiency[1]. Expanding on this idea, we predicted that the number of spindles and the mean amount of sigma power during Stage 2 would be related to the subjects' intelligence scores on the MAB-II IQ test.

Methods: Ten subjects (range: 18 – 29) were used in the study. Subjects were screened for abnormal sleep patterns and excessive drinking/drug use. Subjects completed the MAB-II[2] IQ test and spent the subsequent two nights (acclimatization and baseline) in the laboratory. The number of sleep spindles (12-16Hz) and the mean Sigma Power (12-14Hz) was assessed for each page of Stage 2 sleep on the baseline night. Epochs with large body movements or major artifacts were excluded. Spindle activity and sigma power were assessed for both the C3 and C4 derivations.

Results: The total number of spindles (C3+C4) for the night was highly correlated with Performance IQ ($r = .71$, $p = .022$), and with Full Scale IQ ($r = .76$, $p = .010$), but not with Verbal IQ ($r = .56$, $p = .094$). The mean sigma power of both C3 and C4 for the entire night was highly correlated with Performance IQ ($r = .76$, $p = .011$) and Full Scale IQ ($r = .77$, $p = .009$). When the night was divided into thirds, the mean sigma power in the last third of the night showed the strongest correlation with both Performance IQ and Full Scale IQ ($r = .87$, $p = .001$, and $r = .84$, $p = .002$ respectively). None of the correlations with Verbal IQ were significant.

Sigma power was found to be the most highly correlated with two of the sub-tests of the Performance IQ scale: Picture Completion and Object Assembly. Both tasks require perceptual and analytical skills for successful completion.

A forward stepwise multiple regression was conducted with mean sigma for the second and last thirds of the night as the independent variables and Performance IQ as the dependent variable ($R^2 = .85$, $F_{2,7} = 20.06$, $p = .001$).

Conclusions: We suggest that sigma power is a powerful indicator of how an individual will perform on the Performance portion of an IQ test, particularly with respect to tasks that require perceptual and analytical skills. While a high level of sigma power is correlated with a high aptitude for these skills, the longitudinal nature of this relationship is not yet understood. Whether sigma power influences an individual's Performance IQ or whether the skills required affect the amount of sigma power a person exhibits is not yet clear.

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Research supported by Canadian Institutes of Health Research (CIHR)

Polysomnographic study of REM Sleep Behavior Disorder in Parkinson's Disease

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Introduction : REM sleep behavior disorder (RBD) is characterized by complex movements occurring specifically during REM sleep. Diagnostic criteria for RBD include excessive augmentation of chin electromyographic (EMG) tone and videotaped behavioral manifestations during REM sleep[1]. RBD has been found to be associated with neurodegenerative diseases called synucleinopathies, that include Parkinson's disease (PD), multiple system atrophy (MSA), progressive supranuclear palsy (PSP) and Lewy body dementia (LBD). In PD, the prevalence of RBD has been estimated to be around 15%[2], but this estimation is based on results obtained from a sleep questionnaire. Polysomnographic recordings would be more reliable in estimating the prevalence of RBD amongst PD patients. The present study is an attempt to better document RBD in PD, by using polysomnographic recordings.

Methods : Twenty-three PD patients (17 men and 6 women) were recorded for one night in a sleep laboratory. In addition to the EEG, EOG and chin EMG, the polysomnographic recording included infrared video monitoring to detect movements during REM sleep. The mean age was 65.29 ± 11.06 years. All the patients were kept on their usual treatment for PD (14 patients on levodopa ; 12 patients on dopamine agonists ; 7 patients on MAOI-B).

Results : Partial or complete loss of muscular atonia during REM sleep was observed in 52% (12/23) of PD patients. Simple or complex motor behaviors during REM sleep (talking, laughing, yelling, gesturing, reaching) were observed in 30% (7/23) of patients.

Discussion : Prevalence of RBD in PD patients appears to be higher in the present study than in studies based on a sleep questionnaire[2]. This suggests that polysomnographic recordings could help in identifying individuals with mild RBD that would remain undetected by questionnaires. Further studies comparing clinical criteria and polysomnographic recording in the same PD population would better answer this question. Longitudinal polysomnographic studies would help in clarifying whether or not those patients with partial atonia during REM sleep would eventually develop RBD.

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The Effects of Selective Sleep Deprivation on the Memory for a Declarative Learning Task

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Introduction: Declarative memory has not been as consistently linked to sleep as procedural memory, but there have been hints that a relationship exists. REM augmentation studies have found that some sleep parameters change after tasks with a declarative learning component (e.g., REM density[1], and percent REM sleep[2]). We hypothesized that memory for a purely declarative non-verbal learning task would be adversely affected by REM sleep deprivation.

Methods: 16 undergraduate Trent University students, who were screened for any sleep abnormalities, participated in the study. *The Declarative Memory Task*: fifteen diagrams of the human brain were presented on the computer, each highlighting a different area in the brain. A 60-item multiple-choice exam was given after learning; if a score of 80% was not achieved, a second version of the task was given. *Procedure:* Participants spent an acclimatization night in the sleep laboratory. On the second day, participants were taught the declarative task in the afternoon and were tested immediately for retention. Participants were told prior to learning the material that a score of 80% or more would earn them an extra twenty dollars (to create the motivation to learn). That night, participants were subjected to one of the following sleep conditions: REM deprived, Stage 2 interrupted, in-lab control, or home control.

Results: An ANCOVA was conducted to examine the effects of the sleep deprivation condition on re-test scores; the score on test one was the covariate. The groups did not differ in terms of their score on the re-test [$F(3,11) = 1.064$, $p = 0.404$, $MSE = 157.350$, $Eta^2 = 0.0967$].

The REM deprived group spent significantly less time in REM sleep than did the controls or the Stage 2 interrupted groups [$F(2,9) = 21.730$, $p = 0.000$]. The REM deprived group also spent significantly less time asleep than either the Stage 2 interrupted or the controls [$F(2,9) = 4.503$, $p = 0.044$].

Conclusions: The data from this study did not support the hypothesis that REM sleep is necessary for the effective consolidation of non-verbal declarative material. Neither REM deprivation nor Stage 2 interruption had a detrimental effect on memory for the declarative learning task. These findings differ from other studies that have found signs of a relationship between declarative learning and sleep. However, many of these previous studies actually required a procedural component for retrieval; participants were often directed to apply their knowledge, rather than using a rote recall method. Since procedural memory has been found to require REM sleep for efficient learning, it is not surprising that they found significant results. When both the learning and the recall methods are declarative, it appears that sleep deprivation does not negatively affect memory.

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Preoptic/Anterior Hypothalmic (POAH) Warming Suppresses Laryngeal Muscle Activity During Sleep

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Introduction: Reduction in upper airway dilator muscle activity during sleep is thought to be a key pathophysiological element of obstructive sleep apnea (OSA). Some evidence suggests that abnormal regulation of airway dilator muscle activity in OSA is coupled to a sleep regulatory abnormality. For example, sleep deprivation worsens OSA. POAH thermoregulatory processes were found previously to facilitate sleep. We hypothesize that POAH thermoregulatory processes may also modulate respiratory motor functions during sleep. We have previously reported that POAH warming can suppress diaphragmatic activity during sleep in the rat (McGinty et al, 1997) and laryngeal dilator activity in the cat (McGinty et al, 1998). The present report extends those preliminary data.

Methods: Gold-plated prong electrodes mounted on an acrylic base were placed chronically in the laryngeal dilator muscle, the posterior cricoarytenoid (PCA), and in the diaphragm of three cats. EEG, EOG and neck EMG electrodes, POAH thermocouples, and bilateral water perfused thermodes for local POAH warming were also implanted. The EMG from both muscles was recorded simultaneously during different states of vigilance (Awake, NREM and REM sleep). Trials of POAH warming of 0.4 to 1.2 degrees C. lasting 1-2 minutes were then carried out in each state. Integrated respiratory motor amplitudes were displayed and analyzed with the Spike 2 software (Cambridge Electronic Design) together with the other polygraphic signals. We compared successive diaphragm and PCA integrated signals from matched pre-warming and warming samples.

Results: PCA recordings showed discrete inspiratory bursts, closely coupled to diaphragmatic inspiratory bursts as reported previously (Orem and Lydic, (1978). PCA inspiratory burst onset preceded the diaphragmatic burst onset by about 0.31, 0.45, and 0.21 sec in Awake, NREM and REM, respectively. During REM sleep, PCA integrated amplitude declined markedly. During NREM sleep mild POAH warming induced small but consistent reductions in both diaphragm (range: 4.9%-7.2%) and PCA peak amplitudes (20.4%-36.2%, $p<.05$). POAH warming during NREM sleep also reduced integrated PCA signal amplitude (26%) and the PCA-diaphragm onset time difference (31%). POAH warming did not have consistent effects in REM, although responses were seen in some animals.

Conclusions: Our findings demonstrate that activation of POAH thermosensitive neurons can suppress airway dilator muscle as well as diaphragmatic muscle activity during NREM sleep. POAH warming had greater effects on airway dilator muscle activity than on diaphragm, which could increase airway collapsibility during inspiration. Weight gain and snoring in OSA patients often begins in early adulthood, suggesting a coupling between metabolic control and airway regulation. Elevated activation of POAH thermosensitive neurons could induce reduced metabolic rate, reduced airway dilator muscle activity, and increased sleepiness. Therefore, abnormally elevated activity of these neurons could play a role in the pathogenesis of OSA.

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Sleep and 24-hr body temperature in young women with ovulatory cycles and in women taking oral contraceptives

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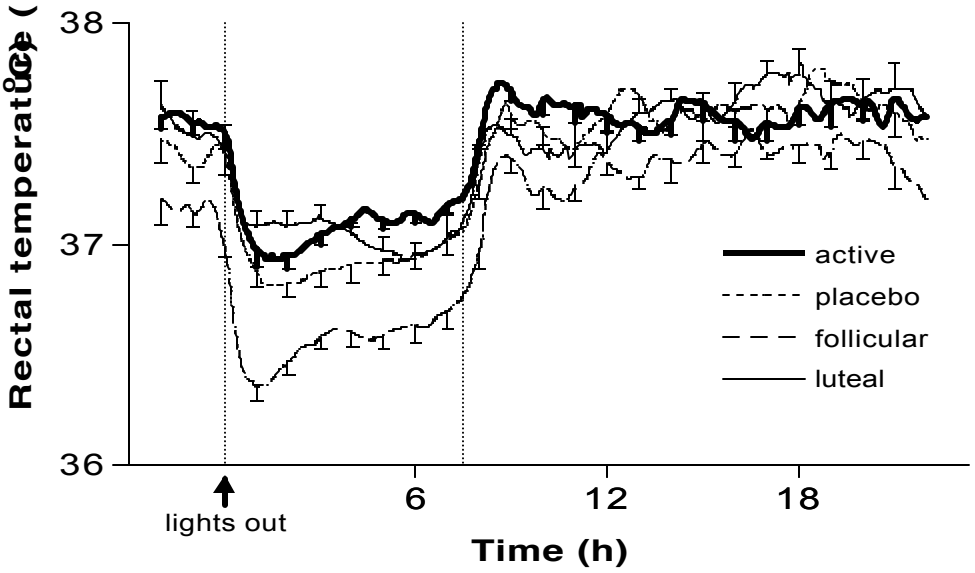
Cyclical changes in the female reproductive steroids across the menstrual cycle influence thermoregulation and sleep. Chronic administration of synthetic steroids, as occurs in women taking oral contraceptives, may affect these regulatory systems differently from endogenous oestrogen and progesterone. We recorded body temperature and sleep in nine young women with ovulatory cycles, in the mid-follicular and mid-luteal phases, and compared them to 10 women taking oral contraceptives, in the active and placebo phases of the contraceptive pack. Women in the luteal phase and women in the active phase of the oral contraceptive pack had raised 24-hr body temperatures compared to women in the follicular phase. The women taking oral contraceptives continued to have raised body temperatures in the placebo phase, indicating a prolonged action of synthetic reproductive steroids on body temperature.



Rectal temperatures (mean and S.E.M.) for 24 hours from two hours before lights-out. Vertical lines indicate average time in bed.

Endogenous and synthetic reproductive steroids influenced sleep independently of body temperature. Women taking oral contraceptives had more stage 2 non-rapid eye movement sleep in the active phase both compared to their placebo phase and the naturally-cycling women, whereas their slow wave sleep was reduced in both phases compared to the naturally-cycling women. Exogenous reproductive steroids therefore influence body temperature and sleep differently from endogenous progesterone and oestrogen.

Relations between objective and subjective measures: Cognitive performance and quality of sleep in elderly suffering of chronic insomnia

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Introduction: Relations between the objective and subjective quality of sleep and the objective and subjective cognitive performance are examined in 20 chronic users of benzodiazepines (BZD) and 20 drug-free elderly insomniacs. These measures are compared to measures obtained from 20 self-defined elderly good sleepers. The main hypothese are: 1) objectively, good quality of sleep is associated with better cognitive performance; 2) the subjective impression of good quality of sleep is associated with better cognitive performance; 3) the subjective impression of good performance is associated with better cognitive performance and, 4) the subjective impression of good quality of sleep is associated with the impression of good performance.

Methods: Participants are older than 55 years and all community-resident. Participants suffering of a sleep disorder other than insomnia are excluded. Furthermore, exclusion criteria also include suffering of a medical disorder interfering with sleep or a major psychopathology as well as using a medication disrupting sleep architecture. The insomnia duration (INS and INSBZ) was of 25,8 years (SD = 15,7), the duration of BZD use (INSBZ) was13,5 years (SD = 10,0) and the frequency of BZD use (INSBZ) was 6,6 nights/week (SD = 1,1). Objectively, sleep is assessed through polysomnographic recordings (PSG) while measures from the sleep diary reflect the subjective quality of sleep. Neuropsychological measures assessing attention, concentration, memory, executive functions and reaction time constitute the objective evaluation of performance. Participants also estimate the subjective perception of performance after the Neuropsychological testing.

Results: A correlational study reveals that cognitive performance and quality of sleep are associated, but differently among the three groups, for both subjective and objective measures.

Table 1 Objective measures of sleep and performance

	SOL	TWT	TST	SE
		Good Sleepers		
VVM		-0,48$_b$*		-0,45$_b$*
		-0,53$_a$*		-0,45$_a$*
PS	-0,48$_c$*			
AC				
EF	-0,79$_f$**			
		Insomniacs without medication		
VVM				
PS	0,69$_d$**			
AC	-0,58$_e$**			
EF				
		Benzodiazepine Users		
VVM	0,49$_a$*			
PS				
AC				
EF				

Note. VVM = Visual/verbal memory; PS = Psychomotor speed; AC = Attention/concentration; EF = Executive Functions; $_a$ Paired I. $_b$ Paired II. $_c$ Purdue. $_d$ Reaction Time $_e$ Digit span (backward) $_f$ Wisconsin;* $p < 0,05$. ** $p < 0,01$

There are positive relations between sleep quality and cognitive performance, for objective as well as subjective measures (r = .44 to r = .85; a = .05; Table 1). In that regards, insomniacs have more difficulty to accurately estimate their cognitive performance than good sleepers. On the other hand, BZD users and drug-free insomniacs appear more accurate in their estimations of sleep than good sleepers.

Discussion: Although high cortical arousal in insomniacs might contribute to their misperceptions about performance, it could also enhance their ability to perceive slight variations in sleep depth. Insomniacs may also spend more energy to maintain a good performance despite a poor sleep quality. Furthermore, benzodiazepine intake, even if chronic, might also alter performance perceptions.

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A Comparison between the Effects of Repeated Practice and prolonged wakefulness on Simulated Driving Performance

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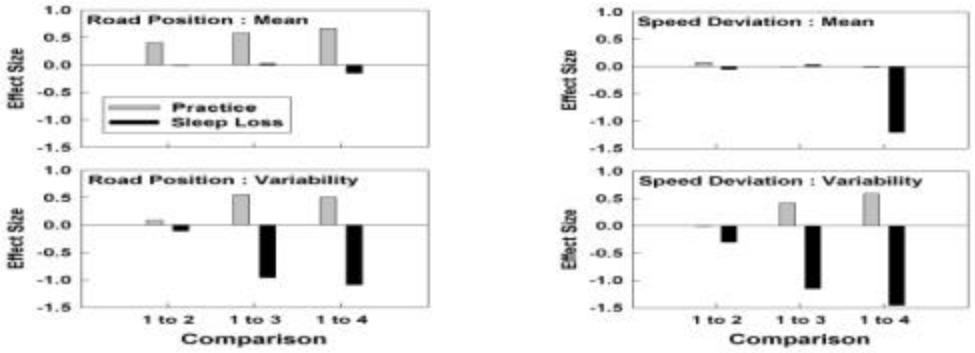
Introduction: An important issue to consider when examining the influence of sleep loss on any behavioural task is that performance may be influenced by practice or repeated exposure to that task. Improvements due to practice may mask decrements attributable to factors such as sleep loss. The objective of the present research is to examine the influence of repeated practice on simulated driving performance and to compare the size of these effects to those observed under conditions of prolonged wakefulness.

Method: Data for this comparison were obtained from two separate experiments investigating performance on the York Driving Simulator. In one study, 28 subjects (mean age = 24.8 years) completed four 20-minute daytime driving sessions over the course of a week in which they maintained their usual sleep-wake schedules. In the second, 32 subjects (mean age = 18.9 years) completed four 30-minute sessions at 2230, 0100, 0330, and 0600 during one night of prolonged wakefulness. In both studies, subjects were required to "drive" along a monotonous stretch of highway whilst staying as close to the posted speed limits and centre of their lane as possible.

Results: For both samples, performance on the following variables was examined: Mean Road Position, Mean Speed Deviation, Road Position Variability, and Speed Deviation Variability. Effect sizes (Glass' Delta) were calculated for comparisons between baseline and sessions 2, 3, and 4, respectively, within each sample. These effect sizes are presented in the figures below. In general, repeated practice improved driving performance notably within the first three sessions and had limited benefits thereafter. Sleep loss, on the other hand, clearly lead to a significant deterioration in performance that was maximal in the final session.



Conclusions: Improvements in performance due to repeated practice are of substantial concern - particularly in applied settings where appropriate experimental control is not readily available. In studies where performance deterioration is likely to occur, treatment effects are likely to be found but underestimated. Conversely, interventions aimed at improving performance or counteracting the detrimental effects of sleep loss may be overestimated. Researchers should either ensure that sufficient practice sessions are included or that appropriate designs are used to control the influence of practice effects.

Spectral analysis of REM sleep EEG in Asperger's Syndrome

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Introduction: Asperger's Syndrome (AS) is a Pervasive Developmental Disorder related to autism. Neuropsychological and brain imagery observation suggest that frontal functions and visuo-perceptual processes may be impaired in AS. Low visual counts of EEG spindle activity during stage 2 non-Rapid Eye Movement (REM) sleep have also been reported in patients with AS.[1] The present research sought to determine whether abnormal EEG is also present during REM sleep in AS using quantified EEG analysis.

Method: Six adult patients with AS (5M, 1F, 26.0 ± 5.6 years) were compared to six age- and gender-matched participants (5M, 1F, 26.8 ± 5.9 years) screened for psychiatric, neurologic and clinical sleep disorders. All participants were recorded for two consecutive nights in the sleep laboratory, using a 12-electrode montage. Power amplitude values (μV) of the night 2 Beta frequency band (13.75 – 20.0 Hz) from both groups of participants were compared using the Mann-Whitney *U*-test for independent samples.

Results: As patients showed a significantly lower absolute Beta over the primary (O_1, O_2) and associative (T_5, T_6) cortical visual areas.

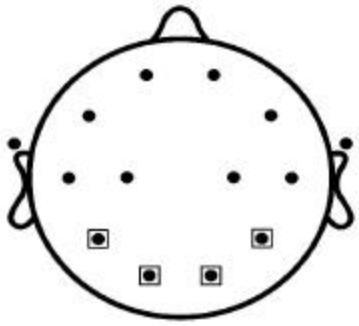

Figure 1. EEG montage with significantly different derivations between AS patients and control participants.

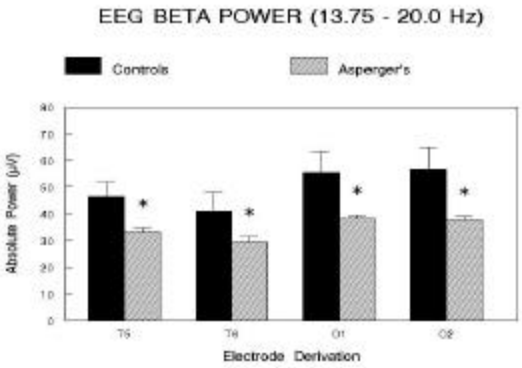

Figure 2. Bilateral absolute power amplitude for bilateral homologous electrode derivation, for significant placements.

Conclusions: These results support the hypothesis of an abnormal visuo-perceptual functioning in the autistic spectrum. Using brain imagery techniques, recent studies have shown decreased blood flow in the temporo-occipital region of high-functionning (i.e., normal IQ) patients with autism during face recognition tasks.[2] The present results extend these observations to REM sleep and may provide neurophysiological support for the poor quality of dream reports in autistic disorders.

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Dream content analysis in Asperger's Syndrome

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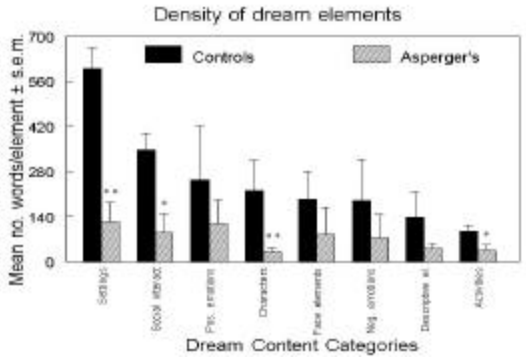
Introduction: Asperger's Syndrome (AS) is a Pervasive Developmental Disorder related to autism. Clinical observations and recent pilot study suggest that patients with autism and AS present a poor quality of dream reports. To better address the dreaming features of this disorder, we analyzed the dream content of patients with AS.

Method: Five adult patients with AS (5M, 1F, 20.6 ± 3.8 years) were diagnosed according to DSM IV criteria and using the Autism Diagnostic Interview. They were compared to five age- and gender-matched participants (5M, 1F, 20.6 ± 2.9 years) screened for psychiatric, neurologic and clinical sleep disorders. All participants were recorded for two consecutive nights in the sleep laboratory. On both nights patients were awakened during REM sleep and interviewed for dream content. Two independent judges analyzed one dream report from each participant using the coding system of Hall and Van de Castle. Number of words in dream reports, number of verbal interventions by the experimenter and dream content elements from both groups of participants were compared using Mann-Whitney *U*-tests for independent samples.

Results: Compared to controls, dream reports of patients with AS had fewer words despite an equal number of verbal interventions by the experimenter and included significantly less total number of objects (15.0±1.6 vs 5.4±2.8) and descriptive elements (10.0±2.8 vs 3.2±2.1) in their reports. When the number of words in dream reports were controlled for, patients with AS used less words than control participants to express most of scored dreamed elements.



Dream report dependant variables (mean number ± s.e.m.)

Variables	**Asperger's**	**Controls**	**p**
Words	240.0± 78.7	599 ± 65.2	<.01
EVIs	12.4 ± 3.1	15.8 ± 3.3	n.s.
Words following an EVI	19.7± 7.9	43.0 ± 7.0	<.05

EVI : Experimenters' Verbal Interventions

Conclusions: These results show that patients with AS are able to report dream contents following REM sleep awakenings in a laboratory settings but that such reports are less elaborate than that of control participants. Patients with AS produced a lower number of words to describe most of dream element categories with the exception of Objects. These results are in accordance with the problems related to the development of a Theory of Mind in autistic disorders spectrum.

Reference: Godbout R, Bergeron C, Stip E, Mottron L. A laboratory study of Sleep and Dreaming in a Case of Asperger's syndrome. Dreaming 1998; 8: 75-88.

Supported by "Fonds de la recherche en santé du Québec" and "Fondation de l'Hôpital L-H. Lafontaine".

Evening and morning gender differences in waking delta EEG activity in young adults

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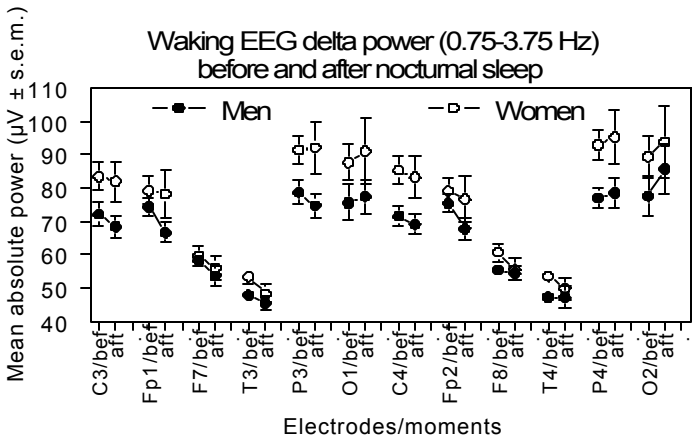
Introduction. Studies have reported that women tend to have more EEG delta power in non-REM sleep than men, while others have not.[1-3] Hypotheses proposed to explain these gender differences include possible differences in sleep regulatory mechanisms.[1] In the present study, we analyzed waking EEG activity in young adults prior to and following a night of sleep.

Methods. 13 healthy women (age: 21.5 ± 1.8 years) and 13 healthy men (age: 22.9 ± 3.9 years) spent two consecutive nights in a sleep laboratory. Night 1 served as an adaptation night and screening of sleep disorders. On night 2, waking EEG recordings lights on with eyes closed were performed for five minutes in the evening before going to bed (between 22h00 and 23h00) and on the following morning (between 07h00 and 08h00). A 12-electrode montage refered to linked ears was used: C3, C4, Fp1, Fp2, F7, F8, T3, T4, P3, P4, O1, O2. EEG amplitude power (μV/Hz, 0.75Hz to 19.75Hz) was determined with spectral analysis performed on 10 to 15 four-second artefact-free epochs. Frequency bands were created and the present report will be restricted to delta (0.75-3.75 Hz). Gender x Moment Anovas and LSD post-hoc tests were used and hemispheres were analyzed separately.

Results. The Anova showed a significant Gender effect on the evening and morning conditions in each hemisphere (Figure 1); Moment and interaction were not significant. Post hoc comparisons showed significantly ($p<.05$) higher delta values in women for Central and Parietal leads while Temporal and Frontal leads were not different; results for occipital leads were equivocal.



Conclusion. Our goal was to verify whether gender differences in waking delta EEG activity vary according to the moment of recording in young adults. The fact that women had higher delta power spectral values both in the evening and in the morning suggest that time, whether it is spent sleeping during the night or awakened during the day, does not interfere with this gender difference, and that gender differences previously shown for delta activity during NREM sleep[1-2] are also present at various moments of the waking state. Topographic distribution of the observed differences further substantiate the need for a better understanding of the underlying neural networks involved in EEG gender differences.

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Average Sunrise Time Predicts Depression Prevalence

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The striking differences in depression prevalence between nine European cities in the EURODEP geriatric depression Programme, or between five U.S. centres in the Epidemiologic Catchment Area (ECA) Study, lack satisfactory explanations to date. Given the links between light and depression, published prevalence data from these two studies were analysed to look for a relationship to sunrise time.

Depressive neurosis prevalences from the EURODEP Programme and one-year depression prevalences from the ECA Study were plotted against each centre's sunrise time, averaged over one year, and Pearson correlation coefficients calculated. For both studies, depression prevalences are highly negatively correlated with average sunrise time.

This suggests that a city's average sunrise time, determined primarily by east-west position within its time zone, may predict depression prevalence, and that simple public health measures such as going to Daylight Saving Time year-round or shifting time-zone boundaries, could reduce depression rates.

Cancer Fatigue and Sleep: Possible Role for Rising Time?

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Background: Fatigue is the symptom most frequently reported by cancer patients, and is often the most distressing. Given that fatigue and insomnia are closely linked not only in cancer but also in other illnesses, and that fatigue is frequently associated with depression, the connection between sleep patterns, fatigue, and depression was explored.

Method: A convenience sample of outpatients being followed by the Medical Oncology and Radiation Oncology clinics at the SMBD-Jewish General Hospital were asked to fill in a self-response questionnaire about their sleep habits, insomnia complaints, attitudes about sleep, fatigue, depression, cancer type and current treatment, and use of psychotropic medication.

Results: One hundred and twenty-five usable questionnaires were returned. Those receiving cancer treatment had a significantly higher average level of fatigue compared to those not undergoing treatment.
When respondents were divided into a low fatigue group (N=74) and a high fatigue group (N=49) using as a cutoff the mean fatigue score for the entire sample, the high fatigue group was significantly younger, more depressed, more insomniac, used sleeping pills more, arose later, and napped more than the low fatigue group. They were also significantly more likely to believe that "Sometimes it's necessary to miss work or school because of lack of sleep or really poor sleep."

Discussion: The intriguing finding that sleeping late is associated with higher levels of fatigue, can be explained by a variation of Michael Wiegand et al's "Depressiogenic sleep theory" that excessive REM sleep may induce depression. It is hypothesized here that cancer patients are more likely to attempt to sleep more than usual, either to escape painful feelings, or because of somnogenic effects ascribable to their illness or its treatment, or because of increased opportunity. Sleeping late results in large increases in REM sleep. If physiologic needs for REM sleep are exceeded, clinical depression may result in individuals who are genetically predisposed. Others may develop a subclinical form of depression, such as chronic fatigue. This hypothesis also suggests that treatment approaches such as earlier rising times or psychostimulants taken early in the morning may be helpful in cancer fatigue.

Fibromyalgia Symptoms and Sleep Patterns

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Background: Almost all fibromyalgia patients suffer from fatigue, and most also have sleep disturbances, which have been suggested as a possible cause for the fatigue. Insomnia, the sleep disturbance most often reported by fibromyalgia patients, has been hypothesized to result in some cases from attempts to extend one's sleep beyond physiological sleep needs.

Objective: To look for a possible link between the sleep habits and attitudes towards sleep of fibromyalgia sufferers and their symptoms of fatigue, pain, and depression.

Method: A questionnaire study was carried out with the members of a self-help organization for fibromyalgia sufferers. The questionnaire was given to the 156 members who came to an evening meeting; participants were asked to return the filled-in questionnaire as they were leaving. Respondents answered questions about their sleep habits, insomnia, attitudes about sleep, fatigue, pain, and demographics.

Results: Useable questionnaires were returned by 62 individuals; 60 (93%) were women. Mean age was 48.4 years. Fifty-two (84%) reported having received a medical diagnosis of fibromyalgia; 13 (21%) chronic fatigue syndrome, and 19 (31%) depression. Eighteen (29%) used sleep medication daily. Respondents spent on average 8.8 hours in bed, and got up at 7:57 am. For the entire sample, fatigue scores and pain scores were significantly correlated with insomnia scores, as was a single-item visual analog scale for self-rated depression. Fatigue was highly significantly correlated with pain score. Respondents' scores on the sleep attitudes questionnaire, where higher scores indicated increased importance attached to having a good sleep, also correlated significantly with pain and fatigue ratings.
For the 52 respondents who reported having been diagnosed with fibromyalgia, fatigue was significantly correlated with time in bed, and also with the amount of time spent in bed after 6 am (including naps) on workdays. Time in bed after 6 am was also found to correlate with self-rated depression score and with sleep attitudes score.
Daily users of sleep medication were significantly more likely to have fibromyalgia than those who never used sleep medication, and spent significantly more time in bed than never-users (9.6 hours vs 8.5 hours), Their self-rated depression scores were significantly correlated with rising time and with time in bed after 6 am. For the 41 respondents (66%) who take naps, time in bed correlated significantly with fatigue and with insomnia.

Conclusions: This questionnaire study supports a link between sleep patterns and fibromyalgia symptoms: more time in bed and later rising times are associated with increasing severity of fatigue, pain, and insomnia. Moreover, the more importance that is attached to getting sufficient amounts of quality sleep, including sleeping late or missing work or school, the greater the pain and fatigue experienced. The author discusses the possible role of late rising in bringing about both insomnia as well as excessive Rapid Eye Movement (REM) sleep. As excessive REM sleep is hypothesized to cause depression, it is possible that it may also bring about some fibromyalgia symptoms. This suggests that early rising may be helpful in treating fibromyalgia.

Hori's 9 Sleep Onset Stages: FFT and Coherence Analyses

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Introduction: The purpose of this study was to perform a detailed examination of changes in EEG coherence and power spectral (FFT) analysis during the sleep onset period (SOP). Others[1, 2] have examined coherence and FFT in a broader fashion—for example, as a function of standard R & K stages. However, there are many distinct changes in EEG from waking through the onset of sleep, ending when stage 2 is established.[2, 3] Many of these systematic processes are conflated when R & K stages are used. Therefore, in order to provide a much finer analysis of the SOP, a slightly modified version of Hori's 9-stage system[3] was used. These nine stages correspond to the standard stages of awake to stage 2.

Method: Participants consisted of 9 individuals with mild traumatic brain injury (MTBI) and 9 matched controls with mean ages of $\underline{M}$ = 21.4 ($\underline{SD}$ = 2.4) and $\underline{M}$ = 20.7 ($\underline{SD}$ = 2.1), respectively. Participants spent 3 nights in the lab, the first two serving as adaptation nights. On the third night, participants were allowed to sleep without interruptions, and PSG data were recorded. *Analysis:* Frequencies bins used were: delta, theta, alpha-1, alpha-2, alpha-3, sigma, beta-1, and beta-2. Interhemispheric coherence channel pairs used were: C3-C4, O1-O2, P3-P4, T3-T4, T5-T6, F3-F4, F7-F8, and Fp1-Fp2. And sites for FFT analysis were Cz, Pz, O2, and T4.

Results: *Between Subjects Differences:* FFT and coherence values between the MTBI and control groups were very similar, and the groups were combined to increase sample size. *FFT Results*: General trends showed that as subjects progress from stage H2 to H9, delta and theta increase; alpha-1 and alpha-2 decrease; alpha-3 initially decreases, and then increases at H5; sigma increases; and beta-1 and beta-2 decrease. *Coherence Analysis:* Generally, coherence increased from H2 to H9. There was a significant decreased across a number of channels and frequencies between H3 and H5, followed by a significant increase between H5 and H6.

Conclusion: The FFT results were the same as previous studies[2, 3]; the slower frequencies increased and the faster frequencies decreased as participants progressed from awake to H9 (R & K stage 2). Coherence generally increased from awake to H9, which was consistent with other studies[1]. There was however a reliable decrease in coherence between H3 and H5, which corresponds to the elimination of alpha, and the establishment of theta as the predominant EEG frequency. This was immediately followed by a moderate increase in coherence from H5 to H6—which marks the appearance of vertex waves. These findings may indicate that coherence—as a measure of information processing—reaches a minima with the elimination of alpha (and concomitant increase in theta), and that vertex waves may signal the beginnings of synchronization associated with deeper sleep.

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Music, Tones and Sleep: An EEG Analysis

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Introduction: A number of researchers have reported that auditory stimulation can influence physiology[1], and a number of therapists report using music to help their patients relax, or to induce sleep[2]. There are currently a number of musical selections on the market that claim to help people sleep. Thompson[3] has created a commercially available CD that purports to "improve sleep by entraining EEG rhythms in the delta frequency." Since it is possible to entrain EEG using visual stimulation—as in the phenomena of photic driving—could it also be possible to entrain EEG in the delta frequency via auditory stimulation? And if so, would this improve sleep. This study used both objective and subjective measures to compare sleep parameters between the music of the Delta Sleep System, with a control condition and a tones condition. Sleep onset latency, sleep efficiency, and WASO were used as indicators of sleep quality, and percent SWS (stage 3 + 4) and power spectral analysis were used as indicators of the amount of delta activity. It was hypothesized that sleep quality and the amount of delta activity would be greatest in the music condition.

Method: 10 female students—with no sleep difficulties—between the ages of 17 and 24 ($\underline{M}$ = 19.90, $\underline{SD}$ = 1.91) participated. Each completed the Pittsburgh Sleep Quality Index, Personality Assessment Inventory, and a weekly sleep log prior to PSG recordings. Subjects spent 4 consecutive nights in the lab. The first served as an adaptation night, and the other nights had music, tones, and the control condition in counterbalanced order. The "music" was derived from the first of 2 CD's of the Delta Sleep System, and 300 Hz tones were 1 sec in duration, with 1.5 sec between tones. Both stimuli commenced at "lights off" and were discontinued 5 min after the first spindle or K-complex.

Results: The results indicate that there was no significant difference in any of the objective sleep measures between the music, tones, and control conditions. That is, sleep onset latency ($\underline{F}(2,18) = 1.15$, $\underline{p} = .339$), sleep efficiency ($\underline{F}(2,18) = .59$, $\underline{p} = .547$), WASO ($\underline{F}(2,18) = .10$, $\underline{p} = .800$), and percent Stage 3 and 4 combined ($\underline{F}(2,18) = .20$, $\underline{p} = .780$) were all non-significant. Also, 95 of 96 power spectral analyses were not significant. However, for the subjective measures, participants rated the music as being more soothing ($\underline{t} = 4.27$, $\underline{p} = .002$), comforting ($\underline{t} = 2.77$, $\underline{p} = .022$), pleasant ($\underline{t} = 4.59$, $\underline{p} = .001$), and relaxing ($\underline{t} = 3.132$, $\underline{p} = .012$) than the tones.

Conclusion: While participants found the music more enjoyable to listen to than the tones, there was no evidence that the music had any influence on subjects' neurophysiology during sleep compared to either the control or tones condition. That is, when listening to the music, participants did not sleep better, nor did they show any increases in delta activity. It appears that in a sample of normal sleepers, the claims of the manufacturer are unsubstantiated.

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Psychophysiological Analysis of the Sleep Onset Period:
The Dream Properties Scale and Alpha wave EEG

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Introduction: Sleep onset (SO), or the process of falling asleep, is generally defined as the transition from waking EEG to sleeping EEG (Wake to Stage 2), based on physiological behavioural and psychological changes. This process, however, is not well understood, and the present study was aimed at describing microscale changes within this period. Specifically, Dream Property Scale (DPS) measures of hypnagogic imagery (HI) and FFT changes during the sleep onset period (SOP) were examined. The methods for scoring sleep into discrete portions have remained largely unaltered since Rechtschaffen and Kales' (1968) methods became ubiquitous. It is probable that more fleeting changes in EEG can be examined, as per the 9 stage system developed by Hori. It was hypothesized in the present study that Bizarreness ratings of HI, as measured by the DPS, would increase as a function of Hori Sleep Onset stages (II<III<V). It was also hypothesized that the right hemisphere would show greater alpha power than the left when subjected to FFT analysis.

Method: The sample was composed of freshman students, 2 males and 9 females (ages 18 to 30 years ($\underline{M}$ = 21.09, $\underline{SD}$=3.62)), who did not have any sleep disorders and who were right handed. Two nights were spent in the laboratory. The participants were awoken three times at sleep onset, at Hori Sleep Onset stages II, III and V, to assess SO mentation, using the Dream Properties Scale. FFT analysis was performed on Fp1, Fp2, F7, F8, FZ, C3, C4, CZ, T5, T6, 01, 02 to examine laterality shifts in alpha wave power.

Results: Bizarreness ratings increased at HSO V, though statistically non-significantly. FFT analysis showed no differences between the hemispheres in alpha band power, and no significant changes in alpha power from HSO stage II to III to V.

Conclusion: The current study explored brain wave and cognitive differences at Sleep Onset, the convergence of which represents an area that is lacking in scientific research. The present study validates the use of the DPS for HI studies, and the use of HSO stages for scoring sleep onset, pointing the way for future studies with similar foci. DPS findings indicate that the Bizarreness subscale of the DPS is particularly sensitive to the disappearance of alpha during SOP, as was expected in the hypotheses, making it particularly useful for analyzing dream-like imagery. Alpha frequency EEG appears to parallel processes that regulate thought such that when it disappears; bizarre imagery begins to occur. The DPS provides a tool with high validity for examining SO imagery, because it removes variability encountered when examining mental imagery at SO; objectively providing assessments of HI, and allowing for statistical analyses. Language problems are eliminated by the use of DPS for analyzing dream reports, in that participants may not be able to explain themselves accurately, or researchers may interpret the prose of the dream report based on their own understanding of the English language. Further studies might expand upon the DPS, specifically tailoring it for visual imagery by increasing the number of items sensitive to visual hypnagogic imagery.

The Effects Of Simple Motor Procedural Learning On Sleep Spindle Activity

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A number of recent studies have suggested that simple motor procedural tasks are Stage 2 sleep dependent. Simple motor procedural learning involves habituating motor plans or procedures to perform tasks that have very simple cognitive attributes. It has been found that Stage 2 sleep deprivation is detrimental to memory for the pursuit rotor, a simple motor procedural task (1, 2). Another study found that the number of spindles in Stage 2 sleep is positively correlated with procedural learning (3). Other studies have investigated the relationship between number of spindles in Stage 2 sleep and intelligence (IQ) (4). This last mentioned study found that the number of spindles was related to performance IQ and full scale IQ, but not to verbal IQ. It was hypothesized that number of spindles would increase as a result of new motor procedural learning. It was also expected that performance IQ would be related to the number of baseline Stage 2 spindles.

Method: Five undergraduate female students were asked to spend an acclimatization night and a baseline recording night in the sleep lab and have their IQ tested using the MAB-II (5). Following this they were assigned to either the control or experimental group. The control group (N=2) was allowed to have a normal night of sleep. The experimental group (N=3) was also allowed to have a full night of sleep, but they participated in four simple motor procedural tasks for the duration of approximately 1h 45min before sleep. These participants were retested on the tasks one-week after learning. The tasks included the pursuit rotor, a simple tracing task, the ball and cup game, and the operation game that all subjects were able to learn.

Results: Sleep Data: It was found that the number of baseline Stage 2 spindles increased significantly after simple motor procedural learning ($F_{(1,3)}=11.65$, $p<.05$). On the other hand, no significant differences were found between baseline and test nights for either group in the duration of stages 2, 3/4, or REM sleep.

IQ Data: A strong positive relationship between Performance IQ and the number of baseline spindles approached significance ($r=.83$, $p<.10$). No relationship was found between verbal IQ or full scale IQ and the number of spindles ($r=.41$, $r=.62$ respectively).

Conclusions: These results indicate that after exposure to simple motor procedural learning, spindles during Stage 2 increase. It is probable that this activity is involved in the consolidation of simple motor procedural memory. The relationship between baseline spindles and IQ indicate that they are also related to aptitude for learning. These results suggest that simple motor procedural memory processing is a distinct subtype of procedural memory that involves widespread activation of thalamocortical loops involved in the production of spindles.

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The effects of 25-hour sleep deprivation in middle-aged men and women

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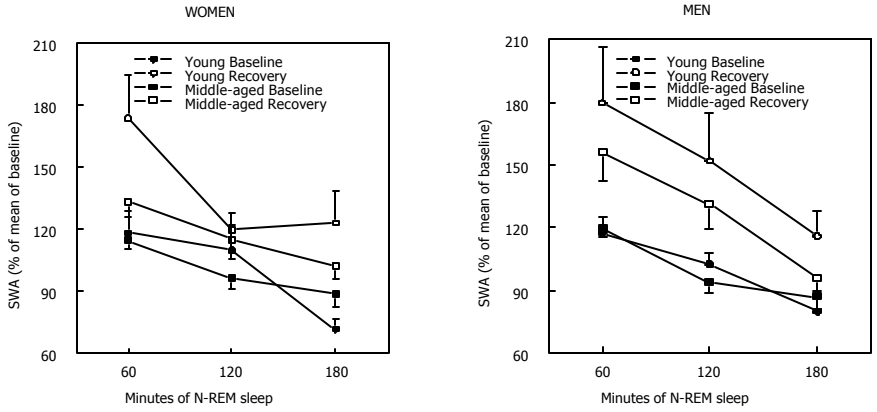
Compared to men, middle-aged women report more sleep and fatigue complaints. However, when studied in the sleep laboratory, men show less SWS and lower EEG slow-wave activity (SWA: spectral power between 0.5 and 4.5 in N-REM) than women. These results led to the hypothesis that age might have differential effects in men and women during the middle years of life. Challenges to the sleep-wake cycle help to understand the mechanisms underlying gender and age differences. The aim of this study was to evaluate gender differences in the effects of a 25 hours of sleep deprivation on daytime recovery sleep in young and middle-aged subjects.

Subjects: Thirty-three subjects were studied. They were separated into 2 groups: Young: (20-39 years, 8 women, 8 men) and Middle-aged (40-59 years, 8 women:, 9 men). All subjects came to the laboratory for 4 consecutive nights and 2 days. Baseline sleep was recorded on the 3rd night. The morning after, subjects entered a mini-constant routine during which they were kept awake in bed for the next 25 hours. The sleep recuperative episode started the morning following the sleep deprivation. Factorial ANOVAs with between group factors (age, gender) and repeated factors (sleep episode, cycle) were used to compare the effects of the sleep deprivation.

Results: Both age groups showed a decrease of sleep efficiency during daytime recovery sleep, but the Middle-aged had a more abrupt decline than the Young (interaction: $p<0.02$). No interaction was found between age group and gender or between age group, gender, and night. Slow-wave sleep was potentiated in both age groups following sleep deprivation. However, the rebound of SWS was less pronounced in the Middle-aged group compared to the Young (Interaction: $p<0.03$). No interaction with gender was found. The Figures illustrate hourly mean SWA (and sem) for the first 180 minutes of N-REM sleep separately for women and men. The rebound of SWA following the sleep deprivation was less pronounced in the Middle-aged compared to the Young (interaction: $p<0.05$). No interaction with gender was found.

Discussion: These results suggest that middle-aged men and women do not differ in their response to sleep deprivation during daytime recuperative sleep. Compared to the young, middle-aged subjects clearly show a higher vulnerability to an abnormal phase angle between sleep and the circadian signal. The observed reduction of SWA following sleep deprivation in the middle-aged subjects suggests that the homeostatic recuperative response is already attenuated in the middle years of life and that this attenuation is similar in both genders.



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Intra-Amygdala Scopolamine Infusions Before and After a Paradoxical Sleep Window: The Effects on Conditioned Cue Preference Acquisition

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There is now substantial evidence that paradoxical sleep (PS) is involved with memory processing in animals (1,2) and in humans (3). In rats, paradoxical sleep windows (PSWs) have been identified for several tasks. PSWs are short periods of time following task acquisition in which PS levels are increased above normal. PS deprivation during PSWs results in memory impairments for the previously acquired task (1,2). Although memory processing is believed to occur during PSWs, very little is known about the neural systems involved. Recently, a PSW 9 to 12 hours after conditioned cue preference (CCP) acquisition has been identified (4). It has also been demonstrated that an intact lateral amygdala is required for CCP acquisition (5). In the present study, scopolamine was used to suppress acetlycholinergic activity in the lateral amygdala in order to examine the involvement of the lateral amygdala in a PSW for the CCP task.

Method: Cannulae were implanted bilaterally into the lateral amygdalae of Sprague - Dawley rats (n=15). The rats were then trained on the CCP task for 8 consecutive days (4 days in dark arm/ 4 days in light arm paired with food reward). Experimental rats were given bilateral intra-amygdala infusions of scopolamine either 9 hours (to be active during the PSW, n=4), or 12 hours (to be active just after the PSW, n=4) after each training session. Control rats were given bilateral intra-amygdala infusions of scopolamine either 9 hours (n=4) or 12 hours (n=3) after each training session. Following training, rats were given one testing session on the CCP task. Time spent in the light arm, dark arm, and centre platform were recorded. The number of correct responses (light arm entries) and incorrect responses (dark arm entries) were also recorded.

Results: During testing, rats that received scopolamine infusions either 9 or 12 hours after CCP training spent significantly less time in the light arm ($p<.05$) and significantly more time in the dark arm and centre platform ($p<.05$) than the rats that received saline infusions 9 hours after training. Rats that received scopolamine infusions 12 hours after training made significantly fewer correct responses during testing than the rats that received saline infusions 9 hours after training ($p<.05$). No other differences were significant.

Conclusions: Bilateral infusions of scopolamine administered either 9 or 12 hours after training appeared to disrupt CCP acquisition. Scopolamine was able to impair memory when infused in the PSW and in the hours just following the PSW. This suggests that the lateral amygdala may be processing previously acquired material several hours after training. In future, groups getting Scopolamine infusions at 5 to 8 hours and 17 to 20 hours after training should clarify the time during which the amygdala is having the effect on memory.

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Effects of a 25-hour sleep deprivation on subjective alertness and waking EEG in young and middle-aged subjects

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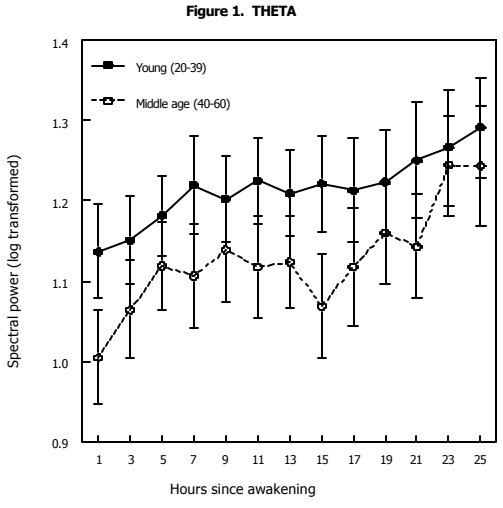
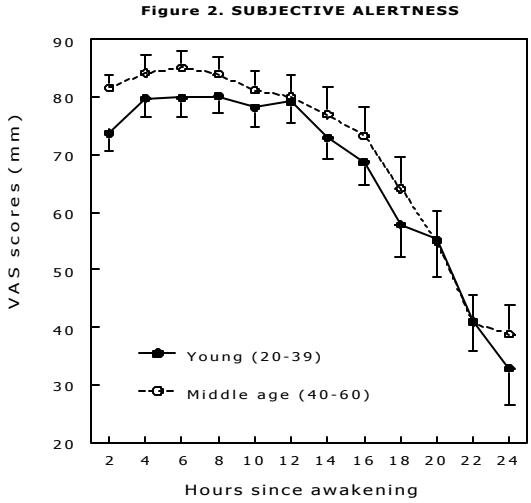
The wake-dependent modifications of the sleep EEG and of the spectral power in the alpha and theta ranges of the waking EEG may represent the same underlying homeostatic process. Compared to the young, middle-aged subjects show a reduced rebound of slow-wave activity (SWA: spectral power 0.5 Hz to 4.5 Hz in N-REM sleep) following an acute sleep deprivation, suggesting an attenuation of homeostatic drive in this population. The aim of this study is to evaluate the effects of a 25-hour sleep deprivation on quantitative waking EEG and subjective alertness (SA) in young and middle-aged subjects. We anticipate middle-aged subjects will show reduced wake-dependent modifications of these parameters.

Twenty-six subjects: 13 Young (20-39 years, 6 W,7 M) and 13 Middle-aged (40-59 years, 7 W, 6 M) spent four consecutive nights in the laboratory. On the morning following the third night, subjects entered a 25 hours mini-constant routine. Waking EEGs with eyes open were recorded every two hours and SA was evaluated every 30 minutes by visual analog scales. Waking EEGs (C3-A2; sampling rate: 256 Hz) were subjected to spectral power analysis (FFTs) for consecutive 2-sec epochs and a resolution of 0.5 Hz. Averages were computed on artifact-free EEG signals (C3-A2). Two-way factor ANOVAs with one repeated measure (Time) followed by trend analyses have been used on log transformed data.

Theta and alpha power showed a significant time effect ($p<0.0001$) and a significant linear trend ($p<0.0001$), increasing with time awake (no significant Group effect nor interaction between Group and Time; Figure 1). SA showed a significant time effect ($p<0.0001$) and a significant linear trend ($p<0.0001$), decreasing with time awake (no significant Group effect nor interaction between Group and Time; Figure 2).





Both groups showed similar time courses of SA and theta and alpha power during a 25-hour sleep deprivation. The build up of homeostatic pressure seems to have no differential impact on the waking EEG in the two age groups. These results are in contrast with the sleep EEG data showing a reduced rebound of SWA in the middle-aged population after a sleep deprivation. The functional relationship between wake-dependent modifications in the waking EEG and the sleep EEG is still unclear and deserves further investigation.

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Assessment of a Simulated Driving Task For Sleep Research

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Introduction: Sleep scientists commonly use neurobehavioral tasks to examine performance deficits induced by sleep loss. Simulated driving tasks are "face valid" performance tasks that require some of the constituent skills necessary for actual driving. As with any neurobehavioral task, however, the importance of establishing the psychometric properties of such instruments before incorporating them into experimental protocols cannot be overestimated. Therefore, as our group begins studies of simulated driving in adolescents, we first sought to determine the number of practice trials necessary to learn a driving task (YDS, Kingston, Ontario, Canada).

Methods: Seven adolescents (mean age= 13.8 years, sd=1.6, 2 girls) and two adults (ages 20.3 and 24.8 years, both females) maintained an 8-hour sleep schedule for two nights before participating, and refrained from caffeine, alcohol, and illicit drugs for at least 24 hours. Testing occurred in a 3-hour block between 0900 and 1800 hours on a driving simulator. The driving task appears as a two-lane highway during the daytime, with lane markings, signs, and occasionally, other simulated vehicles. Participants drove nine 10-minute sessions with 5-minute breaks between sessions. One participant drove eight sessions. Participants were instructed to maintain the simulated car's position in the middle of the right-hand lane, to obey speed signs, and to keep both hands on the steering wheel, while operating the pedals with the right foot only. Dependent measures on the driving task included mean and standard deviation of lane position (mean tracking and tracking variability), mean speed deviation and speed variability, and off-road incidents.

Results: Repeated measures ANOVA with eight participants indicated that performance on four driving measures remained constant throughout nine trials, while tracking variability increased significantly [$F(8,56) = 3.59$, $p<.05$]. This small decline in performance was notable only after trial five. Slopes for each driving measure were calculated for all nine participants separately to examine individual differences in practice that might be obscured by group data. Table 1 shows that mean slopes for four measures — mean tracking, tracking variability, speed deviation, and speed variability — were very close to zero. The higher mean slope for off-road incidents was due primarily to four participants whose driving was more erratic during trials five to nine.

Table 1. Slopes for dependent measures on the driving task.

Driving Measure	Slope		
	Mean	SD	Range
Mean tracking	-.006	.036	-.05 to .05
Tracking variability	.037	.050	-.02 to .13
Speed deviation	.009	.157	-.25 to .29
Speed variability	.029	.059	-.03 to .17
Off-road incidents	.184	.238	.00 to .57

Conclusions: Even for nondrivers, practice effects on the driving simulator in healthy, well-rested individuals are negligible, and the task appears to be rapidly "overlearned." Visual inspection of individual data, corroborated by participants' self-reports, indicates both rapid acquisition of the task and appearance of performance-affecting fatigue and boredom with repeated testing. Taken together, these findings indicate that little pre-study practice is necessary on this neurobehavioral task and that investigators may consider limiting session length to 60 minutes or less to avoid fatigue and boredom with the task.

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Spatial orientation in pups born from mothers who were chronically deprived of paradoxical sleep during pregnancy : a pilot study

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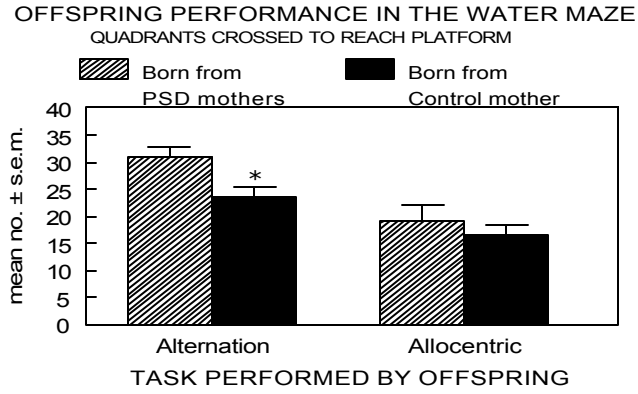
Introduction: One of the early functions of Paradoxical Sleep (PS) is to contribute to brain maturation. The first 10 days of pregnancy in the rat are known as the organogenesis period. In the present study we used chronic partial PS deprivation (PSD) during the organogenesis period of pregnancy in order to verify its effect in spatial performance of the offspring.

Methods: Twenty-two rats born from three mothers were studied. Fourteen rats (7 M, 7 F) were born from two mothers deprived of PS using the flower pot technique. Eight rats (4 M, 4 F) were born from a control mother submitted to an equally stressful procedure but without the severe deprivation effects on PS (large platform). Mothers were exposed to their respective platform for four hours a day, starting at the beginning of the light period (8h00), after which they were returned to their individual home cage. Half he offspring was tested from postnatal day 21 to day 24 in either one of two versions of a water maze.[1] In the standard version ("Morris-type", allocentric), rats had to rely on external cues to find the hidden platform while departing from a different quadrant of the pool on every trial. In the alternation version, the hidden platform alternated between two quadrants while rats always started from the same quadrant. The rats had 4 trials of 40 seconds each to locate the platform where they remained for 30 seconds. If they did not succeed in time, they were placed on the platform by the experimenter where they stayed for 30 seconds. Time latency and number of quadrants entered before finding the platform were measured for each trial; results on postnatal day 24 were compared using Mann-Whitney U-tests.

Results: Rats from PSD mothers crossed more quadrants than controls to reach the platform (30.8 ± 1.9 vs 23.7 ± 1.8, $p<.05$). The time spent finding the platform was similar in the two groups (141.0 sec. ± 9.5 and 143.3 sec. ± 7.9, respectively). There were no differences between the two groups in the allocentric water maze (19.0 ± 3.2 vs 16.5 ± 3.6 quadrants and 90.2 ± 11.9 vs 91.5 ± 24.3 sec., respectively).



Discussion: This learning deficit in offspring of PSD pregnant females is comparable to what was described following acute short-term PSD in adult rats.[1] Since the alternation water maze task used in both studies was shown to rely on the integrity of the prefrontal cortex,[2] we propose that repeated interference of PS during the synaptogenesis period of pregnancy is associated with learning difficulties in task that require an intact prefrontal cortex network. The accumulation of a PS deficit in the pregnant mother upon chronic partial PSD may be a determining factor.[3]

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Spectral analysis of REM sleep in an animal model of Alzheimer's disease

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Quantified EEG analysis during REM sleep in patients with Alzheimer's Disease (AD) show a shift toward slow frequencies and this effect is particularly apparent for theta activity over the temporal cortex.[1] Cholinergic deficits constitute prominent features of AD and this has led to animal models, including the acute effects of scopolamine, a non-selective muscarinic receptor antagonist, on cognitive performance in the rat. The aim of the present research was to evaluate the acute effects of scopolamine on REM sleep quantified EEG measures in the rat. Particular attention was paid to Rho activity (20-30 Hz), a cortical EEG-frequency thought to be selectively associated to REM sleep in the rat.[2]

Methods. Twenty-three Long-Evans rats aged three to four months were implanted for sleep recording under pentobarbital anesthesia; EEG electrodes were placed over frontal, centro-medial, and centro-lateral cortices; a reference electrode was placed over the cerebellum. After at least 7 days of recovery, rats were distributed in either one of two treatment groups: scopolamine (01.mg/kg s.c.; nine rats) or vehicle (0.5 c.c. NaCl 0.9%; 14 rats). Rats were injected at 08h00 (two hours after onset of the light period) and sleep recording was started immediately, for four hours. Sleep stages were determined visually in 10 sec epochs. Fifteen four-seconds epochs (60 sec) of artefact-free EEG were selected from REM sleep and submitted to Fast Fourier Transform with a cosine window smoothing and a resolution of 0.25 Hz. Power amplitude was calculated and six frequency bands were extracted: Delta (0.75-3.75), Theta-1 (4-6.75), Theta-2 (7-9.75), Sigma (10-13.75), Beta (14-19.75), and Rho (20-30). Relative activities were computed ([band power/total power] * 100) and results were compared using *t*-tests for independent samples.

Effect of scopolamine on quantified EEG

(mean ± s.e.m.)

	Saline	Scopolamine	p*
Frontal Delta	17.2 ± 0.3	18.7 ± 0.4	.01
Frontal Beta	14.9 ± 0.1	16.5 ± 0.7	.03
Frontal Rho	21.6 ± 0.7	17.1 ± 0.8	.0006
Centro-lateral Sigma	11.3 ± 0.4	13.0 ± 0.8	.06
Centro-lateral Rho	23.7 ± 1.0	19.5 ± 1.1	.02
Centro-medial Sigma	11.6 ± 0.1	12.9 ± 0.6	.03
Centro-medial Beta	12.9 ± 0.2	14.9 ± 0.4	.0005

** t- test for independent samples*

Results. Scopolamine generally increased EEG activity in all frequency bands at the three electrode sites with the exception of Rho activity which was decreased. The most salient results are presented in table 1.

Conclusion. The present observation that scopolamine increases EEG activity replicates previous reports on conventional frequency bands. The fact that Rho activity was decreased may point to particular neurophysiological networks involved in the rat model of AD.

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Analysis of the first night effect on evening and morning waking quantified EEG

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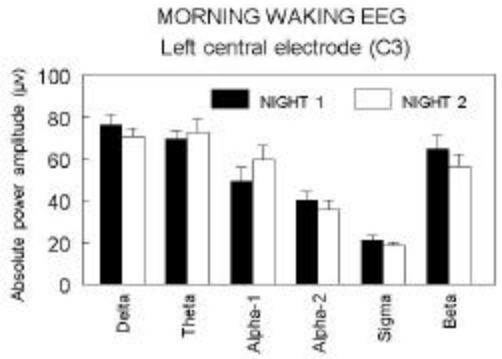
Introduction: The detrimental effect of having to sleep for a first night in a laboratory is a well-known phenomenon. The aim of the present study was to verify whether the first-night effect had an impact on waking quantified EEG measures taken in the morning.

Methods: Eight healthy participants (4 men, 4 women, aged 26.9 ± 4.9 years) were recorded for two consecutive nights in a sleep laboratory. All were free from sleep disorders and from a personal or a familial (first degree) history of psychiatric or neurologic disorders. Subjects were asked to keep a regular sleep-wake schedule for 14 days before coming to the laboratory. Napping was not allowed on days prior to recordings. Both nights were scored according to Rechtschaffen & Kales (1968). EEG recordings were obtained in the evening (between 22h00 and 23h00) and in the morning (between 07h00 and 08h00). Subjects were recorded for five minutes with eyes closed. Fifteen 4-second artefact free epochs from the C3 electrode (referred to linked earlobes) were selected and submitted to Fast Fourier Transform with a resolution of 0.25 Hz and a cosine window smoothing. Absolute power amplitude was extracted (μV/Hz, 0.75Hz to 19.75Hz) and six frequency bands were created: Delta (0.75-3.75 Hz), Theta (4.00-7.75 Hz), Alpha 1 (8.00-10.00 Hz), Alpha 2 (10.25-12.75Hz), Sigma (12.00-14.00) and Beta (13.00-19.75 Hz). Data is expressed as means ± SE.M. Statistical comparisons were made using a 2 x 2 ANOVA.

Results: Sleep architecture showed a typical first night effect (see Table 1). EEG spectral analysis however did not reveal any significant differences (see Figure 1).

Table 1. First night effect on sleep parameters

	Night 1	Night 2	P
Sleep onset latency	17.5±3.5	12.6±2.2	.08
SWS latency	29.0±6.8	13.9±2.6	.06
REM sleep latency	124.0±18.6	114.8±16.9	ns
% Stage 1	15.2±2.2	13.2±2.0	ns
% Stage 2	57.1±2.8	51.7±2.5	ns
% Stage 3	6.4±0.6	10.2±1.0	.01
% Stage 4	7.0±1.5	6.4±1.3	ns
% REM sleep	14.3±1.4	18.4±1.5	.06
REM period	4.3±0.3	4.4±0.4	ns
Wake (min)	36.1±9.7	20.8±14.1	ns
% Sleep efficiency	92.1±2.1	15.8±0.8	ns
Total sleep time	444.5±13.7	472.6±5.2	.05



Conclusion: These results show that the minimal sleep disturbances associated with the first-night effect in young healthy participants has no impact on the following morning waking EEG activity in recorded with central electrodes. We have reported comparable results in a different set of subjects using quantified EEG measures obtained during REM sleep.[1] The fact that frontal and temporal electrodes show an overnight effect on waking EEG activity following a normal night of sleep suggests that different neural networks have different sensitivity to the effect of sleep.[2]

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Higher Arousal Levels (Alpha Attenuation Test Scores) prior to Sleep Onset are Related to NREM Dream Recall more than SOREMP Dream Recall

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Introduction: Sleep Onset REM periods (SOREMPs) are expressions of REM at sleep onset typically associated with Narcolepsy. SOREMPs also occur in normal sleepers under conditions in which the sleep cycle has been disrupted **1**. Measures of subjective sleepiness are higher after SOREMPs compared to normal NREM sleep onset naps (NREMPs)**2** and MSLT latencies are shorter for SOREMPs**3**. This study wished to determine whether the Alpha Attenuation Test**4** (AAT, the ratio of eyes closed/eyes open alpha), an objective measure of sleepiness, Stanford Sleepiness Scores (SSS) and Visual Analogue Sleepiness Scales (VASS) measured before sleep onset would be related to the occurrence of SOREMPs and/or dream recall.

Procedures: Eight students spent 7 nights in the lab (experimental nights 4^{th}-7^{th}) undergoing a modified Sleep Interruption Technique (SIT)**5**. In each experimental night, participants were awakened when 50 minutes of NREM sleep had elapsed from the termination of the 3rd REM period and kept awake for one hour, during which they completed a Vigilance task (30 min.), an AAT (6 min.), SSS and VASS. Participants were then allowed to return to sleep and awakened after 5 minutes elapsed from the first appearance of either REMs (SOREMP) or spindle/K-complex wave (NREMP) and asked about their mental experiences prior to being awakened. These were classified as Dream, Thought, Forgot or Nothing. This procedure was repeated twice. Alpha EEG power (8.2-12.9Hz) was computed for O1 & O2 during eyes open and eyes closed sessions. The Alpha Attenuation Coefficient (AAC) was calculated as the ratio of eyes closed/eyes open alpha power and normalized (z scores) for each participant. Final analyses were conducted on the 6 participants who showed both SOREMP and NREMP episodes. To determine which factors were related to dream recall (DREAM) and nap type, (2X2) ANOVAs were performed on the SSS, VASS and normalized AAC scores with SOREMP/NREMP and DREAM/OTHER as I.V.s (Thoughts, Forgot, Nothing were all considered as OTHER).

Results: There was a significant interaction of AAC scores ($F(1,38)=4.4$, $P=.04$) such that participants were most highly aroused (less sleepy) before NREMP naps after which Dreams were reported. There was also a significant main effect of DREAM ($P<.001$) and a trend of SOREMP ($P=.07$) such that participants demonstrated increased sleepiness (lower AACs) before SOREMP naps and naps from which Dreams were recalled. Neither the SSS or VASS scores had any significant effects although the VASS did show weak trends ($p=.13$) towards increased sleepiness before naps which exhibited SOREMPs and those from which Dreams were recalled.

Discussion: These results are consistent with previous research showing that dream reports from NREMP during sleep onset are related to arousals from sleep**6** and increased sleepiness after awakening from SOREMP naps**2,7**. These results also lend support to the hypothesis that different factors underlie dream occurrences between REM and NREM sleep**2,6,7**.

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Diagnosing Insomnia: A Decision Tree to Guide Clinical Decision-Making

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The key to successful treatment of insomnia is proper diagnosis. Since the diagnosis of a sleep complaint has a considerable impact on the eventual choice of treatment [1], strategies must be developed to guide clinical judgment as well as decisions in regards to when it becomes appropriate to refer a patient to a sleep disorders clinic.

We thought that one useful strategy would be a decision tree approach to the differential diagnosis of insomnia. Nowadays, many different health care professionals (practicing nurses, family physicians, psychiatrists and psychologists) are often faced with the responsibility of evaluating and diagnosing a complaint of insomnia. Because these professionals all come from discipline-bound training and schools of thought, it should be no surprise that the manner in which the diagnostic process is undertaken can significantly differ from one professional to another, thus increasing the probability of errors in judgment, of errors in diagnosis and decision-making, and ultimately, of errors in the judicious selection of treatment(s).

Given the diverse training populations and the inherent diagnostic uncertainty of insomnia, we decided to construct a decision tree as an aid to the differential diagnosis of insomnia. Decision trees have often been used in situations where a clear understanding of a certain decision-making process is needed. This decision tree is thus an attempt to provide a concrete formulation to the implicit reasoning a clinician must engage in when diagnosing a complaint of insomnia. It is a memory jogger: a visual cognitive representation that reveals a process that otherwise goes unverbalized. The use of this model of reasoning or diagnostic tool should thus reduce reliance on rote memorization and unsubstantiated inferences based on each interviewer's personal conceptualization of insomnia. However, one must recognize that a decision tree is not a standalone tool that replaces sound clinical judgment and experience.

Because of its emphasis on the decision-making process, the decision tree will be somewhat agreeable to modifications and therefore suit the needs of different audiences: that is, from the general practitioner's office to the highly specialized sleep clinic. In professional training and continuing education, such a model could prove to be a valuable pedagogical tool.

Since the International Classification of Sleep Disorders is the reigning classification for sleep disorders, we decided to elaborate our decision tree using its nomenclature and diagnostic categories. Beginning from the sleep complaint, the decision tree consists of an outline of the basic reasoning one has to employ in order to investigate all of the etiologies potentially responsible for initiating and/or maintaining the insomnia. A plausible diagnosis corresponds to each of the individual levels of questioning. Because insomnia often has multiple etiologies, one must not abandon the decision-making process once a plausible diagnosis has been identified. Rather, it is essential to employ all levels of questioning, while keeping in mind other diagnostic possibilities, before arriving at a more definite diagnosis.

Future endeavors should focus on validating this decision tree as a diagnostic tool as well as developing decision trees for the other two nosologies.

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Effects of Sleep Deprivation on P3a Novelty Evoked Response Potentials

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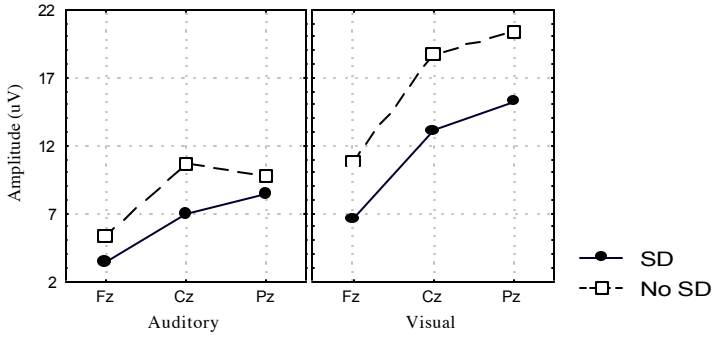
Introduction: Electrophysiological[1](EP) and behavioral[2] evidence suggest that frontal lobes are particularly vulnerable to the effects of sleep deprivation (SD). Few studies have recorded event-related potentials (ERP) during SD. The detection of an infrequent "target" stimulus occurring among a train of more frequently presented standard stimuli is associated with a late positive wave called P3. This P3 is maximum over parietal areas of the scalp. Presentation of an infrequent but highly novel stimulus is associated with an earlier positivity, P3a, which is more frontal dominant. This paradigm is called the *Novelty Oddball Paradigm* (*NO*). P3a is much reduced in frontal lobe injury patients, while the classic P3 remains[3] relatively unaltered. The aim of the present study is to assess the effects of SD on P3a, by using the *NO* paradigm.

Method: 6 right-handed, drug free participants (2 men and 4 women; age range: 19 to 26 yrs) volunteered for the study. They were tested over a 36 h SD period and a recuperation night. The EEG was recorded from midline frontal, central and parietal placements (Fz, Cz, Pz). EOG (vertical and horizontal) were also recorded to monitor blink artifacts. In order to control for practices effects, two versions of the *NO* were administered. In the visual *NO* task, participants were asked to button press upon detection of a rare (p=0.10) inverted triangle occurring among a train of more frequently presented (p=0.80) triangles. On 10% of trials, a novel highly coloured abstract drawing was presented. In the auditory *NO* task, participants were asked to detect a rare high pitch tone (p=0.10) occurring among more frequently presented low pitch tones (p=0.80). A novel environmental sound was presented on 10% of trials. In both versions, participants were told to ignore the novel stimuli. The visual *NO* was administered at 12 h and 36 h SD and the auditory *NO* was administered at 36 h SD and after recuperation. ERPs were averaged separately for each stimulus type, for each session. The ERPs following the novel stimulus are discussed in this presentation.

Results: Both the auditory and visual novel stimuli elicited a positive wave peaking from 275-350 ms that was maximum over central-parietal areas of the scalp. P3a was larger to the novel visual than novel auditory stimuli, but this difference did not reach significance ($p<0.1$). As indicated in Figure-1, SD resulted in a significant reduction in the amplitude of P3a in both the visual and auditory tasks ($p<0.017$).

Discussion: These results suggest that SD causes a reduction in the amplitude of P3a. This effect remained whether or not the first testing period took place during SD, and therefore cannot be explained by repetition of the novel stimuli over the various sessions. However, these results could also be explained by a global attention deficit caused by SD rather than a specific effect of SD on frontal lobes[4]. Further analyses, comparing the results of the *Novelty Oddball paradigm* with those of the classic *Oddball paradigm* could increase our understanding of this phenomenon.

Figure 1- Variation of the Amplitude of the P3a generated by an Auditory and a Visual Task with and without SD



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The sleep of patients with schizophrenia: A meta-analysis

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Introduction. Despite numerous efforts, inconsistencies remain regarding the characteristics of sleep in patients with schizophrenia.(1,2) The goal of the present study was to integrate the results of published articles having compared the sleep of patients with schizophrenia to healthy participants.

Method. To be included, the studies had to meet a set of criteria, including the presence of a control group made of healthy participants and the formal presentation of descriptive statistical data corresponding to a set of predetermined dependant variables (see Table 1). A total of 14 studies met the criteria, involving an average of 14.6 ± 6.7 patients and 15.0 ± 5.7 healthy participants. These studies were included in a statistical meta-analysis, using the method of Hunter and Schmidt.(3) Data was subjected to common metric "t-tests" and the average "T" was calculated for each sleep variable.

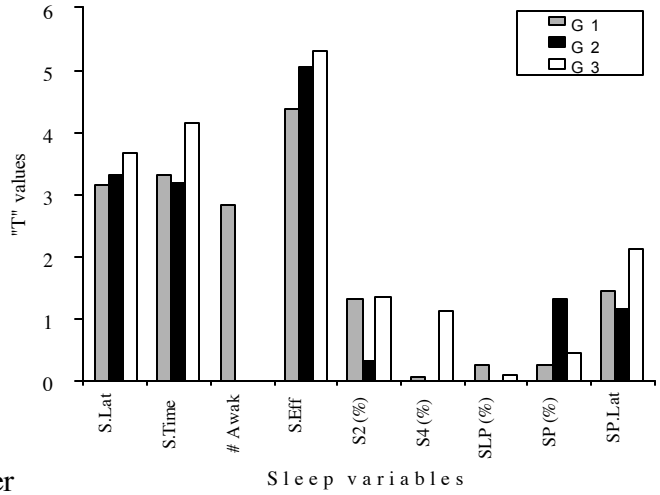
Results. The following variables showed a significant difference between patients with schizophrenia and healthy participants: sleep latency, total sleep time, number of awakenings, sleep efficiency and REM sleep latency.

Table 1. T and sigma statistics for sleep variables observed in 14 studies

	S.Lat	S.Time	#Aw	S.Eff	S2(%)	S4(%)	SLP(%)	SP(%)	SP.Lat (%)
T	3.93*	-4.29*	2.21*	-4.29*	-0.96	-1.37	-0.29	0.05	-2.01*
σ	0.22	0.27	0.36	0.19	0.13	0.68	0.08	0.30	0.26

Moderator analysis showed that variance decreased when these were grouped according to treatment: never treated (G1, n=5 studies), without treatment for more than 2 weeks (G2, n=4), without treatment for 2 weeks or less (G3, n=5). The three groups of patients were significantly different from healthy participants on sleep latency, total sleep time, number of awakenings and sleep efficiency. T values increased from G1 to G3 (Figure 1). Stage 4 (%) and REM sleep latency reached significantly only when subjects from G2 and G3 studies were grouped ($T = -2.27$; $p<0.05$ and $T = -3.28$; $p<0.05$, respectively).

Figure 1. Moderator variables: Neuroleptic treatment



Conclusion. Increased sleep latency and number of awakenings, decreased sleep efficiency and sleep time, and shorter REM latency are consistently present in patients with schizophrenia. Moderator analysis showed that neuroleptic withdrawal have residual effects that amplify these sleep disorders.

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c-Fos Expression in Dopaminergic and GABAergic Neurons of the Ventral Mesencephalic Tegmentum after Paradoxical Sleep Deprivation and Recovery

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Lesion and pharmacological studies have indicated that dopaminergic neurons may regulate behavioral arousal and sleep-wake states, yet recording studies have not reported changes in average discharge rate for presumed dopaminergic neurons, although they have for non-dopaminergic neurons of the ventral mesencephalic tegmentum (VMT) across waking, slow wave sleep (SWS) and paradoxical sleep (PS). In the present study in rats, we examined by using c-Fos expression, as a reflection of neural activity, combined with dual-immunostaining for tyrosine-hydroxylase (TH) or glutamic acid decarboxylase (GAD), whether dopamine (DA) and adjacent GABA -synthesizing neurons in the substantia nigra (SN) and ventral tegmental area (VTA) are differentially active in association with PS deprivation and recovery. The number of GAD+/c-Fos+ neurons was higher with PS recovery than PS deprivation and control conditions. In contrast, the number of TH+/c-Fos+ neurons did not change in the SN, however in the VTA, their number was higher with PS recovery than PS deprivation and control. The results suggest that the DA VTA neurons were more active during waking and PS than SWS and most active during PS. Together with evidence from recording studies, it would appear that neither SN nor VTA DA neurons are tonically inhibited by local GABAergic neurons during PS, but they may be submitted to differential inhibitory and excitatory influences such that VTA mesolimbocortical, and not SN nigrostriatal, DA neurons may change their pattern of discharge to be most active in association with PS and therein contribute to the unique physiological and cognitive aspects of that state.

Supported by MRC

Discharge Properties of Juxtacellularly Labelled and Immunohistochemically Identified Cholinergic and GABAergic Basal Forebrain Neurons in Relation to Cortical EEG Activity

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It is well recognized that the basal forebrain plays an important role in the regulation of cortical activity and behavioral state. It has been shown to be essential for stimulating cortical activation in association with waking (and paradoxical sleep), yet it is also important for attenuating cortical activation and promoting slow wave sleep. The cholinergic neurons of the basal forebrain are thought to be a major contributor to the modulation of cortical activity across states. However, in addition to cholinergic neurons, numerous GABAergic neurons are located in the basal forebrain from where they give rise to cortical or diencephalic long projections in addition to local projections, and could accordingly modulate cortical and subcortical activity in unique ways. In order to examine the functional roles of these two groups of codistributed cell types, we employed juxtacellular labeling of cells with neurobiotin combined with immunohistochemical staining for choline-acetyl transferase (ChAT) or glutamic acid decarboxylase (GAD) to identify recorded cells as cholinergic or GABAergic. Cells were recorded in the magnocellular preoptic area (MCPO) and substantia innominata (SI) in association with EEG activity recorded from limbic cortical regions in urethane-anesthetized rats. EEG activity was changed from large irregular slow activity to low amplitude rhythmic slow activity by pinching of the tail, thus eliciting a degree of cortical activation without evoking a behavioral response in the anesthetized animal.

Cholinergic neurons exhibited distinct characteristics in their neuronal discharge. First, all cholinergic cells increased their firing rate with stimulus evoked cortical activation. Second, many cholinergic cells shifted from irregular discharge during EEG slow irregular activity to a slow rhythmic pattern during evoked EEG slow theta-like rhythmic activity. The rhythmic discharge was comprised of bursts or trains of spikes that correlated with the rhythmic cortical EEG activity.

GABAergic neurons displayed heterogeneous discharge characteristics and comprised distinct physiological subgroups. The majority of these cells decreased their firing rate in association with stimulus evoked cortical activation. One subgroup discharged in a phasic bursting manner in relation to the slow irregular cortical activity and become virtually silent with stimulation evoked cortical activation. Two other subgroups discharged in a more tonic manner during large irregular slow cortical activity. One of these subgroups simply decreased its rate of discharge with stimulation, while the other subgroup decreased its rate of discharge concomitant with the onset of a slow rhythmic pattern of activity with stimulation.

These results indicate that cholinergic basal forebrain neurons may evoke or enhance cortical activation by increasing their rate of discharge, discharging rhythmically and thus potentially modulating cortical activity in a rhythmic manner, as may be manifest by theta activity that occurs during active waking and paradoxical sleep states. By different subgroups, GABAergic cells could potentially inhibit codistributed cholinergic neurons during slow irregular cortical activity characteristic of slow wave sleep and others could shape that cortical activity. These different cholinergic and GABAergic cell groups may accordingly control cortical activity and influence behavioral state across the sleep-waking cycle.

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Judicious Control of the Pattern of Light Exposure in Circadian Readaptation to Night Shift Work

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Introduction. As demonstrated in laboratory and field studies of shift work, bright light phototherapy in the workplace may be effective in helping the shift worker adjust to an inverted sleep/wake schedule (1,2). Investigations have also demonstrated that morning bright light may impede the circadian adjustment to a night-oriented schedule (4). This field and laboratory study investigated the efficacy of an intervention regimen in the adjustment of the endogenous circadian system of night shift workers.

Methods. Fifteen full-time night shift workers (6 male, 9 female; mean age ± SD: 40.8 ± 8.4 years) were recruited for this study. Following a vacation period including ≥ 10 days on a daytime schedule, workers were admitted to the laboratory for a 36-hour constant routine procedure (CR). Workers then returned to their regular night shift work schedule for an average of 12 shifts, under one of two experimental conditions: treatment or control. Workers assigned to the treatment condition were exposed to bright light (2000-7000 lux) during the first 6 hours of each night shift, and wore dark goggles on the commute home. Control group workers were observed in their habitual lighting conditions. Subjects maintained regular 8-hour day sleep/dark periods beginning 2 hours after the end of their night shifts. At the end of the ambulatory period, workers were readmitted to the laboratory for a final 36-hour CR. Endogenous circadian phase was determined from CR temperature data via a dual-harmonic regression model without serial correlated noise.

Results. At the start of the study, both groups were adjusted to a day-oriented schedule and no between-group differences were observed ($F_{(1,34)}$=0.36, p=0.4). Mean initial circadian phase of the treatment and control groups were (± SEM) 4.72 ± 0.60 and 5.90 ± 1.14 hours, respectively. Following the period of night shifts, the treatment group displayed a -10.37±1.41 hour phase delay. The observed phase delay in the control group was of only -5.33±2.11 hours. Final circadian phase in the control and treatment groups were 10.06±2.23 and 16.27±1.16 hours, and were significantly different, ($F_{(1,34)}$=9.97, p=0.003).

Conclusions. Mean final phase in the treatment group was aligned with a night-oriented rest/activity cycle. These results suggest a practical means of promoting circadian adjustment in the night shift worker. Moreover, they emphasise the importance of the pattern of exposure to light throughout the day, in the process of circadian entrainment to an inverted schedule (3).

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Analyse spectrale de l'EEG du sommeil paradoxal de patients narcoleptiques

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La physiopathologie de la narcolepsie est associée aux mécanismes neurophysiologiques du sommeil paradoxal (SP). Nous avons utilisé l'analyse spectrale de l'EEG en SP afin de mieux comparer l'activation corticale de patients narcoleptiques à celle de participants contrôles sains.

Méthode: Nous avons comparé six patients narcoleptiques (4 H, 2 F; 24.7 ± 4.8 ans) à six participants contrôles sains (4 H, 2 F; 26.4 ± 7.1 ans). Suivant une veille contrôlée de 16 heures, la deuxième nuit d'enregistrement a été lue visuellement par pages de 20 secondes suivant les critères de Rechtschaffen and Kales (1968). Pour chacune des quatre premières périodes de sommeil paradoxal, trois pages de 20 secondes sans artefacts ont été sélectionnées, pour un total de 60 secondes d'échantillon EEG. La puissance spectrale de l'EEG des dérivations C4/A1 et C3/A2 a été determinée de 0.25 à 19.75 Hz en tranches de 0.25 Hz par transformée rapide de Fourier. Les valeurs des groupes ont été comparées par le test U de Mann-Whitney.

Résultats: La puissance spectrale de l'ensemble des bandes de fréquence diminue de la première à la quatrième période de SP chez le groupe contrôle mais pas chez le groupe narcoleptique, surtout pour les fréquences alpha ($p < .05$) et sigma ($p < .01$). Comparativement aux contrôles, les patients narcoleptiques ont une puissance spectrale moins élevée dans les fréquences delta rapides (de 1.75 à 2.25 Hz; $p<.05$) et sigma rapides (de 14.0 à 14.75; $p<.01$) pendant la première période de SP seulement.

Conclusion: La diminution de la puissance lors de périodes de SP successives chez les participants contrôles sains corroborent les résultats de Merica et Blois[1]. L'absence de ce phénomène chez les patients narcoleptiques suggèrent un déficit d'hyperpolarisation thalamo-corticale (surtout marqué par les fréquences delta et sigma en début de nuit)[1] et/ou un déficit de l'homéostasie du SP (surtout marqué par la diminution de la puissance alpha au cours de la nuit).[2]

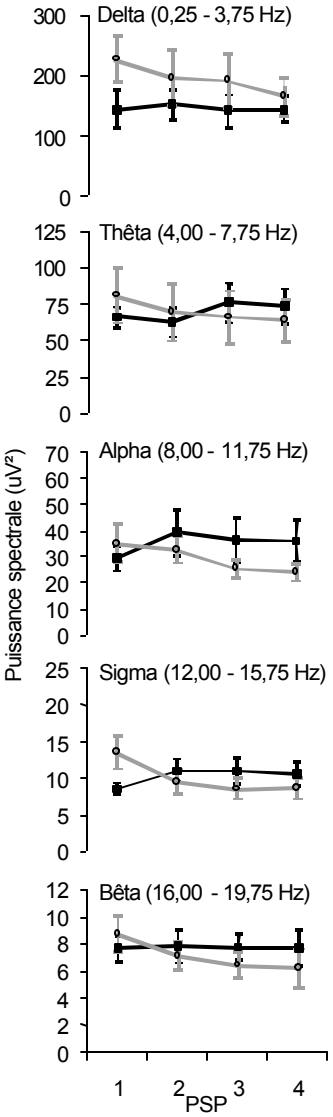

Figure 1 : Puissance spectrale en fonction des périodes de SP pour les patients (en noir) et pour les contrôles (en gris).

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Correlation between sleep abnormalities and cognitive dysfunctions in drug-naive patients with schizophrenia

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Objective. Many studies have analyzed sleep in schizophrenia in order to better understand the pathophysiology of this disease but only one reported on the relationship between cognitive performance and sleep.[1] This study, however, only analyzed REM sleep variables and reported only on explicit memory, whereas patients were recently withdrawn from chronic neuroleptic treatments. For the present report, we have performed sleep recordings and a thorough neuropsychological evaluation of attention and memory in drug-naive patients with schizophrenia, a group of patients very rarely described in the literature.

Method. Five schizophrenia patients never treated with neuroleptics (1W, 4M; 36.2 ± 20.4 years old) were recorded for two consecutive nights in a sleep laboratory. Sleep stages and phasic events (sleep spindles and rapid eye movements (REMs)) of night 2 were scored according to standard criteria. In the morning following night 2, attention and memory were evaluated using a computer-assisted battery. Correlation between sleep variables and cognitive performances was calculated using Spearman's Rho.

Results. As expected, patients with schizophrenia showed impaired performance in attention and explicit memory and normal performance in implicit memory (Data not shown). The table below shows the significant correlation coefficients we found between cognitive performance and sleep variables.

	Selective attention (RT)	Sustained attention (RT)	Memory span (Nb)	Explicit memory (CR)	Explicit memory (FPE)	Implicit memory (CR)	Implicit memory (FPE)
Sleep efficiency(%)	-.90*						
Stage 2 %	.90*		-.95**		-.95**		
Stage 4 %	-.90*		.95**				
REM sleep (min)						.90*	-.97***
Spindles (Nb/min)	-.90*			.97***			
REMs (Nb/min)					-.95**		-.82

CR: correct response; FPE: False positive errors; RT: reaction time. *** p<.005; ** p<.01; * p<.05

Discussion. The present results show that the relationship between attention/memory and sleep organization in neuroleptic-naive patients with schizophrenia is comparable to what is found in healthy subjects.[2,3] Moreover, explicit memory was found to correlate with both REM and nonREM sleep. Given the fact that patients with schizophrenia display impairments in attention and memory, these results suggest that neural networks involved in cognitive processing during sleep could be impaired in this disease.

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EEG spectral analysis before and after sleep in high-functioning autism spectrum disorders

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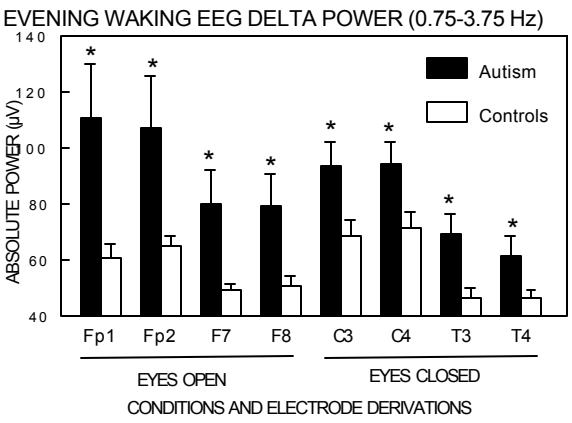
Most studies have found evidence of frontal and temporal lobe dysfunction in autism (Schultz et al., 2000). We wanted to examine if these abnormal brain activity could be evident during waking EEG and if this measure was changed dependant of the day moment (before and after sleep).

Method: Seven autistic subjects (7H; 22.1 ± 14.5 ans) with normal IQ have been compared with seven healthy subjects (7H; 25.7 ± 14.0 ans). Wake EEG before and after the night have been obtained in each subject using a monopolar recordings refered to linked ears. Subjects were recorded for five minutes with eyes closed (EC) and 5 minutes with eyes opened (EO) on each moment. Spectral analysis was performed on 10 to 15 four-second epochs on absolute power (µV/Hz, 0.75Hz à 19.75Hz). Four frequency bands were created: Delta (0.75-3.75 Hz), Thêta (4.0-7.75 Hz), Alpha (8.0-12.75 Hz), and Beta1 (13.00-19.75 Hz) and recorded into different cerebral regions: frontal (FP1,FP2,F7,F8), central (C3,C4), temporal (T3,T4) and occipital (O1,O2). Groups comparisons on EEG spectra before and after sleep have been performed using t-tests.

Results: Before sleep, compared to controls, autistic subjects have elevated delta activity in the EC condition in central (C3: $t=2.5$, $p < .03$; C4: $t=2.4$, $p<.03$) and temporal regions (T3: $t=2.8$, $p<.02$; T4: $t=2.0$, $p<.07$). In EO condition before sleep, autistic subjects showed an elevated spectral power in delta (FP1: $t=2.5$, $p<.03$; FP2: $t=2.0$, $p<.07$; F7: $t=2.5$, $p<.03$; F8: $t= 2.3$, $p<.04$) and theta activity bands (FP1: $t=2.9$, $p<.01$; FP2: $t=2.2$, $p<.05$; F8: $t=2.1$,$p<.06$) in frontal regions. After sleep, spectral EEG is no more different between groups in any condition.



Discussion: We observed that at the end of the day, autistic subjects shows elevated spectral power compared to healthy subjects for delta and theta bands, especially in central, temporal and frontal regions. These differences are however no more evident after sleep. Two possible causes may explain these observations: 1) an increase diurn fatigability in autism, or 2) a better nocturn sleep beneficial effect.

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The all-or-none law revisited:
Stimulus parameters affect the amplitude of the K-Complex.

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The evoked K-Complex is a very large amplitude waveform that can be elicited during NREM sleep. In an oddball task, a subject is presented with a regular series of "standard" stimuli. At odd and random times, the standard is changed to a "deviant" stimulus. This rare, deviant stimulus may elicit a K-Complex. The purpose of the present study is to determine the effects of deviant probability on the K-Complex. Three groups of 7 young adults (total n = 21) spent a single night in the sleep lab. An 80 dB SPL 1000 Hz standard tone pip was presented every 1.5 s. In different conditions, the standard was changed to a deviant. In one condition, the pitch of the deviant was altered to be 2000 Hz. In another condition, the intensity of the deviant was changed to be 100 dB. The three different groups of subjects heard the deviant on either 20, 10 or 5% of trials. The EEG was recorded from midline frontal, central and parietal placements. The probability of eliciting a K-Complex varied with the type of deviant and its probability of occurrence. A K-Complex was elicited on 40-50% of the high intensity deviant trials while it was elicited on 20-40% of high pitch deviant trials. The K-Complex occurred more frequently when the deviant occurred less frequently. A large amplitude negative wave peaked at about 600 ms. The amplitude of this negative wave was however not affected by the probability of deviant occurrence. Its amplitude was significantly larger following intensity compared to pitch deviants. The present study thus indicates that the time between deviants has little effect on the K-Complex. On the other hand, the amplitude of the K-Complex is largest when stimuli are very loud. This finding contradicts Bastien and Campbell's 's (1992) claim that whenever a K-Complex occurs, its amplitude does not vary.

An Investigation of Spontaneous K-complexes in High and Low Sleep-Efficiency Nights

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Introduction: Is the K-complex (KC) sleep protective or does it represent an arousal? The function of this sleep phasic event remains unknown. Wauquier et al.[1] proposed that by investigating the number of KCs in various sleep disordered populations, we may be able to determine its function. Specifically, they hypothesized that if the KC was sleep protective, then one would expect the number of KCs to be *lower* in sleep disordered groups. We investigated this hypothesis in low and high sleep-efficiency groups. We further classified KCs as Type A, Type B, or Type E according to methods described Paiva & da Rosa[2]. The distinct morphology of the KC types may be functionally different.

Procedures: Archival polysomnographic data were examined from patients visiting the Paris Sleep Clinic between 1994 and 1998. None were diagnosed with apnea or periodic limb movements. All records represent first nights in a sleep laboratory. Sleep files were grouped according to high (>80%) and low (<80%) sleep efficiency. The low sleep-efficiency group (n=9) had a mean SE=44.9% (SD=21.5, range 19.0-74.0), and the high sleep-efficiency group had a mean SE=89.3% (SD=5.1, range=82.3-97.5). KCs were identified in stage 2 by visual inspection and then classified as follows: Type A is a large negative deflection, without the subsequent positive component (i.e., N550). It may be viewed as an 'immature' KC, slower than a vertex sharp wave, but not the classic shape of the KC. Type B is the classic KC with the biphasic negative peaks, followed by a positive deflection (i.e., N350-N550-P900). Type E is similar to Type B except that the smaller earlier negative peak is not apparent (i.e., N550-P900).

Results: The groups did not statistically differ with respect to age (M_L=43.9 yrs; M_H=37.7 yrs), percent period time spent in stage 2 sleep (M_L=53%; M_H=51.7%), and the total number of K-complexes observed (M_L=85.1; M_H=119.2). A mixed design ANOVA was conducted to compare the number of KCs within each classification (Type A, B, E) and between groups. A significant KCtype by group interaction ($F(2,32)=4.91$, $p<.01$, $eta^2=.24$) revealed that the low sleep-efficiency group had less Type B and Type E KCs (i.e., classic type), yet more Type A KCs, relative to the high sleep-efficiency group. Since Type B and E were thought to be functionally similar (i.e., the lack of N350 merely due to overlapping frequencies obscuring its appearance), they were combined for a subsequent analysis. The significant interaction remained in the 2 (Type A, Type B + E) x 2 (group) ANOVA, $F(1,16)=4.87$, $p<.05$, $eta^2=.23$.

Discussion: Fewer KCs (Type B & E) in the low sleep-efficiency group replicates Wauquier's findings and supports the hypothesis that the KC functions to protect sleep. The Type A KC may represent an immature KC that fails to form completely in the poor sleepers. The greater proportion of Type A KCs in the low sleep-efficiency group may be a result of more frequent arousals, and thus more transitions to sleep where such immature KCs are often observed.

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Canadian sleep centers: A survey

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Background: Chronic insomnia is the most common sleep complaint confronting health care practitioners today. It is estimated that 20% of Canadians suffer from insomnia.[1] Poor quality of sleep followed by daytime consequences such as fatigue, irritability, and decreased memory and concentration and general functioning are complaints commonly associated with insomnia. Most patients complaining of insomnia are seen by primary care physicians[2] who may not have had the benefit of thorough training in the new methods of sleep medicine.[3-5] One study evaluating physician response to a hypothetical insomnia case found that only a half of the primary care physicians included questions about sleep in their history taking, and less than a quarter asked about caffeine intake.[6] Another survey found that 70% of patients with chronic insomnia do not bring up their sleep problem with the physician.[7] In an attempt to provide better care for insomniacs some sleep centers in Canada are developing programs to specifically target this group of patients. We surveyed 83 sleep centers located across Canada as to what services are available for patients with complaints of insomnia.

Methods: All 83 sleep centers listed on the Canadian Sleep Society website were contacted by phone. The survey was conducted from October 2000 to March 2001. The sleep centers contacted included facilities that were privately and/or provincially funded and were clinical and/or research oriented. Using a 10-item questionnaire, we asked about the type of services provided to patients complaining of insomnia. Specific enquires were made into the types of referral sources, assessment procedures, use of questionnaires and sleep logs and treatment modalities offered.

Results: The response rate to the survey was 80%. Most centers (70%) indicated that they do not provide treatment to patients complaining of insomnia due to the facilities specialized orientation (research or respiratory only). A majority (72%) of the clinics do provide screening for other sleep disorders such as restless legs and sleep apnea to patients complaining of insomnia. If the sleep study is inconclusive, some (30%) of clinics will provide basic sleep hygiene education or prescribe a sleeping medication. Only six (7%) sleep centers had services available onsite or in the local community. These sleep clinics either provide services as part of their clinic or they have a community referral source (psychologist) or an inter-hospital relationship with the psychology department. In one clinic, the lack of resources in the hospital and in the community has led to a waiting list of over 100 patients in just one year.

Conclusions: Few Canadian sleep clinics provide treatment to patients with insomnia in a programmatic fashion. This reflects a combination of factors: limited resources, long waiting lists, the inconclusive diagnostic usefulness of sleep studies on the diagnosis of insomnia and the relative lack of trained professionals in this area; Canadian universities provide little teaching about sleep in either the medical or the psychological curricula. It appears that most cases involving insomnia are treated by the family practitioner or in certain areas by a specialized professional (psychologist) but not directly by the majority of sleep clinics.

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The Impact of "Sleepiness" on Adolescent Students

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There has been little research on the effect of sleepiness on students. This study is an attempt to identify the extent of the problem in a population of Canadian students.

Method: An anonymous questionnaire (117 items) was completed by 2200 students from three secondary schools in different socioeconomic neighborhoods; two city schools (middle class, working area and an inner city multicultural area) and a rural school. These were completed between 10 and 11 AM at all schools. Questions focussed on sleepiness (Epworth, Stanford scales), performance (grades, lates, extracurricular activity, sleepiness at school), sleep times, sleepiness and time of day, sleep "hygiene", symptoms suggestive of primary sleep disorders. An initial test-retest reliability study was performed at one of the participating schools.

Results: The results from all three schools were essentially the same.

- 18% of the students indicated excessive daytime sleepiness.
- 24% of the students felt their grades had dropped because of sleepiness, these students reported 25-30 minutes less weeknight sleep than their peers.
- 17% of the students reported they were involved in fewer daytime activities because of sleepiness.
- 19% had missed social, sporting events or work because of sleepiness school "lates" correlated directly with level of daytime sleepiness.
- 47% of students had less than 8 hours sleep per weeknight, only 30% had 8.5 or more hours of sleep per weeknight.
- 50-60% of the students reported that they were "most sleepy" between 8 and 10 A.M. "good" bedtime routines were endorsed only 18-19% of the time. "poor" bedtime routines were associated with decreased performance and increased daytime sleepiness.
- 7% of the students responded to the questionnaire in a manner suggesting the presence of a primary sleep disorder (insomnia, restless legs syndrome, narcolepsy, sleep apnea), of these less than 20 % gave any indication of being aware of, or of being told about, any disorder.

Discussion: For logistic and compliance reasons the questionnaire from this first phase of the study was anonymous and therefore the results cannot be validated. However, the large number of students and the similar results from three different schools provide a degree of confidence. While most of the differences identified were statistically significant, the simple descriptive statistics indicate that sleepiness may be a significant factor in the education and quality of life of adolescents. The majority of students had less than adequate sleep and sleep habits, the consistent reporting of degrees of sleepiness throughout the day has implications for school start times and academic scheduling. There is concern that there may be a significant number of adolescent students with undiagnosed sleep disorders.

Conclusion: The study indicates that sleepiness has an important negative impact on the education and quality of life of adolescent students. The problem needs to be seriously addressed by all the partners in education and further research is clearly necessary.

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Suggested Immobilization Test (SIT) in schizophrenia: an exploratory study

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The Suggested Immobilization Test (SIT) is used to diagnose Restless Legs Syndrome (RLS), a sensorimotor disorder causing insomnia. Neuroleptic-induced akathisia is also a sensorimotor phenomenon. We wanted to verify whether these disorders coexist in schizophrenia. We studied two groups of neuroleptic-treated patients with schizophrenia, diagnosed according to DSM-IV: Sch-A were 8 patients (age: 21-58 yrs) with a high score (≥4) on the Barnes (1989) akathisia scale. Sch-NA were 7 patients (age: 22-53 yrs) without akathisia (Barnes <1). None of the patients took antiparkinsonia drugs. Controls were 8 healthy subjects (23-58 yrs). SIT involves surface EMG recording of anterior tibialis muscles for 60 min. while the participant is sitting with legs outstretched. Participants reported level of discomfort in the lower limb on a visual analog scale every 5 minutes. Both group of patients showed a higher discomfort during the SIT, compared to controls. Only Sch-A patients showed a higher rate of EMG-detected leg movements compared to controls (49.38±19.4 vs 13.5±5.36). SIT results showed an association between RLS and neuroleptic-induced akathisia and we suggest that common neural, possibly striatal, structures may be involved. Subjective results point to the existence of a paresthesia-type of hypersensitivity that would be independent from objective measures in schizophrenia.

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